IOWA

Alumni Celebration and Annual Student Retreat

June 12th–13th, 2025

Brown Deer Golf Club

Thursday, June 12

Psychological and Brain Sciences Building, Room B70

Parking: Tower Place Parking Structure - 1 S Gilbert St, Iowa City, IA 52240

2:30 pm Debashish Bhattacharya, PhD
Public Seminar co-sponsored by the Department of Biology

“Developing knowledge about coral biology to build tools for reef conservation"

Medical Education Research Facility, Atrium

Parking: Newton Rd Ramp—426 Newton Rd, Iowa City, IA 52246

3:30 – 5:00pm Early Registration & Social Reception
4:00 – 5:00pm Lab Tours

Friday, June 13
Brown Deer Golf Club
1900 Country Club Dr, Coralville, IA 52241

8:30am **Breakfast/Registration**

9:00am **Opening Remarks**
Lori Wallrath, Ph.D.—Director
Andy Russo, Ph.D.—Retreat Committee Chair

9:10am **Matthew Strub, Ph.D. '20** (McCray Lab)
"Building Bioinformatic Resources for Immunology: The NIH's ImmPort Project"

9:30am **Alyssa Wetzel, Ph.D. '22** (Darbro Lab)
"Gateway to Clinical Genetics"

9:50am **Program Reflections—50 Years of Leadership**
Panel discussion with former program directors

11:00am **Coffee Break**
Sponsored by Integrated DNA Technologies

11:20am **Poster Spotlights**

11:45am **Remarks** - Interim Graduate College Dean, Saba Rasheed Ali, Ph.D.

11:50am **Group Photo**

12:00pm **Lunch & Discussion Tables** (2 sessions, 12:30-1:20pm)

1:15pm **Poster Session 1** — odd numbered posters

2:00pm **Poster Session 2** — even numbered posters

2:45pm **Break**

3:00pm **Life After a PhD: How to Succeed in Science**
Panel discussion with Genetics alumni

4:30pm **Closing Remarks & Poster Award Ceremony**
Andy Russo, Ph.D.—Retreat Committee Chair
Aislinn Williams, MD, Ph.D.—Retreat Committee Co-Chair

5:30pm **Social Gathering** at Field Day Brewing Co.
925 Liberty Way, North Liberty, IA 52317

IOWA | Interdisciplinary Graduate Program in Genetics

Discussion Tables

Table	Discussion Topics	Discussion Leaders
1	Teaching at a non-R1 institution	**Megan Ealy, PhD,** Associate Professor, Drury University **Janine Martin, PhD,** Associate Professor, Kirkwood Community College
2	Transitioning to industry	**Zach Kockler, PhD,** Molecular Lab Manager, JMI Labs/Element-Iowa City
3	Transitioning to clinical research	**Alyssa Wetzel, PhD,** Laboratory Genetics & Genomics Fellow, University of Iowa
4	Applying bioinformatics/ computational genetics skills in diverse fields	**Matthew Strub, PhD,** Lead Data Curator/ Bioinformatician, NIH (NIAID)
5	Writing a strong, fundable grant proposal	**Kim Bekas, PhD,** Science Writing Consultant, BioScience Writers
6	Finding a post-doctoral position	**Bruce Citron, PhD**, Professor, Rutgers
7	Navigating the world as a woman in STEM	**Theresa Zucchero Scocca, PhD**, Senior Research Scientist, Rho, Inc. **Rita Shiang, PhD**, Associate Professor, Virginia Commonwealth University

Discussion Tables will take place over the lunch hour

12:30 –12:50pm **Discussion Tables**—session 1
12:50 –1:10pm **Discussion Tables**—session 2

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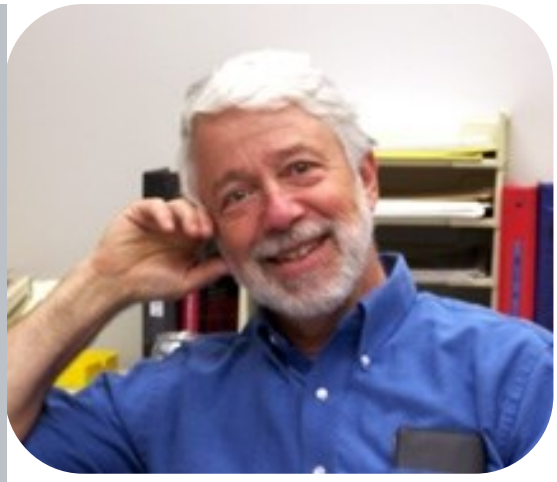
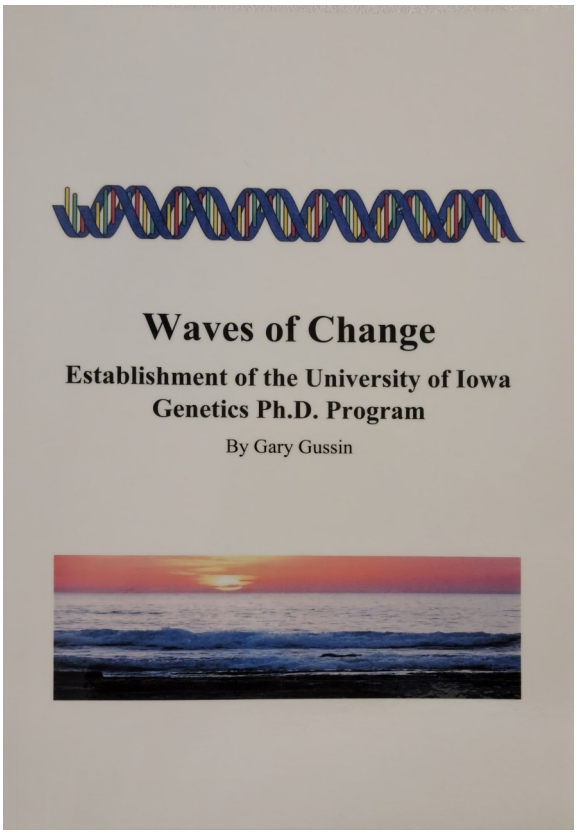


Dr. Gary Gussin
Professor Emeritus
Department of Biology

University of Iowa

Dr. Gary Gussin is a Professor Emeritus in the Department of Biology. Dr. Gussin was instrumental in the establishment of the Interdisciplinary Graduate Program in Genetics during the 1960's and was elected as the first Chairperson of the Program in 1975. Dr. Gussin published a book in 2021 titled *Waves of Change: Establishment of the University of Iowa Genetics Ph.D. Program*. The book recounts the early years of the program and successes, struggles, and contributions by the many faculty and administrators that helped develop the Program.

Waves of Change can be obtained by providing a minimum $20 donation to the Interdisciplinary Graduate Program in Genetics.



Program History

The Interdisciplinary Graduate Program in Genetics at the University of Iowa is a broad-based training program that incorporates an expansive range of techniques, model organisms, and approaches to actively address critical issues in biology, medicine, evolution, and genomics, with genetics as a common intellectual thread. Its purpose is to optimally prepare trainees to be at the forefront of genetics research and become future leaders in any area of modern genetics, from bioinformatics and -omics to molecular genetics, gene discovery and mapping, cancer and medical genetics, and personalized medicine. Our mission for society is to train a diverse cohort of graduate students in the broad discipline of genetics who are highly prepared and well-equipped to educate all members of society, who will increasingly need to understand genetic aspects of their own family's health care and individualized genomes.

The Genetics Program curriculum is designed to provide a solid and broad foundation in genetics and foster strong independent critical thinking skills and multidisciplinary training to equip students to meet modern challenges. Throughout the span of their educational training, there is emphasis on rigor and reproducibility, responsible conduct and ethical, safe practices and inclusivity in the sciences. The Genetics Program also provides flexibility tailored to individual needs to ensure student success. In addition, the program offers a Computational Genetics subtrack for students who want to develop strengths in the biological aspects of genetics and computational approaches to analyzing large and diverse sets of genomic and genetic data. Students thus equipped have been extremely successful in filling a growing niche in the contemporary science workforce. Research opportunities within the program span the spectrum of genetics, from bacterial to model organism to human genetics, and from developmental genetics to evolution, from epigenetics to genomics to disease mechanisms.

After several formative years of interdisciplinary research activity in Genetics on the University of Iowa campus, the Interdisciplinary Graduate Program in Genetics was approved as a degree-granting PhD program in 1975. This program that began with 7 primary faculty has grown to currently having 76 faculty and 45 students in four colleges and 16 academic departments across our campus, with almost 200 graduates. While the history of the program makes the University and the state of Iowa proud, it is the continued pursuit to change and incorporate novel ideas, modern methods, and societal needs that keeps successfully preparing our students for their scientific careers. We have an consistent track record of program completion, on-time graduation rates, publications, and awards, as well as career advancement to postdoctoral fellowships, faculty positions at research-intensive universities and primarily undergraduate institutions, and biotechnology companies. Since 1997, the Genetics Program has been supported by a T32 Predoctoral Training Grant from the National Institutes of Health, last awarded in July 2022.

