

## From the Director

As we are at the beginning of 2024, I wish everyone a successful and productive year ahead. I am pleased to report that the Center for Human Genetics continues to grow. Last year, Drs. Shyamalika Gopalan and Kelsey Witt Dillon joined the Center, adding exceptional strength and original perspectives in population genetics. We were also fortunate to recruit Dr. Aaron Masino, whose expertise in applying artificial intelligence paradigms to clinical data adds an important dimension to the Center and is likely to contribute significantly to the Clemson University Precision Medicine Initiative.

Drs. Subham Dasgupta and Kylie Rock from the Department of Biological Sciences, Dr. Christopher Farrell from the School of Nursing, and Dr. Joanna Fiddler from the Department of Food, Nutrition and Packaging Sciences joined the Center as affiliate members, adding expertise in toxicogenomics and nutritional health deficiencies.

This January, we will be interviewing candidates for an additional faculty position at Self Regional Hall in Greenwood to further build strength in epigenetics and gene regulatory networks. Faculty affiliated with the Center for Human Genetics now represent nine departments and five colleges in addition to associated members from the Greenwood Genetic Center.

Predictably, the explosive growth of the Center has resulted in space constraints at Self Regional Hall with no further possibilities for expansion within the current facility. Plans for a second building are well underway. Architectural plans have been completed for a state-of-the-art multi-level building which will be three times the size of the current building and will include wet lab space, computational space, and some rental space for start-up companies. The building will also include a flexible conference facility with a catering kitchen, a fitness room, a roof terrace, and an outdoor volleyball court. The building will be connected to Self Regional Hall with a covered walkway and there will be a walking trail around the 16-acre Clemson property. The building will be energy efficient and designed to promote a pleasant working environment that values life-work balance while enabling leading edge research in human genetics and genomics. The Clemson University administration is actively raising the funds necessary to bring this exciting project to fruition.



Dr. Trudy F. C. Mackay, FRS, is the Self Family Endowed Chair of Human Genetics. She is a Fellow of the Royal Society of London, a member of the National Academy of Sciences of the USA, a member of the American Philosophical Society, and recipient of the 2016 Wolf Prize.

The spring semester will once again bring an outstanding cadre of excellent speakers in our Distinguished Lectures in Human Genetics series. Dr. Brenda Andrews from the University of Toronto will lead the series off by presenting her phenomenal work on systems genetics in yeast, from which fundamental principles can be applied to human genetics. Dr. Karen Miga, who is a co-leader of the telomere-to-telomere project at the University of California at Santa Cruz, will visit us in February. Dr. Nathaniel Comfort from the Johns Hopkins University is a science historian who will present new insights in the history of Rosalind Franklin. This lecture will also be featured as a Women in Science lecture in the College-wide Discover Science series. Our last speaker will be Dr. Rasmus Nielsen from the University of California at Berkeley who will discuss human evolutionary adaptations inferred from genome sequences.

I encourage everyone to attend the annual Darwin lecture on February 12, which will be delivered by Dr. Mohamed Noor from Duke University. A former president of the Society for the Study of Evolution,

Dr. Noor is also the ultimate “trekkie” who serves as a technical science consultant to the Star Trek television franchise and at Duke teaches a course on “Genetics, Evolution and Star Trek.” Captain Kirk is likely to make an appearance during the Darwin lecture.

I also encourage everyone to attend our weekly Advances in Human Genetics meetings on Monday afternoons from 2:00-3:00 pm via Zoom as well as our monthly lunch-and-learn sessions which will begin in February.

Finally, I want to congratulate Zhana Duren and Heather Flanagan-Steet on obtaining NIH grant support, and Baxton Munn on successfully completing his Master of Science degree with Dr. Lela Lackey. I also want to welcome our new research technician, Olivia Rose Hamilton, to the Center.

I look forward to continue working with all of you as we pursue our research endeavors to increase our insights in the genetic mechanisms that underlie human health and disease.

# Understanding Cellular Dynamics in Complex Disorders

by Bibhu Simkhada

The Clemson University Center for Human Genetics hosted Dr. Yoav Gilad on September 15 as part of its Distinguished Lectures in Human Genetics. Dr. Gilad is Professor of Medicine, Chief of the section of Genetic Medicine and Dean for Biomedical and Health Informatics at the University of Chicago. His primary research focuses on understanding how genetic variation and differences in gene regulation contribute to complex traits. His talk titled “No cell left behind: Using new in vitro system to study dynamic eQTLs in all cell types” presents cutting-edge technology in single cell eQTL (expression quantitative trait loci) mapping using iPSC derived cell lineages.

The growing use of single-cell RNA sequencing (scRNA-seq) has highlighted the significance of cell diversity and cell specificity in understanding complex disorders. One outstanding challenge in studying complex diseases in humans, however, is understanding the spatiotemporal dynamics of gene expression. Historically, investigators have used directed differentiation to generate terminal cell-types from iPSCs to perform bulk RNA sequencing on these cultures. However, this often severely underrepresents cell-type heterogeneity.

One alternative to these methods is to perform time-course sampling of differentiating cell types by collecting samples at various time points of differentiation. Another alternative can be to use directed differentiation to produce more complex organoids from iPSCs. However, both methods are laborious and require long-term cell culture/differentiation. Dr. Gilad's team developed a novel guided differentiation method as a fast and relatively cost-effective way to differentiate iPSC cultures into multiple cell types while preserving the dynamic cellular diversity.

Dr. Gilad's team differentiated iPSCs into a cardiac lineage. This organoid-like culture showed contraction at 8 days of differentiation. scRNA-seq performed on 10-day culture harbored cell lineages from all three germ layers. Early developmental pluripotent cells were marked by *POU5F1* and *L1TD1* expression, and neural ectoderm were marked by *SOX2* and *CRABP1*. Foregut endoderm was marked by *FOXA2* and *HHEX*, and hepatic endoderm was marked by downregulation of *HHEX* and upregulation of *AFP*. Endothelial cells were marked by *KDR*, *CDH5* and *PECAM1*, and endocardial cells were further marked by *NFATC1*. Cardiomyocytes were classified by the expression of *TNNI2*, *ACTN2*, *RYR2* and *CACNA1C*, and also showed expression of *TNNI3*, indicating maturation. Guided differentiation, as opposed to directed differentiation, generates cell types at various stages of differentiation, i.e., both terminal and



Dr. Yoav Gilad

progenitor cell populations. This study is comparable to a previous time-course study done over a 15-day period, where a single time-point guided differentiation produced cells from the entire time-course sampling. Additionally, this experiment also showed comparable levels of cell diversity and transcriptional similarity to a previously published scRNA-seq done on 100-day organoid culture. This proof-of-concept study provides a fast and reliable method to generate diverse cell lineages to perform inter-individual and population level studies, facilitating eQTL mapping across a broad spectrum of complex disorders.

Beyond research, Dr. Gilad is also passionate about supporting young scientists. He advocates the need for a shift away from a culture of overwork and underappreciation. He strongly believes that it is important for established scientists in influential positions to support young scientists who are often motivated by the fear of failure rather than the joy of discovery. During a discussion with graduate students, Dr. Gilad shared invaluable insights on factors to consider while transitioning from doctoral to postdoctoral programs, including funding, resources, freedom to pursue ideas and flexibility in work dynamics.

Bibhu Simkhada is a graduate student in the Center for Human Genetics at Clemson University.

## Pillars of the Social Brain

*by Ishita Debnath and Spencer Hatfield*

The College of Science was delighted to welcome Dr. Gene Robinson, a prominent entomologist and director of the Carl R. Woese Institute for Genomic Biology at the University of Illinois at Urbana-Champaign, to give a lecture on October 2, 2023, as part of the Discover Science and Distinguished Lectures in Human Genetics series. Robinson is a member of the National Academy of Sciences and the National Academy of Medicine and received the distinguished Wolf Prize in Agriculture for his work, among many other accolades.

Robinson is well known for his pioneering work in the field of behavioral genetics, with a focus on understanding the genetic basis of social behavior in honeybees. He is responsible for fundamental advancements in this field, such as defining the genetic, neurological, and evolutionary mechanisms responsible for variation in honeybee behavior and social order.

His seminar, titled "Pillars of the Social Brain: Lessons from the Honeybee", was a deep dive into the complex social world of honeybees, and drew remarkable comparisons to human social behaviors. Much like us, honeybees have a complex division of labor, where individual bees play different roles in the maintenance and defense of their communal dwelling — the hive. The division of labor in the beehive is dynamic, and changes quite drastically over a honeybee's 6-week lifespan. The cyclical harmony observed within the hive is orchestrated by a delicate interplay of genetic, social, and environmental factors.