Current Genetics Leadership

Director, Lori Wallrath, Ph.D.
Professor and Chair of Undergraduate Studies,
Department of Biochemistry and Molecular Biology

Dr. Lori Wallrath is a Professor of Biochemistry and Molecular Biology (BMB) at the University of Iowa. She currently services as the Director of BMB Undergraduate Studies and the Director of the Interdisciplinary Graduate Program in Genetics. She received her BS in Microbiology and her PhD in Genetics from Michigan State University. Her laboratory's research is focused on chromatin structure, gene expression, and nuclear envelope function, as it relates to human genetics disease.

Associate Director, Bryan Phillips, Ph.D.
Professor, Department of Biology

Dr. Bryan Phillips is a Professor of the Department of Biology at the University of Iowa and currently serves as the Associate Director of the Interdisciplinary Graduate Program in Genetics. He received his B.S. from the University of Illinois at Urbana-Champaign and his PhD in Biology (with a focus on developmental genetics) at Texas A&M University. His lab focuses on the role of cell signaling and cell polarity in gene expression and the role of protein aggregation and liquid phase separation in RNA regulation.

Program Administrator,
Rob DuBay

Program Associate,
Sydney Schmeltz

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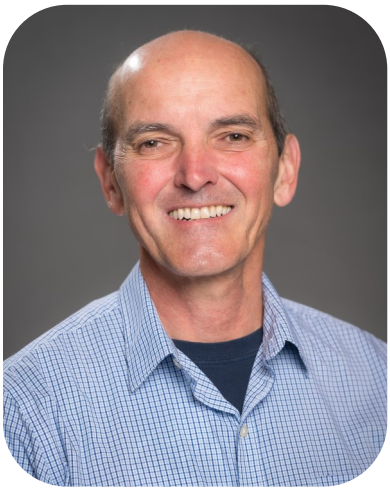


Genetics Retreat Chair, Andrew Russo, Ph.D.

Professor, Molecular Physiology and Biophysics

Dr. Andy Russo is a Professor of Molecular Physiology and Biophysics and Professor of Neurology at the University of Iowa. He received his PhD in Biochemistry from UC Berkeley and did postdoctoral training at UCSD on the neuropeptide CGRP. The focus of Dr. Russo's research is the molecular basis of migraine. He is currently using mouse genetic models to study how CGRP contributes to the pain and altered sensory processing of migraine. The overall goal of his studies is to develop effective diagnostic and therapeutic strategies for migraine and post-traumatic headache.

Genetics Retreat Co-Chair, Aislinn Williams, MD, Ph.D.

Associate Professor, Psychiatry

Dr. Aislinn Williams is an Associate Professor of Psychiatry at the University of Iowa and the Associate Director for Research at the Iowa Neuroscience Institute. She received her MD and PhD in Neuroscience from the University of Iowa, and then completed psychiatry residency and her postdoctoral fellowship at the University of Michigan. Dr. Williams's lab studies the molecular and cellular mechanisms by which genetic risk factors contribute to psychiatric disease from a developmental perspective. Her lab uses induced pluripotent stem cells and transgenic mouse models to study how risk genes like voltage-gated ion channels and the 16p11.2 duplication CNV alter neuronal development, neural circuit function, and behavior.

Genetics Student Committee Members

Lucas Casten
Michaelson Lab

Psychiatry

Ellen Koufer
M. Schultz Lab

Pediatrics

Jordan Mayberry
Kuehn Lab

Ophthalmology

Emily Fontenoy
Frank Lab

Anatomy & Cell Biology

IOWA | Interdisciplinary Graduate Program in Genetics

Matthew Strub, Ph.D.
Alumnus 2020, McCray

Lead Data Curator and Bioinformatician
NIH

"Building Bioinformatic Resources for Immunology: The NIH's ImmPort Project "

Matthew Strub was born and raised in Dubuque, Iowa and completed a bachelors of science in biology from St. Ambrose University in Davenport before joining the Interdisciplinary Graduate Program in Genetics in 2013. While at the University of Iowa, he was a member of the Paul McCray, Jr. Lab, where he performed transcriptomic and pathway analyses of cystic fibrosis lung disease to identify candidate therapeutic targets and small molecules. Following his thesis defense in 2020, he completed a post-doctoral fellowship at Mayo Clinic, where he developed new analysis pipelines for large-scale proteomics data. In 2023, he joined ICF International, where he serves as a bioinformatics consultant for the National Institute of Allergy and Infectious Diseases of the National Institutes of Health.

Abstract: The Immunology Database and Analysis Portal (ImmPort) is an NIH-funded public repository designed for sharing immunology data with the research community. From transcriptomics, proteomics, and metabolomics, to flow cytometry, immunoassays, and clinical results; ImmPort supports large, complex datasets that empower research. Established over 20 years ago, ImmPort currently contains over 7.5 million experimental results across more than 1,200 studies, 175,000 subjects, and 175 diseases. ImmPort allows the broader research community to explore a wide spectrum of clinical and basic research data and associated findings. The ImmPort team performed a meta-analysis of publicly available ImmPort data, called the 10,000 Immunomes Project, which describes variations in serum cytokines and leukocytes by age, race, and sex; defines a baseline cell-cytokine network; and describes immunologic changes in pregnancy. As additional data are added to ImmPort, further analyses are being performed and tools are being developed to benefit data consumers and re-users.

Alyssa Wetzel, Ph.D.
Alumnus 2022, Darbro

Laboratory Genetics & Genomics Fellow
University of Iowa

“Gateway to Clinical Genetics”

Alyssa Wetzel, PhD ('22) attended Cornell College in Mt. Vernon, Iowa where she earned a B.A. in Biochemistry & Molecular Biology and a minor in Pyschology. She had the opportunity to do undergraduate research at North Dakota State University with Dr. Christopher Colbert's and Baylor College of Medicine with Dr. James Lupski's. Dr. Wetzel earned her PhD from the genetics program in 2022 and defended her thesis entitled 'A Data-Driven Framework for CNV Pathogenicity Classification'. She stayed on as a post-doc in her thesis advisor's laboratory where she was involved in the launch of clinical exome sequencing here at the University of Iowa's Shivinand R. Patil Cytogenetics and Molecular laboratory. Dr. Wetzel will complete a fellowship in Laboratory Genetics & Genomics this month.

Gary Gussin, Ph.D.

Director of Genetics IDGP 1975-76, ‘84-89, ‘93-95

Dr. Gussin’s interest in viruses was fueled by a basic genetics course he took in the fall of 1960 that included reading a “little” book called “Virus”, which considered the question of whether or not viruses were “alive”. He recalls that most molecular biologists at the time knew what viruses were, how they functioned, and their potential to make profound contributions to understanding gene function, whether or not anybody cared to include them in the definition of life.

When he began at Iowa in 1969, he focused on the study of gene regulation using bacteriophage lambda. He used lambda genetics and biochemical kinetics to study regulation of two promoters (PRE and PRM) that were active in the establishment and maintenance of repressor synthesis. Of all the experiments performed in the Gussin lab, he found the ones involving looking at phage plaques the most satisfying.

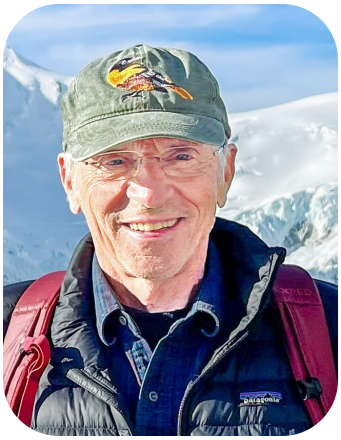
In 1967, inspired by the new President of the University, Harold Bowen, the U of I had applied for and was awarded an NSF Centers of Excellence grant. One of the areas to be strengthened by the award was genetics. Several original faculty members of the Genetics Ph.D. Program were appointed using funds from this award and an interdisciplinary genetics committee was formed. Under the auspices of this committee, 1975 became a significant year in our history.

- First, Dawson Mohler, who was then chair of the committee, suggested that unused funds could be used to support a symposium; it was decided that the focus would be Developmental Genetics. NSF provided additional funds to enable us to invite several outstanding scientists. The symposium was attended by nearly 200 participants and a review of the meeting was published in Cell.
- Second, NIH training grant programs were revised in 1974 to include Genetics as a potential target. Tom Conway, who had become chair of the interdisciplinary committee on genetics, collaborated with Dr. Gussin to write a proposal that was funded, with a starting date of July 1, 1975.
- The proposal for a Ph.D. Program in Genetics (after several revisions) was approved by the Regents, also with a starting date of July 1, 1975.

The next five years were hectic and exhilarating. The first graduate students (Bernie Possidente, Tim Shea, and Bruce Citron) arrived in fall, 1976. Several new faculty (some of whom were appointed independently by participating departments under the auspices of the NSF Centers of Excellence grant) joined the Program. Sufficient progress was made for the 1980 NIH grant renewal application to be successful. The training grant also provided funds that enabled the Genetics Program to host an exciting series of distinguished seminar speakers (in addition to geneticists hosted by the departments of Zoology, Botany, Biochemistry, and Microbiology).

After retiring in 2010, Dr. Gussin continued to teach. His classes included a few mini-courses focusing on topics in molecular genetics as they would have been taught in 1974-76 as he hoped it would be of interest for students to appreciate the foundations of their discipline. He relates how he loved teaching them and that several students and post-docs actually enrolled in or audited them, but they were obviously more of interest to geneticists of that era than to the very busy student population in general. Dr. Gussin also participated in Emeritus faculty affairs, serving on the Emeritus Faculty Council for 6 years, and as chair of the Association of Emeritus faculty for two of those years.

For Dr. Gussin, the advances in genetics and molecular biology continue to be breathtaking.



John Donelson, Ph.D.

Director of Genetics IDGP 1982-84

Soon after retiring from the University of Iowa, John Donelson became a Visiting Scientist for three months/year at the Universidade Federal do Rio Grande do Norte in Brazil, funded by a grant from the Brazilian government. This Brazilian university and its medical school are similar in size and mission to the University of Iowa. He taught biochemistry courses there and participated in infectious-disease research projects until Covid hit. Since then, he's been fully retired. He now lives on a farm outside of Iowa City and grows corn, soybeans and hay, and has a beef cow/calf herd. He enjoys traveling, photography and visiting his four adult children and their families.

Jeff Murray, MD

Director of Genetics IDGP 1993-99

In 2014 Jeff Murray took a five year leave of absence to work at the Bill and Melinda Gates Foundation. At BMGF he was the deputy director for Family Health and developed new expertise in global partnerships working with pharma and global health partners (e.g. GSK, Merck, Novartis, WHO, USAID, UNICEF, Welcome Trust) to identify novel approaches to decreasing the burden of maternal and neonatal mortality in sub-Saharan Africa and South Asia. Dr. Murray feels very fortunate to have worked with outstanding collaborators, students, support staff and others and to enjoy a far more successful career than he ever might have imagined (~600 peer reviewed papers, H index of 119).

Dr. Murray served as chair of the Interdepartmental Genetics PhD program for ten years and in parallel was the PI on the NIH T32 training grant. He was the primary thesis advisor for 28 graduate students (23 in Genetics) and served as a committee member for 25 more. Many of his students have gone on to distinguished careers in education, industry and academia. In 2021 he retired to spend more time with family, pursue interests in outdoor recreation and literature, and walk his dog.

Robert Deschenes, Ph.D.

Director of Genetics IDGP 1999-04

After leaving Iowa, Robert Deschenes served as the Chair of Biochemistry at the Medical College of Wisconsin from 2004 to 2008. In 2009, he joined the Morsani College of Medicine at the University of South Florida as chair of Molecular Medicine and Sr. Associate Dean of Graduate Education. Work in the Deschenes lab focuses on the spatial regulation of signal transduction via protein posttranslational modification. Following the discovery of a family of palmitoyltransferases (DHHC PATs) while in Iowa, the lab explored the molecular basis of disease-causing mutations *DHHC* genes. For example, they have investigated how mutations in *DHHC9* lead to X-linked intellectual disability. Other DHHC proteins have been associated with various neurological, cardiovascular, and infectious diseases. Our group has developed several tools to study protein palmitoylation, including high-throughput assays to identify DHHC inhibitors, proteomic tools for quantifying palmitoylation, cell imaging methods for studying localization, and 3D structural studies of DHHC proteins.

Debashish Bhattacharya, Ph.D.

Director of Genetics IDGP 2005-08

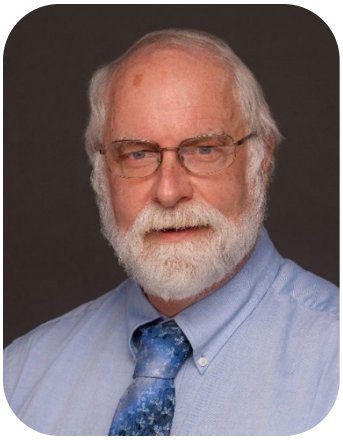
Debashish Bhattacharya fell in love with the ocean while growing up in Nova Scotia, Canada. The major goals of his lab's (dblab) research program at Rutgers University are: 1) to understand how symbioses form and are maintained, 2) to reconstruct the evolutionary history of photosynthesis on our planet, 3) to study the evolution of coral genomes and their stress response pathways using comparative and functional approaches, and 4) to develop tools for utilizing seaweed resources such as Sargassum that washes upon the shores of the Caribbean. These areas of expertise (symbiosis, evolution, algal and seaweed biology, multi-omics, and tool development) converge in ongoing, private foundation-supported projects aimed at marine resource diagnostics via his startup OceanOmics. During his time at the University of Iowa, including as Director of the Genetics Graduate Program, the dblab focused on reconstructing the tree of life, algal evolution, and ribozyme catalysis and evolution.

Daniel Eberl, Ph.D.

Director of Genetics IDGP 2009-23

After growing up on a small farm in Canada, Daniel Eberl completed undergraduate and graduate training in genetics in Canada, then moved state-side for postdoc training. He pioneered genetic and molecular dissection of the Drosophila auditory system. Using a novel electrophysiological assay, he and his lab here at University of Iowa made great strides in understanding the anatomy and molecular workings of the fly ear. He has recently expanded into using laser Doppler vibrometry to refine analysis of active mechanisms in fly hearing. In addition, he is using similar approaches to study the antennal auditory system of mosquitoes.

Throughout his time at Iowa, Dr. Eberl was a member of the Genetics Program, serving as Chair of the Graduate Affairs Committee for several years before accepting the Director role in 2009. At that time, he also shifted into the PI role of the Genetics T32 Training Grant. In 2023 he passed the Director torch to Lori Wallrath, advocating to the Graduate College to support an Associate Director, Bryan Phillips. Dr. Eberl continues as PI of the T32, along with Co-PIs, Josep Comeron and Seth Tomchik. He is proud of the training accomplishments of the program, and grateful for all the efforts of the students, faculty and staff that continue to make Genetics a great program.



Martin Burg, Ph.D. '87, Wu Lab

Professor, Biomedical Sciences and Cell and Molecular Biology
Grand Valley State University

Martin Burg was admitted to the Genetics Ph.D. program at the University of Iowa in 1980, eventually joining the laboratory of Prof. Chun-Fang Wu as one of his first Ph.D. students. Martin developed his research interests in genetic control of behavior and neuronal development, initiating studies that continued in the Wu lab for many years. In 1987, he defended his thesis entitled "Genetic and Mosaic Analysis of Mutations which Alter Nerve and Muscle Excitability in Drosophila melanogaster: Effects on Development and Behavior". Martin continued as a post-doctoral fellow with Dr. Wu before taking a postdoctoral position in 1989 with Dr. William (Bill) Pak at Purdue University. He used the electron microscopy skills he learned at the University of Iowa to characterize Drosophila mutants with vision defects. He was awarded an Individual NRSA Postdoctoral Fellowship from the National Eye Institute (1990-1993), during which time he discovered mutations in the histidine decarboxylase gene, leading to the identification of histamine as the neurotransmitter used by photoreceptors. Martin continued his work as an Asst. Research Scientist in the Pak lab from 1993-1999 and then accepted a Visiting Assistant Professorship at Grand Valley State University. He joined the faculty of the Biomedical Sciences department at GVSU in 2001, teaching courses in Human Physiology. Martin received a joint appointment in the Cell & Molecular Biology Department in 2007 and developed a CURE-type course in Drosophila Genomics in 2010. He achieved the rank of Full Professor in 2014. Martin is dedicated to mentoring undergraduate students in his research and to bringing an active research project into the classroom to help students 'learn by doing'. He was recognized for these efforts with the GVSU Distinguished Undergraduate Research Mentor Award in 2022.

Rita Shiang, Ph.D. '90, Murray Lab

Associate Professor of Human Genetics
Virginia Commonwealth University

Dr. Rita Shiang received her B.S. in Biology at the University of California, Los Angeles in 1984, her PhD from the Interdepartmental Genetics Program at the University of Iowa in 1990. Her mentor was Dr. Jeff Murray and she was his first PhD student. She did her postdoc at the University of California, Irvine with Dr. John Wasmuth and helped to identify the genes that cause hyperekplexia, achondroplasia, thanatophoric dysplasia and Huntington's Disease. She became a faculty member in the Department of Human Genetics at Virginia Commonwealth University where she continued to identify genes for Mendelian disorders (Wolfram Syndrome) and studied the function of the Treacher Collins syndrome genes. She currently works on identifying variants for craniosynostosis and cleft lip and palate. At VCU she was the Graduate Program Director for the Human Genetics program and is the-Director of VCU PREP, a postbaccalaureate program to help underrepresented students get admitted to and matriculate into PhD programs. Her hobbies include taking care of her chihuahua mix, Millie, reading, gardening, lindy hop and traveling with her husband, Andy.