The genetic makeup of individual bees influences their predisposition to certain roles, while social interactions among hive members contribute to the fluidity and adaptability of their responsibilities. For example, there is a pareto-like distribution in terms of foraging behavior, where 10-15% of foragers are responsible for 50% of the successful foraging. However, if these high performers are removed from the hive, other foragers will increase production in order to maintain the health of the hive.

Moreover, environmental factors play a crucial role in shaping the collective behavior of the hive, responding to changing conditions and ensuring the overall well-being of the community. By investigating these different factors, Robinson's goal is to piece together the complex puzzle of social behavior, from which we may gain a better understanding of both honeybees and humans.

The honeybee is also an excellent model for studying gene expression, due in part to the controlled hive environment. This, combined with the large sample sizes of bees with specified roles, allows for the interrogation of differences in gene expression between groups of bees, such as soldiers and foragers. It can also be used to determine potential genetic contributions to within-group variation, such as successful versus unsuccessful foragers.

Despite the brains of the honeybee being seemingly simple compared to that of a human, they contain subsets of neurons called Kenyon cells that are essential for discriminating value, reward, and behavior. Robinson posits that, "Genomics is better than neurobiology at interrogating conservation". In fact, there are deeply conserved genetic



*Dr. Gene Robinson*

mechanisms between honeybees and humans that dictate social behavior. He described a fascinating transcriptomics experiment that identified the genetic factors involved in aggression, reproduction, and productivity. These genes translate to orthologous genes in humans that have been implicated in antisociality, drug abuse, and autism spectrum disorder.

Robinson's work indicates that there is a genetic toolkit for behavior. And, remarkably, this genetic toolkit likely evolved independently in different species, as the most recent common ancestor of humans and honeybees shows no social behavior.

Robinson expressed his excitement at the cutting edge of neurobiology and genetics. He explained that the advent of single-cell gene expression profiling is how we can unite these two fields, allowing researchers to unite at the level of the cell. This, he argues, is the next big step in understanding the complexity of the brain and behavior.

His work continues to unravel the complexity of the sociobiological world and enhance our understanding of some of nature's most intriguing creatures.



Ishita Debnath and Spencer Hatfield are graduate students in the Center for Human Genetics at Clemson University.



# Protocadherins and Development of the Nervous System

*by Spencer Hatfield and Ishita Debnath*

On November 13th, 2023, Dr. Tom Maniatis, an esteemed investigator at Columbia University's Zuckerman Institute and Isidore S. Edelman professor of Biochemistry and Molecular Biophysics, presented an enlightening seminar as part of the Distinguished Lectures in Human Genetics series. Maniatis received his Ph.D. in molecular biology from Vanderbilt University, followed by postdoctoral studies at Harvard University and the Medical Research Council Laboratory of Molecular Biology in Cambridge, England. He has also received honorary Ph.D. degrees from the Cold Spring Harbor Graduate School, the University of Athens, and Rockefeller University.

Maniatis acts as the Scientific Director and Chief Executive Officer of the New York Genome Center. He has garnered numerous accolades for his research contributions, including the Richard Lounsbery Award for Biology and Medicine and the Lasker Koshland Special Achievement Award in Medical Science. He is a member of the American Academy of Arts and Sciences, the National Academy of Sciences, and the National Academy of Medicine.

Maniatis is renowned for his groundbreaking work in molecular biology. On top of writing the book on molecular cloning, his laboratory was also pivotal in the discovery of RNA splicing, a finding that revolutionized how we understand the fundamental mechanisms of RNA function.

His seminar, titled "The Genomic Organization and Functions of Large Human Gene Clusters:  $\beta$ -globin and the Clustered Protocadherins", explored and analyzed the genomic structure and functional aspects of two important gene clusters in the human genome. One of the gene clusters explored by Maniatis is the  $\beta$ -globin gene cluster, which is located on chromosome 11. This cluster comprises five genes that work together to regulate the production of hemoglobin, a vital component of red blood cells responsible for oxygen transport. Maniatis used a variety of molecular biology techniques to interrogate the genomic organization of this cluster. His research aimed to unravel the complexities of how these genes are organized within the cluster and how they function in concert to control hemoglobin synthesis. This investigation is particularly significant due to its implications for a diverse range of hemoglobinopathies, which are disorders characterized by abnormal hemoglobin production, as well as  $\beta$ -thalassemia, a genetic disorder resulting in reduced or absent synthesis of beta-globin chains.