Fedik Rahimov, Ph.D. '08, Murray Lab

Principal Scientist, AbbVie

Fedik Rahimov is a Principal Scientist in the Genomics Research Center at AbbVie, a global biopharmaceutical company headquartered in North Chicago, Illinois. He also leads the Human Genetics group at the company. Dr. Rahimov earned his PhD in genetics from the University of Iowa in 2008 under the mentorship of Dr. Jeff Murray. In Dr. Murray's laboratory he worked on the genetics of cleft lip and palate. He subsequently completed a postdoctoral fellowship at Boston Children's Hospital and Harvard Medical School, where he studied muscular dystrophy supported by a career development grant from the Muscular Dystrophy Association. At AbbVie, Dr. Rahimov leads the human genetics strategy across multiple therapeutic areas. His research leverages computational and statistical genetic approaches to identify genetic factors underlying common, complex diseases such as inflammatory bowel disease, Parkinson's disease, and others. The overarching goal of his work is to discover and prioritize therapeutic targets rooted in human genetics. He actively collaborates with large-scale biobank initiatives, including the UK Biobank, FinnGen, the Alliance for Genomic Discovery, All of Us, and the Emirati Genomes Project.

Colleen Campbell, Ph.D. '10 Smith Lab

Director, Genetic Counseling
University of Iowa Health Care

Colleen A. Campbell, PhD, MS, LGC, serves as Immediate Past President of the National Society of Genetic Counselors. She also serves as the Director of Genetic Counseling Operations for University of Iowa Health Care where she oversees the administration, standardization and continuous improvement of genetic counseling operations across the health system. In addition, she is a Clinical Associate Professor in the university's Department of Internal Medicine. Dr. Campbell is an experienced clinician, researcher and educator in clinical genetics, and a recognized leader in the genetic counseling community for her passionate work growing the genetic counseling workforce in underserved areas and advocacy work. She led the successful Iowa legislative effort to license genetic counselors and has been actively involved with the Access to Genetic Counselor Services Act, to improve patient access to genetic counseling services. Some highlights of her presidential term include leading the development of the 2025-2027 NSGC Strategic Plan, convening the global genetic counseling community in conversations about the global genetic counselor workforce, and leading the development of a four-prong approach to improve reimbursement for genetic counselor services.

Dr. Campbell is also Assistant Director for the Iowa Institute of Human Genetics, a statewide initiative focused on educating patients, clinicians, and researchers about how genetics can improve medical care, and on bringing precision medicine, principally by offering more and better genetic tests and genomic technologies, to the region. The focus of her work is to integrate and improve patient access to precision medicine throughout healthcare based on her unique perspective and understanding of the interactions between the lab, providers, patients, legislature, and the health care system. Through the Iowa Institute of Human Genetics she has been active in recruiting students to the genetics workforce.

As a result of her work, she was awarded the 2018 National Society of Genetic Counselors Strategic Leader Award, 2020 Sarah Lawrence College Joan H. Marks Graduate Program in Human Genetics Distinguished Alumna Award, and 2024 Saint Mary's College Distinguished Alumnae Award.

Megan Ealy, Ph.D. '11, Smith Lab

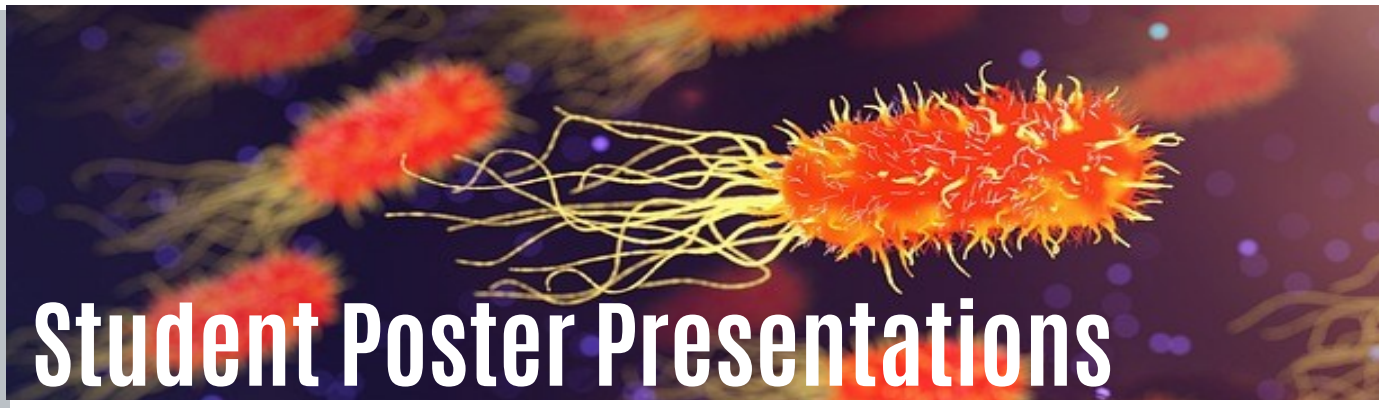
Associate Professor of Biology
Drury University

Megan Ealy graduated from the Genetics Program with her PhD in 2011, under the mentorship of Dr. Richard Smith. She accepted a post-doctoral position in the laboratory of Dr. Stefan Heller at Stanford University from 2012-2017. In 2017, Dr. Ealy transitioned to a tenure-track faculty position at the private liberal arts institution, Drury University in Springfield, Missouri. She is currently an Associate Professor of Biology teaching genetics, developmental biology, and pathophysiology and runs an undergraduate research laboratory focused on inner ear neuron development.

Katie Weibrecht, Ph.D. '17, Sheffield Lab

Manager of Laboratory Policy
Avalon Healthcare Solutions

Katie Weibrecht did her PhD in Dr. Val Sheffield's lab, working on a BBS gene. She did a lot of protein-protein interaction work in her PhD and was able to apply that protein-protein interaction work in a translational pharmacy project on sepsis during her time as a postdoc in the College of Pharmacy, working with Dr. Ethan Anderson. However, throughout her PhD and postdoc, one thing rang true- while she loved doing science, she also really enjoyed writing about science and helping others write about the science they had done. While she originally hoped to find a position as a writer/editor for a department at a university, Dr. Weibrecht ultimately branched out into industry, finding a position as a medical writer. She has been with this company since completing her postdoc four years ago, moving from a medical writer to a manager, employing both medical writing and medical editing to develop and maintain a library of science based medical policies to help people get the right care at the right time.



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1. Arhgap29 regulates epithelial morphology and contractility during murine palatogenesis

E. Adelizzi[1,2], L. Rhea[2], M. Dunnwald[1,2]

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ARHGAP29, Rho GTPase Activating Protein (GAP) 29, functions in the cyclical regulation of RhoA, a major modulator of the actomyosin cytoskeleton. Arhgap29 is expressed in many cell types, including epithelial cells, where it promotes the inactivation of RhoA. Arhgap29 has been implicated in a range of tissue morphogenesis events, including palatogenesis, however what role Arhgap29 plays in different cell types during this process remains unknown. Using an ectoderm-cell specific mouse knockout of Arhgap29, we found a significant, yet incompletely penetrant cleft palate in mutants compared to wild-type controls. Phenotypic characterization revealed a thickened oral epithelium at the tip of the palatal shelves which we found was due to an increase in cell size and irregular morphology. Further analysis revealed a disorganized lingual epithelium and an increase in epithelial cells positive for phospho-Myosin regulatory light chain (pMRLC) and a-smooth muscle actin, two markers of actomyosin contractility. Collectively, these data suggest that a defect in the actomyosin cytoskeleton may contribute to cleft palate in Arhgap29 mutants. To further investigate the mechanism by which Arhgap29 contributes to cleft palate and what effect the observed cytoskeletal changes may have on cell stiffness, we performed atomic force microscopy (AFM) *in vivo*. Preliminary AFM experiments on wild-type embryos revealed a significant increase in palatal stiffness as palatogenesis progresses, uncovering novel insights into the regulation of tissue stiffness during palatogenesis.

2. Dissecting the Gene Regulatory Network of a Novel Acquired Stress Resistance Trait, its Evolution and Fitness Consequence in a Yeast Pathogen

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A key challenge in microbial pathogenesis is understanding how stress regulatory networks evolve to link unrelated environmental cues, allowing organisms to anticipate and survive future stress. We study Candida glabrata, an opportunistic yeast pathogen that exhibits a novel phosphate starvation-induced Acquired Stress Resistance (ASR) to hydrogen peroxide (H_2O_2), a trait absent in its non-pathogenic relative S. cerevisiae. While CTA1, a catalase gene, is necessary and sufficient for this ASR, the gene regulatory network (GRN) linking phosphate limitation to oxidative stress tolerance remains incompletely mapped. Using a Hermes transposon insertion library combined with a CTA1 -GFP reporter system, we identified mutant populations with impaired CTA1 induction under either phosphate starvation or H_2O_2 oxidative stress. Notably, loss of the oxidative stress transcription factor SKN7 abolishes CTA1-GFP induction, validating our genetic screen. Ongoing efforts include Tn-seq coupled with machine learning to systematically identify regulators differentiating canonical oxidative stress response from ASR. We will further integrate comparative genomics and CRISPRi knockdowns to trace the evolutionary divergence of ASR networks between *C. glabrata* and *S. cerevisiae*. Our model provides a tractable system for dissecting GRN rewiring, revealing how environmental stress anticipation evolves and contributes to pathogenic adaptation.

3. Evolution of Pioneer Factor Ability Between Orthologous Transcription Factors

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Pioneer Factors (PFs) are a special class of Transcription Factors (TFs) that are defined by their ability to access closed chromatin and open it up to allow for transcription. In eukaryotes, PFs are recognized for their crucial role, especially in specifying cell fate during development. The role of PF in other biological processes outside development is still poorly understood. Another limitation in our understanding of PF activity is whether it changes during evolution. Existing studies implicitly treat PF activity as a fixed property of a TF. However, genome-wide screens have shown that TFs vary quantitatively in their PF activities, suggesting the possibility that PF activities are evolvable and could diverge between orthologous TFs. If true, this would have profound implications for the evolution of gene regulation and the consequent phenotypes. In preliminary studies, our lab has found strong evidence supporting such variation in PF activity between two orthologous TFs in related yeasts. In the baker's yeast *S. cerevisiae*, the basic Helix-Loop-Helix TF, Pho4 (ScPho4), fails to induce the reporter gene when the promoter contains only a nucleosome-occluded motif. By contrast, the orthologous Pho4 in the related pathogenic yeast, *C. glabrata* (CgPho4), is capable of inducing the same mutant promoter. The central hypothesis is that the difference in the PF ability of divergent Pho4 orthologs is due to CgPho4's ability to bind nucleosome occluded sites and recruit chromatin remodelers and these mechanisms stem from differences in protein sequences. Chromatin-Immunoprecipitation (ChIP) reveals that CgPho4 binds to the Pho5 promoter in both the wild type and mutant promoter context. This research aims to determine CgPho4's ability to bind nucleosome-occluded motifs genome-wide and investigate the subsequent changes in nucleosome occupancy and gene expression. Expansion of this work to other Pho4 orthologs will contribute to our knowledge of how PF ability evolves.

Poster Presentations

4. 16p11.2 Microdeletion Induces Sex-Specific Defects in Placental Development in Mice

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Neurodevelopmental disorders (NDDs) are extremely common. The prenatal environment significantly influences NDD risk which may include how NDD risk genes affect placental function. However, placental disruptions in mouse models of NDD risk genes remain unexplored. Our objective was to investigate placental function in the 16p11.2 microdeletion model (16p del). 16p del in humans is associated with NDDs. Mice modeling this deletion display sex-specific behavioral phenotypes relevant to NDDs, mirroring the male bias observed in human NDDs. We hypothesized that 16p del would alter placental growth and function with more severe phenotypes observed in male placentas. To test this, wildtype female mice were bred with hemizygous 16p del males, and placental weight, fetal weight, placental morphology, and placental gene expression were assessed.16p del males showed 50% expression level in all three 16p del genes at three embryonic timepoints. Unexpectedly, embryonic day 16 (E16) 16p del female placentas exhibited comparable expression to wildtype *Mapk3*, a 16p del gene that is a major regulator of placental development. Male 16p del placental mass was increased at E16 and E18 compared to litter-matched wildtypes; females showed no group differences. This increased 16p del male placental weight may have been related to increased E16 decidual proportion we found in 16p del males, a phenotype typically associated with poor placental perfusion. Further evidence of poor placental perfusion was seen at E18 as 16p del males had a significant decrease in total placental sinusoidal area. There were no group differences in female placental morphology. Furthermore, 16p del females showed mid-gestation promotion of angiogenic and growth pathways expression in the placenta, conversely 16p del males showed downregulation of these pathways in late-gestation. The sex-specific placental abnormalities in 16p del mice likely impact postnatal development and may contribute to early developmental origins of male bias in NDDs.

5. A New Timeline for Language Evolution: Insights from a Novel Complex Trait Dating Approach

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Minor genetic changes have produced profound differences in cognitive abilities between humans and our closest relatives, particularly in language. Despite decades of research, ranging from single-gene studies to broader evolutionary analyses, key questions about the genomic foundations of human language have persisted, including which sequences are involved, how they evolved, and whether similar changes occur in other vocal learning species. Here we provide the first evidence directly linking rapidly evolved genomic regions to language abilities in current-day humans. Through extensive analysis of 65 million years of evolutionary events in over 30,000 individuals, we demonstrate that Human Ancestor Quickly Evolved Regions (HAQERs) - sequences that rapidly accumulated mutations after the human-chimpanzee split - specifically influence language but not general cognition. These regions evolved to shape language development by altering binding of Forkhead domain transcription factors, including *FOXP2*. Strikingly, language-associated HAQER variants show higher prevalence in Neanderthals than modern humans, have been stable throughout recent human history, and show evidence of convergent evolution across other mammalian vocal learners. An unexpected pattern of balancing selection acting on these apparently beneficial alleles is explained by their pleiotropic effects on prenatal brain development contributing to birth complications, reflecting an evolutionary trade-off between language capability and reproductive fitness. By developing the Evolutionary Stratified-Polygenic Score analysis, we show that language capabilities likely emerged before the human-Neanderthal split - far earlier than previously thought. Our findings establish the first direct link between ancient genomic divergence and present-day variation in language abilities, while revealing how evolutionary constraints continue to shape human cognitive development.

6. Identification of expression modulating variants that influence transcription of genes associated with ophthalmic diseases.

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For 20 years, Genome Wide Association Studies (GWASs) have been used to identify genetic loci associated with complex diseases, including for vision disorders such as glaucoma and age-related macular degeneration. Despite these successes, a limitation of GWASs is that they do not distinguish variations that have a role in increasing disease risk from polymorphisms that merely are associated with disease risk due to linkage disequilibrium. Coupled with the fact that many variants in genome-wide significant loci do not yield protein-coding changes, increasing the difficulty of understanding the molecular mechanisms used by genetic loci to affect disease risk. Therefore, the two major questions asked by researchers are (1) *Which* polymorphisms truly cause complex diseases, and (2) *How* do these polymorphisms alter disease risk?

We hypothesize that many variants in genome-wide significant loci modulate disease risk through altering gene expression, namely through increasing or decreasing transcription of nearby genes. Using a massively parallel reporter assay developed by Kalita et. al. called Biallelic Targeted Active Regulatory Region Sequencing (BiT-STARR-Seq), we have identified several putative expression modulating variants (emVars) that are associated with primary open-angle glaucoma and exfoliation syndrome. Many of these emVars are within predicted transcription factor binding sites, and this may affect transcription rates. We also have expanded our analyses to include variants associated with neovascular AMD. In the future, discovering how these loci affect disease risk through allele-specific transcriptional regulation can reveal therapeutic targets for delaying or even preventing blindness caused by these conditions.

7. Factors that control synaptic homeostasis maintenance also support sleep

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The brain and all the tissues that it excites govern animal behavior. To keep behaviors controlled, nervous system output needs to stay within physiological bounds. At the levels of neurons, synapses, or circuits, this means that homeostatic forms of regulation are needed. One model synapse where this form of regulation is studied is the *Drosophila* neuromuscular junction (NMJ). Our lab and others have found that at the NMJ, a small group of factors has been identified as critical for the long-term maintenance of synaptic homeostasis. These factors can respond to acute challenges to the synapse but cannot maintain synaptic homeostasis with long-term challenges.

The idea of homeostatic regulation has been extended to specific behaviors like sleep. The synaptic homeostasis hypothesis (SHY) in the sleep field posits that sleep is used as an off-line period to restore the brain's capacity for plasticity that is needed during wakefulness. If the general concept of SHY is correct, perturbations that disrupt sleep would affect synaptic homeostasis, and likewise, disruptions to synaptic homeostasis would also affect sleep. Previous research has shown that disruptions in a subset of canonical sleep genes can also cause disruptions to synaptic homeostasis. Here we find that loss-of-function mutations in some of the synaptic homeostasis maintenance factors at the larval NMJ also show abnormal sleep phenotypes in adult *Drosophila*. When compared to wild type, disruptions to the genes RyR (the Ryanodine Receptor), Src64B (Src family kinase), Csp (cystine string protein), and stumps (FGFR signal transduction) all show disruptions to the patterns seen in *Drosophila* sleep. This is seen in a decrease in the daytime sleep with the nighttime sleep being unaffected. We will continue to test the synaptic homeostasis maintenance factors to determine which factors are important for sleep and how their synaptic functions might inform their roles in sleep.