Next, he described the clustered protocadherins. Protocadherins comprise the largest mammalian subgroup of the cadherin gene superfamily that encodes homophilic cell-adhesion proteins. Unlike the  $\beta$ -globin gene cluster involved in hemoglobin regulation, the clustered protocadherins play a crucial role in the intricate development of the nervous system. They are pivotal in regulating neuronal cell fate, influencing both cell survival and death across diverse animal models. The clustered protocadherins also contribute significantly to various facets of axonal development, with their capacity to generate distinctive molecular codes forming the foundation for mechanisms involved in both axon-target and axon-axon recognition. Notably, diverse members from all protocadherin clusters exhibit localization on neuronal soma, dendrites,



*Dr. Tom Maniatis*

axons, as well as at growth cones and synapses, spanning different stages of neuronal differentiation and maturity.

Different subsets of the clustered protocadherin genes are expressed differentially in individual neurons, thereby generating a vast cell surface diversity because of their combinatorial expression. This differential regulation of neuron-specific expression of protocadherin genes led to the hypothesis that protocadherins may provide a synaptic-address code for neuronal connectivity or a single-cell barcode for self-recognition/self-avoidance.

Understanding the logic of these cadherin "barcodes" has implications for neurodevelopmental and neuropsychiatric disorders. By exploring the roles of these highly conserved genes and their single-cell diversity in neurons, Maniatis advances our understanding of the intricate molecular processes that govern brain development and function. This knowledge not only enhances our comprehension of basic neurobiology but also holds potential implications for the development of targeted therapies for neurological and neuropsychiatric conditions.

Maniatis' investigation of the genetic complexity of these gene clusters has greatly advanced our understanding of neurogenetic regulation, accelerating the fields of molecular genetics and medicine. His research and analysis of the gene clusters has unraveled the intricate mechanisms governing gene clusters, shedding light on the complex interplay of genes and their regulatory elements, which will have profound benefits for personalized medicine.

Spencer Hatfield and Ishita Debnath are graduate students in the Center for Human Genetics at Clemson University.

## A Conversation with Kelsey Witt Dillon

### Can you briefly describe your research interest and how you became interested in ancient DNA?

I was a genetics major as an undergraduate student, and my favorite course was population genetics. I remember learning about the “Mitochondrial Eve”, which is this idea that all humans can trace their mitochondrial ancestry to a single original sequence. I was really fascinated by the idea that we can look at human genomes to understand human evolutionary history. Soon after, I learned about the field of ancient DNA, which takes the idea a step further to study past populations by sequencing their genomes. As soon as I learned about the field, I knew I wanted to be a part of it.

### What can we learn from the analysis of ancient DNA that is relevant to contemporary human populations?

Part of the value of ancient DNA is reconstructing human history. Historically, human populations moved around a lot and interacted with other populations, and we can use ancient genomes to piece together those past migrations and admixture events. But this human history also has consequences for us today. For example, we have identified many genetic variants that were favored by selection in the past because they helped humans thrive in a new environment or resist disease. However, some of these genetic variants are no longer adaptive, but instead have negative health consequences. Understanding the context of these variants can help us understand their function and why they may be contributing to disease today.

### What can we learn about the lifestyle and intelligence of Neanderthals and Denisovans from their DNA?

There is often a misconception that archaic humans like Neanderthals and Denisovans lived like stereotypical cavemen – a lot of popular media shows them as unintelligent and easily outmatched by modern humans. However, the archaeological record and the genomes we have sequenced tell a very different story. We know that archaic humans used complex tools, made cave art, and were complex beings just like modern humans. We also know from looking at their genomes that Neanderthals and Denisovans were well-adapted to a variety of environments, including low-UV environments at high latitudes and high-altitude, in the case of Denisovans. Furthermore, these adaptations have also been introduced to modern human populations through gene flow. Therefore, modern humans were able to thrive after expanding out of Africa because of their interactions with Neanderthals and Denisovans.