8. Med13 and Med13L: Critical Redundant Players in Basal Cardiac Function and Gene Expression

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Background: Previous studies have linked mutations in the Mediator complex, specifically Mediator 13 (Med13) and Mediator 13-like (Med13L), with both congenital heart defects and cardiovascular diseases. Med13 and Med13L are mutually exclusive paralogs within the kinase submodule of the Mediator complex that have been shown to have partially redundant functions in embryonic development and transcription, but their combined roles have not been investigated in the adult heart. We investigated the critical yet redundant roles of Med13 and Med13L in adult murine cardiomyocytes for basal cardiac function.

Methods: We generated an inducible Med13 and Med13L cardiomyocyte-specific knockout mouse model to investigate Med13 and Med13L regulation of cardiac function and transcription. We performed RNAseq on mice four weeks after the start of tamoxifen to identify changes in gene expression. Differentially expressed genes were compared across cardiac knockouts of Med13/13L, Med13, Med12, Med1, and Med30 elucidating similar mechanisms of cardiac dysfunction.

Results: Med13/13L knockout resulted in decreased cardiac function leading to lethal heart failure in a median timeframe of 6 weeks from the start of tamoxifen. There is significant gene dysregulation after Med13/13L knockout with similar gene dysregulation of fibrotic pathways and calcium handling across Mediator cardiac knockouts.

Conclusions: Med13 and Med13L function partially redundantly within the heart to maintain basal cardiac function and transcription, as well as redundancies within cardiac phenotypes related to mediator complex disruptions.

9. Studying the Role of the Membrane Attack Complex (MAC) in Age-Related Macular Degeneration (AMD)

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Age-related macular degeneration (AMD) is a leading cause of irreversible vision loss in adults over 50. Many of these patients have early AMD, characterized clinically by central visual distortions and drusen deposition, and histopathologically by death of endothelial cells of the choriocapillaris, among other findings [1]. There are no treatments for early AMD, and progression to later stages can result in blindness. Although age is by far the most important risk factor, a common polymorphism in the *CFH* gene can increase disease risk up to 7-fold in homozygotes, and at least one copy of this polymorphism is seen in 50% of AMD patients [2,3]. Despite high disease prevalence and well-described risk factors, the inciting pathophysiology of AMD remains poorly understood. The *CFH* gene encodes complement factor H, a regulator of the innate immune system's complement cascade, suggesting its involvement. Furthermore, we see deposition of terminal complement, or the membrane attack complex (MAC), in the choriocapillaris of patients with early AMD. Accordingly, we are interested in learning about the relationship between MAC deposition and the death of choriocapillaris endothelial cells in early AMD. To look for differences in gene expression of MAC-bound cells, we performed single-cell RNA-seq on aged human donor choroid with barcoded anti-MAC antibodies. Though we were unable to detect MAC in this experiment, we found we were able to detect other surface proteins (including complement regulators) in our human donor tissue. We then developed an iPSC-derived endothelial cell model, and demonstrated that we can detect MAC deposition via immunocytochemistry on cells derived from patients with both high- and low-risk AMD genotypes. We plan to use this data to guide future experiments to learn more about what happens to endothelial cells after MAC deposition, and how it relates to their death in early AMD.

1. Mullins RF, Flamme-Wiese MJ, Navratil EM, Boese EA, Varzavand K, Riker MJ, Wang K, Stone EM, Tucker BA. Ghost vessels in the eye: Cell free choriocapillaris domains in atrophic age-related macular degeneration. Exp Eye Res. 2024 Nov;248:110128. doi: 10.1016/j.exer.2024.110128. Epub 2024 Oct 16. PMID: 39419369; PMCID: PMC11532014.
2. Klein RJ, Zeiss C, Chew EY, Tsai JY, Sackler RS, Haynes C, Henning AK, SanGiovanni JP, Mane SM, Mayne ST, Bracken MB, Ferris FL, Ott J, Barnstable C, Hoh J. Complement factor H polymorphism in age-related macular degeneration. Science. 2005 Apr 15;308(5720):385-9. doi: 10.1126/science.1109557. Epub 2005 Mar 10. PMID: 15761122; PMCID: PMC1512523.
3. Haines JL, Hauser MA, Schmidt S, Scott WK, Olson LM, Gallins P, Spencer KL, Kwan SY, Noureddine M, Gilbert JR, Schnetz-Boutaud N, Agarwal A, Postel EA, Pericak-Vance MA. Complement factor H variant increases the risk of age-related macular degeneration. Science. 2005 Apr 15;308(5720):419-21. doi: 10.1126/science.1110359. Epub 2005 Mar 10. PMID: 15761120.

10. Investigating protein synthesis and axon regeneration in a dorsal root ganglion model of Charcot-Marie-Tooth disease

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Charcot-Marie-Tooth disease (CMT) describes a group of inherited peripheral neuropathies. Despite extensive research into the genetic underpinnings of CMT, the exact cause of the disease remains unknown. A common feature in certain instances of CMT is mutations in aminoacyl-tRNA synthetases (ARS), suggesting that a defect in protein synthesis may underly the unclear etiology of CMT. However, the potential mechanism(s) through which ARS mutations cause the sensory and muscular phenotypes observed in CMT patients remain unknown. To address this gap in knowledge, I developed an in-vitro model for the effects of mutant ARSs in sensory neurons. This model utilizes neurons cultured from the dorsal root ganglion (DRG) of embryonic day 14 mice and genetically encoded fluorescent reporters to visualize newly synthesized proteins in live neurons. Once grown, DRGs undergo a chemical axotomy to remove axons while maintaining cell integrity. The axons are subsequently regenerated. This approach allows me to assess axon regeneration dynamics in a mutant ARS background. In this DRG culture model, lentiviral expression of a CMT associated mutant tyrosyl-tRNA synthetase (G41R-YARS) induces dose-dependent axon degeneration and cell death when compared to wildtype and empty vector controls. Further, I observed a significant decline in neuronal protein synthesis with G41R-YARS. Interestingly, axon regeneration is unaffected, despite it being a very synthesis dependent process. These findings demonstrate that expression of G41R-YARS in DRG cultures induces axon degeneration, mirroring a prominent event in many CMT neuropathies, and further causes a protein synthesis defect. The unaffected regeneration dynamics suggest that this process may activate certain factors that incidentally confer resistance to the synthesis defect. Identification of these factors and the pathways they interact with may uncover new avenues for treatment of CMT.

11. Exploring mRNA therapeutics in Niemann-Pick type C1 disease

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Niemann-Pick type C1 disease is a recessive monogenic disorder caused by loss-of-function mutations in the NPC1 gene, which makes restoring NPC1 gene function an attractive therapeutic approach. While gene therapy aims to stably and permanently reintroduce the normal NPC1 gene, mRNA can also be used to transiently reintroduce loss-of-function gene products. Here we evaluated the potential of lipid particle mRNA therapeutics for treating Niemann-Pick C1. Using NPC1-null cells, we show that optimized lipid particles containing *Npc1* mRNA effectively restore NPC1 protein, which in turn corrects primary and secondary cellular defects. Furthermore, our findings indicate these lipid particles efficiently transduce the liver in an *Npc1*-null mouse model, resulting in significant restoration of NPC1 protein levels and correction of downstream cellular defects. Ongoing analysis will further optimize the lipid particles for enhanced expression and neurological correction. It is our expectation that mRNA therapeutics have the potential to treat rare monogenic disorders such as Niemann-Pick type C1.

12. Modeling MEK-inhibitor Associated Retinopathy *in vitro* using human induced-pluripotent stem cell-derived retinal pigment epithelial cells

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MEK inhibitors are used to treat patients with cancers carrying mutations in the MAPK/ERK pathway. Many treated patients develop a side-effect known as MEK-inhibitor Associated Retinopathy (MEKAR), which can decrease visual acuity and quality of life. The mechanism of this retinopathy is unknown. The retinal pigment epithelium (RPE) is a monolayer of cells below the retina which contributes to the blood-retina barrier and is critical to maintaining retinal homeostasis. We hypothesized that the cause of MEKAR is drug-induced dysfunction of the RPE. To test this, we used an *in vitro* model of stem cell-derived RPE cells.

We treated mature, human induced-pluripotent stem cell-derived RPE cells (hiPSC-RPE) with selumetinib and measured impacts on RPE-specific function, cell structure, and gene expression. The functional assays evaluated the ability of the hiPSC-RPE to maintain a membrane barrier and phagocytose and internalize photoreceptor outer segments. These assays were carried out using transepithelial electrical resistance (TEER) measurements and Western blot, respectively. hiPSC-RPE cell structure was visualized with scanning and transmission electron microscopy. Additionally, we assessed the effect on gene expression using bulk RNA-sequencing.

Selumetinib increases the ability of hiPSC-RPE to phagocytose rod outer segments and has no effect of the ability of hiPSC-RPE to maintain membrane integrity. We are currently in the process of collecting data examining the effect of selumetinib on hiPSC-RPE cell structure and gene expression.

The clinically used MEK inhibitor, selumetinib, increased the phagocytic activity of these cells which may explain the drug-associate retinopathy. Patients who are susceptible to this retinopathy may be candidates for receiving concurrent therapy with selumetinib to target this mechanism.

13. Utilizing Network based stratification of Known Microdeletion and Duplication syndromes to improve Interpretation of Variants of Unknown Significance

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Among approximately 67 million individuals that are diagnosed with neurodevelopmental disorders (NDD) in USA, 10.5% have intellectual disabilities (ID) leading to major socioeconomic issues. However, comprehensive computational genetic diagnostic methods utilizing both genotype and phenotype information are not yet fully studied in NDD and ID populations for identifying and predicting pathological genetic variants.

Phenotypic heterogeneity is common in patients with ID and is influenced by different pathogenic copy number variants (CNVs). The increase in NDD and ID genomic testing via chromosomal microarrays (CMA) and whole genome sequencing (WGS) has led to the identification of an increasing number of "variants of unknown significance (VUS)". In this project, we hypothesize that specific protein-protein interaction networks are common between established microdeletion / microduplication syndrome (MMS) syndromes and VUS CNVs in patients with similar phenotypes.

To investigate this hypothesis, we used CMA data from approximately 2400 pediatric patients seen at the University of Iowa. We analyzed the detected VUS CNVs from our database in the context of known MMS PPI disease networks and phenotypes. We adopted network-based stratification (NBS), Human Phenotype Ontology (HPO) and pathway analyses to first identify the underlying significant ID PPI networks, genotype and phenotype based patient clusters and subsequently reclassify the VUS CNVs based on their stratification with known CNVs.

Our preliminary experiments using these clustering procedures were successful in genotypically quantifying and phenotypically defining the functional interacting genetic variants in the context of known MMS CNVs for interpreting and re-classifying the detected VUS CNVs. This methodology allows for candidate genetic variant identification which will then be functionally tested utilizing Gal4-UAS RNAi and T-maze assays in the Drosophila melanogaster model system. This will aid in identifying novel genetic variants in cognition and memory and help in VUS CNV interpretation for better diagnostic yield in NDD and ID populations.

14. Mild elevation in intraocular pressure facilitates T cell-mediated retinal ganglion cell loss

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Glaucoma is a neurodegenerative disease that causes loss of retinal ganglion cells (RGC) and loss of vision. We have shown that adoptive transfer of CD3+ cells from glaucomatous animals into healthy recipients results in progressive RGC loss, suggesting a functional role of adaptive immunity in the pathology of the disease. A crucial first step in this process is the extravasation of T cells into the immune-privileged retina. This study sought to determine if elevated intraocular pressure (EIOP) facilitates extravasation using donor mice with a fixed T cell receptor against GFP (*Just EGFP Death Inducing*, JEDI) and Thy1-GFP+ recipient mice that express GFP in a subset of RGC.

Mild EIOP was induced in recipient mice through intracameral injection of Ad5.Myoc^{Y437H} (n=12). Controls received injections of Ad5.empty (n=11-12/per group) and IOP was monitored by rebound tonometry. GFP+ cells were imaged by fundoscopy for 7 weeks. 4 weeks following injection, splenocytes from donor mice were transferred into recipient mice. At week 4.5 following injection, one group received an intraperitoneal injection of lipopolysaccharide (LPS). At the conclusion of the experiment, retinas were collected and imaged.

Following injection and adoptive transfer, all mice experienced RGC loss on fundoscopy. Losses are similar in naive mice (4.46%), transfer only animals (2.83%), and LPS-injected mice (4.5%). However, EIOP animals lost significantly more RGC (18.43%, p=0.0097). In these animals RGC loss was noticeable in the central retina. Additionally, animals with EIOP saw increased VCAM-1 labeling, particularly in the microvasculature.

Slight EIOP facilitates T cell-mediated degradation of RGC. LPS-mediated weakening of the blood-retina barrier by itself is insufficient to achieve RGC loss, suggesting that additional IOP-mediated mechanisms are required to overcome retinal immune privilege. Subsequently, increased VCAM-1 expression in EIOP retinas may be a mechanism by which T cells can efficiently extravasate and influence RGC degradation.

15. Using DNA Methylation and Genetic Variation to Investigate Phenotypic Heterogeneity

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Background: Phenotypic heterogeneity, or variance of comorbidities for the same disease, is found in many genetic diseases, highlighting the gap in our knowledge of modifiers of genetic disease. DNA methylation, a known regulator of disease, has great influence on gene expression but is not accounted for in traditional gene burden analyses (GBA), which is the standard used to measure phenotypic heterogeneity (PH) in genetic diseases. We propose the first GBA that includes genetic information alongside DNA methylation.

Methods: To combine both DNA methylation and variation, we created two scores – Burden Estimate from Weighted Integration of Site-specific Epigenetic Changes (BeWISE) and Burden Estimate from Modified and Associated Genetic Change (BeMAGIC). These two scores combined create the MagicWise Index; a Phred-scaled score of predicted deleteriousness. To minimize noise in the data, we created a novel weighting and filtering scheme at both the nucleotide and gene level.

Results: After filtering, our MagicWise index encompassed 12,007 genes and contained ~65,000 CpG sites associated with promoter, enhancer, and insulator regions. Applied to several different datasets with a wide variety of etiologies, the MagicWise Index identified known causes of genetic diseases, as well as highlighted putative causes of phenotypic heterogeneity.

Conclusions: The MagicWise Index delineates between cases and controls, identifies causative genetic modifiers, and prioritizes biologically significant genes. The MagicWise Index has wide utility for many different genetic diseases, and aims to aid in the holistic, comprehensive analysis of genetic diseases.

16. Sialoglycoconjugate distribution and partial identities in human choroid, RPE, retina, and basal laminar deposit.

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Sialic acids are negatively changed, 9 carbon sugars that perform essential roles in cell-cell adhesion, migration, complement regulation, and immune modulation, each of which may be altered during age related macular degeneration. The purpose of this study was to characterize sialoglycans of the human choroid, RPE, and photoreceptors, including their distributions and partial molecular identities.

This study utilized lectin histochemistry and immunofluorescence on a set of 45 fixed and cryopreserved human donor eyes to investigate sialoglycans. Lectins EBL and MAL-II were used to visualize a-2,6 and a-2,3 sialic acids, respectively. Antibodies were used to investigate colocalization of sialic acids with structures and proteins of interest. Neuraminidase enzyme was used to remove sialic acids, followed by histochemistry with a battery of 37 lectins to reveal penultimate carbohydrate moieties.

a-2,3 and a-2,6 sialic acids have different distributions in the submacular choroid. a-2,3 sialic acid is found in choriocapillaris endothelial cell membranes, while a-2,6 sialic acid shows robust labeling of intercapillary pillars (i.e., the extracellular matrix between choriocapillaris vessels). Both conformations of sialic acid are also found in basal laminar deposits, histological features of macular degeneration, and in subretinal fibrosis in neovascular AMD. Lectins that recognize β-galactose, N-acetyllactosamine, galactose (b-1,3) N-acetylgalactosamine, and a - or b- N-acetylgalactosamine showed increased reactivity in basal laminar deposits after neuraminidase treatment. EBL displays overlap with anti-MCT-1, a marker of RPE microvilli, while a-2,3 sialic acid is internal to the microvilli, in the subretinal space. The fovea displays distinct sialic acid distribution. Foveal, but not extrafoveal, cone inner segments exhibit MAL-II labeling. Similarly, the extrafoveal external limiting membrane does not contain EBL binding sites, which are present in the foveal ELM. This study adds to our understanding of the sialic acid glycan composition of the macula, including topographic distribution in the fovea and changes in aging and AMD.

17. ANALYSIS OF RAD-51 SEPARATION OF FUNCTION ALLELE SUGGESTS DIVERENCE OF THE SYNTHESIS-DEPENDENT STRAND ANNEALING AND DOUBLE HOLLIDAY JUNCTION PATHWAYS PRIOR TO RAD-51 FILAMENT DISASSEMBLY

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DNA double-strand breaks (DSBs) are formed during meiosis, so their repair in the homologous recombination (HR) pathway will lead to crossover formation, which is essential for proper chromosome segregation. HR contains two sub pathways: double Holliday junction (dHJ) that generates crossovers, and synthesis dependent strand annealing (SDSA) that creates non-crossovers. RAD-51, an essential protein for the formation of all products of HR, assembles on the processed DSB, allowing the invasion of the ssDNA into an unbroken region of homology. After successful invasion, RAD-51 is removed by RAD-54.L to initiate repair. Here we investigate a separation of function allele of *rad-51*, *rad-51::FLAG,* as well as two other RAD-51 alleles: wild type-like *rad-51::degron* and impaired *GFP::rad-51*. *rad-51::FLAG* displays slowed repair kinetics, resulting in an accumulation of RAD-51 foci. *rad-51::FLAG* worms also exhibit DSB checkpoint activation, but to a less extant than that of *rad-51* null mutants. In a proximity ligation assay, RAD-54.L and RAD-51 show enriched colocalization in *rad-51::FLAG* germlines (but not in *rad-51::degron*), consistent with stalling during the strand invasion step in HR. The defects in RAD-51 disassembly in *rad-51::FLAG* mutants lead to formation of chromosomal fragments, similar in their magnitude to ones observed in *rad-51* or *rad-54.L* null mutants. However, *rad-51::FLAG* mutants, (unlike a *rad-51* null, *GFP::rad-51* or *rad-54.L* null mutants), displayed no defects in the formation of crossover designated sites (via GFP::COSA-1 localization). Given that *rad-51::FLAG* worms show checkpoint activation and chromosomal fragments, these results suggest that crossover repair concludes normally, while the non-crossover pathway is perturbed. This is strikingly different from *rad-51::degron* and *GFP::rad-51* strains, which are proficient or deficient in both pathways, respectively. These results suggest that non-crossovers vs crossovers have distinct recombination intermediates and diverge prior to RAD-51 disassembly.