### What do you see is the biggest question in the field of population genetics?

I think one of the biggest questions in the field today is understanding admixture, or the exchange of genetic material between distinct populations. Many statistical tools have been developed to reconstruct admixture events and break admixed genomes down into distinct ancestry sources, but it is still a complex problem to solve. We rarely see populations (especially humans) that are the product of a single admixture event – even admixture from



Neanderthals and Denisovans occurred multiple times. Disentangling all these admixture events to understand population history and how those past gene flow events impact modern populations is still a big question in the field.

### What advice do you have for aspiring young geneticists?

My biggest advice is to find a good support network, including both more senior mentors and those at your same career stage. Working in academic and doing scientific research can be really challenging in a lot of ways, and having people around you who will support you when you are struggling can make a big difference. Having other people you can commiserate with when grants are not funded or when papers need a lot of revisions can lighten your load. Also, as you become more independent in your career, those colleagues you have found are a great resource for future research collaborations. Most of my collaborations now are with friends I made in graduate school and as a postdoc, and I really enjoy the positive dynamic of those projects.

### What activities do you enjoy when you are not analyzing ancient DNA?

I really like to read, especially science fiction novels, and knit. I also really enjoy baking! These days my daughter, Lizzie, likes to get involved with baking too.

Kelsey Witt Dillon is an Assistant Professor in the Department of Genetics and Biochemistry and the Center for Human Genetics at Clemson University.

## Viewpoint

### Live Well and Be Happy

by Robert R. H. Anholt

There are many uncertainties in our life, but one thing is for sure: we will die. The question is how long will we live and how long can we live in good health? Will we ever be able to predict individual lifespan and healthspan, and if so, do we really want to know?

There is enormous variation in longevity among and within species. In the animal kingdom, turtles are the champions of long life. Harriet, a legendary tortoise on the Galápagos Islands, lived to be 176 years old. The major event in Harriet's long and boring life might have been her encounter with Charles Darwin (although this is disputed) and her trip to Australia where she died in a zoo in Queensland from cardiac failure. Dogs, cats, and horses have mean lifespans of about 10-13, 12-17, and 20-30 years, respectively, but here we also see lots of variation between and within breeds, and domestication increases survival.

Determining the genetic factors that underlie variation in lifespan is complicated. Human lifespan has increased dramatically over millennia. The young Pharaoh, Tutankhamun, affectionately known as King Tut, died as an 18-year old teenage boy. Examination of his mummified remains revealed a broken leg and traces of malaria. The exact cause of his death still remains mysterious, although malaria might have been a contributing factor. In ancient Rome, life expectancy is estimated to have been between 20 and 30 years. The infamous King Henri the Eighth reached the enviable age of 56, an old man by the standards of his time. The pampered nobility had better lifespan expectations than the impoverished hard working serfs.

One can speculate that from an evolutionary perspective our lifespan should not exceed a woman's reproductive years. Today's common geriatric diseases, such as cardiovascular disease, cancer and Alzheimer's disease, would not have been as prominent in the Middle Ages, when the major cause of death for those who survived child birth was infectious disease. A small wound, when infected, could lead to sepsis and be a death sentence.

The greatest leap in lifespan extension came with the discovery of penicillin and subsequently insulin. Improvements in hygiene and personal care also played a role in increasing our life expectancy. In 1940, the mean lifespan in the United States reached a whopping 62.9 years! Since then, the mean lifespan has increased by another 13 years to 76.1 in 2021. In the last two years, however, our mean lifespan has decreased and is less than in other Western societies, for example Norway. This is due to the COVID pandemic, car accidents, homicides, the opioid epidemic, and socioeconomic disparities in access to health care. Thus, we need to keep the notion of mean lifespan in context. Those of us who are economically comfortable, have survived childbirth and COVID, are not addicted to drugs, have not been in a deadly car accident or be victim of an active shooter, are likely to live decades beyond the mean life expectancy.



*"Live Long and Prosper"*

Centenarians, are no longer uncommon. The oldest woman on record is Jeanne Louise Calment, who died in 1997, 122 years and 164 days old. She maintained an active lifestyle until a very old age and enjoyed chocolates, an occasional cigarette, and port. The oldest living man on record was a Japanese post office worker, Jiroemon Kimura, who died in 2013 after reaching the age of 116 years and 54 days.