18. Regulation and dynamics of intercalated disc transcriptomes

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Intercalated discs (ICDs) are complex and essential structures in the heart. ICDs dysregulation is implicated in heart diseases. In addition to their known structural roles, ICDs likely also serve as hubs for local protein synthesis in Cardiomyocytes (CMs). Studies have found enhanced local translation at ICDs, but only a few ICD-enriched mRNAs are known (e.g., Dsp). Overall, spatial transcript localization and local translation have been shown to be biologically important in many cells and organisms, and dysregulation of these processes are detrimental to cell function. Despite evidence for both ICD localized RNAs and local translation in CMs, no transcriptome-wide unbiased approaches have been used to profile RNAs at ICDs, and few efforts have been made to identify RNA-binding proteins (RBPs) that mediate RNA localization to ICDs.

To begin. We captured >20,000 ICDs from mouse heart tissues, isolated RNAs, and performed RNA-seq. We identified hundreds of unique mRNAs significantly enriched at the ICD, with enriched mRNAs harboring sequence motifs known to bind specific RBPs implicated in heart disease. specific RBPs, with some having already been implicated in heart disease. Gene ontology analyses indicated that many of the ICD-enriched mRNAs encode proteins involved in microtubule stability/transport, RNA metabolism, and cell-cell adhesion. Furthermore, the ICD-localized transcripts are 1) **enriched for mRNAs that are known to be trafficked to cell leading edges in APC-dependent fashion**, and 2) **mRNAs that have been previously found to be enriched in stress granules**, providing overall support for the validity of our ICD-seq method and resulting data. Notably, in tangential work, we identified >100 ICD-localized RBPs. Pilot studies applying ICD-seq to nonfailing human heart tissues revealed strong correlation to mouse data, supporting conserved processes and functional importance.

Future work will define ICD transcriptomes in humans and mice across different conditions of cardiac stress and disease to understand the translational significance of ICD RNA regulation in addition to assess how inducible CM-specific loss of Apc or Kif1c in mice alters mRNA localization to ICDs and cardiac structure and function. We will induce CM-specific knockout (KO) of Apc and Kif1c, perform ICD-seq on heart samples, and verify if "top hit" candidate mRNAs show de-enrichment from ICDs after KO. We will also determine the effects of inducible CM-specific Apc or Kif1c KO in adult mice.

IMPACT: This work will deliver key resources to better understand this important biological realm of local RNA regulation that remains largely underexplored in heart. The comprehensive ICD RNA profiles of normal and diseased hearts will stimulate studies aimed at further defining the precise mechanisms that control RNA localization and translation at ICDs and understanding their disease relevance, with prospects to guide

19. A novel mouse model to study rapid photoreceptor degeneration in vision disorders

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In the vision disorder Retinitis Pigmentosa (RP) there is a sequence of cell death in the retina leading to vision loss. Initially, there is death of the rod photoreceptors followed by the death of the cone photoreceptors. Mutations in 80+ genes cause autosomal dominant RP, including *SNRNP200* which encodes an RNA helicase component of the U5 small nuclear ribonucleoprotein (snRNP) complex, which is essential for pre-mRNA splicing. How mutations in *SNRNP200* cause RP is unknown.

To investigate the pathogenic mechanisms of *SNRNP200*-associated RP, we generated a conditional knockout (cKO) mouse model in which *Snrnp200* was specifically knocked out in the rod photoreceptors. Heterozygotes showed no retinal abnormalities as assessed by electroretinograms (ERGs). By contrast, homozygous deletion of *Snrnp200* in the retina resulted in rapid photoreceptor degeneration. By postnatal day 30, the ERGs showed an absence of rod photoreceptor response, while cones exhibited only reduced activity, despite the presence of wild-type *Snrnp200* in the cones. By six weeks of age, the ERG response of both rod and cone photoreceptors were absent. Consistent with these findings, Optical Coherence Tomography (OCT) showed a rapid loss of the photoreceptor layer over the same timeframe.

Our findings demonstrated that this novel mouse model recapitulated aspects of human RP. In particular, the death of rod photoreceptors triggered the death of cone photoreceptors. Unlike many existing models where degeneration occurred over months, *Snrnp200* cKO mice exhibited complete loss of rod photoreceptors by one month of age. This rapid and severe retinal degeneration offers a unique opportunity to study the earliest molecular events leading to photoreceptor death, making it a powerful tool for dissecting splicing-related disease mechanisms. In addition, the model has the potential to test time-sensitive therapeutic interventions to prevent the secondary death of the cones with the results being broadly applicable to other types of RP.

20. Chemical Molecule Screening by Luciferase Reporter Construct for Inhibitors of *CDR1* Expression in Azole-Resistant Pathogenic *Candida glabrata*

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Antimicrobials are critical for modern medicine; however, a serious problem has arisen: antimicrobial resistance. One such drug-resistant organism is *Candida glabrata*. *C. glabrata* is an opportunistic yeast which can cause infections in immunocompromised patients. These infections are often treated with azole drugs, such as fluconazole. Azoles target the ergosterol biosynthesis pathway, specifically inhibiting an enzyme encoded by the *ERG11* gene. Azole resistant isolates are often found to have a hyperactive allele of a transcription factor encoded by *PDR1*, such as *PDR1-D1082G*. Pdr1 stimulates the expression of a membrane transporter protein called Cdr1 that is thought to act as a drug pump, exporting azole drugs out of the cell. Strains containing the *PDR1-D1082G* hyperactive allele show a tenfold increase in Pdr1 activity compared to wild-type, increasing the azole resistance of the hyperactive strain. In order to reduce azole resistance, we aim to identify small molecule compounds which will reduce the activity of the hyperactive isoforms of Pdr1. We will do this by high-throughput screening of a library of compounds with a hyperactive *PDR1-D1082G* strain of *C. glabrata,* which contains a luciferase reporter gene under the control of the *CDR1* promoter. If a compound reduces the activity of Pdr1 and thus reduces *CDR1* induction, a decrease in luciferase signal will be observed. We have screened 52K small molecule compounds from the library and have identified several compounds which reduce *CDR1* induction in our high-throughput screen and in low-throughput secondary testing. Additionally, these compounds have reduced fluconazole-mediated induction of native *CDR1* expression and lower the concentration of fluconazole necessary to inhibit growth. We will next determine if these compounds could be used in vivo to treat *C. glabrata* infections. We will also be determining how these compounds effect the activity and regulation of Pdr1.

Alumni in Attendance

First Name	Last Name	Defense Year	PhD Advisor
Danielle	Beekman	2013	John Logsdon
Kimberly	Bekas	2022	Bryan Phillips
Stanton	Berberich	1989	Dr. Stoltzfus
Martin	Burg	1987	Chun-Fang Wu
Colleen	Campbell	2010	Richard Smith
Emily	Chen	1988	Gordon Ginder
Bruce	Citron	1982	John Donelson
Megan	Ealy	2011	Richard Smith
Tyson	Fuller	2019	Diane Slusarski
Stephanie	Haase	2019	Bridget Lear
Diane	Jordan	1987	Shivanand R. Patil, PhD
Zachary	Kockler	2020	Anna Malkova
Andrew	Lidral	1997	Jeff Murray
Janine	Martin	2011	Pedro Gonzalez-Alegre
Marcelo	Miranda Melo	2025	Hatem El-Shanti
Kacie	Meyer	2011	Tom Wassink
Michael	Molumby	2017	Josh Weiner
Nathaniel	Mullin	2024	Rob Mullins/Budd Tucker
Laura	Norwood Toro	2005	Lori Wallrath
Rebekah	Peplinski	2025	Adam Dupuy
Fedik	Rahimov	2008	Jeff Murray
Nicole	Recka	2025	Eric Van Otterloo
Elliot	Rosen	1980	Gary Gussin
Danielle	Rudd	2014	Thomas Wassink
Rita	Shiang	1990	Jeff Murray
Matthew	Strub	2020	Paul McCray
Krislen	Tison	2025	Aislinn Williams
Maria	Valdes Michel	2023	Bryan Phillips
Katie	Weihbrecht	2017	Val Sheffield
Alyssa	Wetzel	2022	Benjamin Darbro
Winnie	Xin	1990	Mike Feiss
Theresa	Zucchero Scocca	2005	Jeff Murray


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