Clearly, environmental conditions can move the genetic goal posts in our favor a healthy lifestyle (eat your vegetables!) can increase life expectancy. But that may not always be the case. Golda Meir, the fourth prime minister of Israel, was a heavy cigarette smoker, but the nicotine did not prevent her from reaching the age of 80, in good mental health. Like Jeanne Louise Calment, Golda had good genes that protected against adverse environmental exposures. We are exposed to environmental toxicants in our water, air and food, and to UV and cosmic radiation, and occasional stressful experiences, all of which have to be dealt with by physiological processes orchestrated by our genome.

This raises the question of how we can empower predictions of individual lifespan from genetic studies. The interaction terms, gene-by-gene and gene-by-environment, may be more important determinants for lifespan prediction than additive effects of genetic variants alone. But establishing comprehensive and predictive metrics of fluctuating and changing environmental exposures during a person's life as an accurate measure of environmental influence is a tall order. Until that problem is solved, if ever, we can only hope being lucky enough to harbor gene-gene interactions that mitigate the cumulative adverse effects of environmental exposures. We all wish to live long healthy lives, but let's remember that happiness is also important. So, while we adopt a healthy lifestyle, we may as well occasionally roll the dice and enjoy a good wine, a box of fine chocolates, and a delicious cholesterol-rich crème brûlée.

Robert Anholt is the Provost Distinguished Professor of Genetics and Biochemistry and Director of Faculty Excellence in the College of Science at Clemson University.



## Grants

**Zhana Duren** received a five-year \$1,787,572 Maximizing Investigators Research Award (MIRA, R35) from the National Institute of General Medical Sciences to develop statistical methods for interpretation of genetic variants by gene regulatory networks.

**Heather Flanagan-Steet** received a two-year \$500,000 R01 grant from the National Institute of General Medical Sciences to study pathogenic mechanisms of congenital disorders of glycosylation.

**Austin Herbert** received a \$2,000 Interdisciplinary Graduate Fellowship from the Department of Genetics and Biochemistry at Clemson University.

## Seminars

On Friday, **January 19**, at 2:30 pm, **Dr. Brenda Andrews**, Canada Research Chair in Systems Genetics & Cell Biology in the Department of Molecular Genetics at the University of Toronto, will present a seminar titled “Mapping genetic networks using systematic genetics.” The seminar will be at the Clemson University campus in Freeman Hall, room 078.

On Monday, **February 12**, at 2:00 pm, **Dr. Mohamed Noor**, Professor of Biology and Interim Vice Provost of Academic Affairs at Duke University will present the annual **Darwin Lecture**, titled “Using science-fiction depictions to teach real-world evolution concepts.” The lecture will be delivered virtually via Zoom, <https://clemson.zoom.us/j/95376010710>.

On Friday, **February 23**, at 2:30 pm, **Dr. Karen Miga**, Assistant Professor of Biomolecular Engineering at the University of California, Santa Cruz and Associate Director of Human Pangenomics at the UC Santa Cruz Genomics Institute, will present a seminar titled “Expanding studies of global genomic diversity with complete, telomere-to-telomere (T2T) assemblies.” The seminar will be at the Clemson University campus in Freeman Hall, room 078.

On Friday, **March 15**, at 2:30 pm, **Dr. Nathaniel Comfort**, Professor in the Department of History of Medicine at the Johns Hopkins University School of Medicine, will present a Discover Science lecture titled “Reflections on gender, science, and myth: A new look at Rosalind Franklin and DNA.” The seminar will be at the Clemson University campus in Freeman Hall, room 078.

On Friday, **April 5**, at 2:30 pm, **Dr. Rasmus Nielsen**, Professor in the Departments of Integrative Biology and Statistics at the University of California, Berkeley, will present a seminar titled “Human evolutionary adaptation - as revealed by our genomes”. The lecture will be delivered virtually via Zoom, <https://clemson.zoom.us/j/93838990927>.

## Publications

(*affiliates of the Center for Human Genetics are in bold font*)

Ancona N, Bastola A, **Alexov E**. 2023. PKAD-2: New entries and expansion of functionalities of the database of experimentally measured pKa's of proteins. *J Comput Biophys Chem* **22**: 515-524.

Borja M, Neri-Castro E, Gutiérrez-Martínez A, Bledsoe R, Zarzosa V, Rodríguez-López B, Strickland JL, Becerra-López J, Valenzuela-Ceballos S, **Parkinson CL**, Alagón A and Castañeda-Gaytán G. 2023. Ontogenetic change in the venom composition of one Mexican black-tailed rattlesnake (*Crotalus molossus nigrescens*) from Durango, Mexico. *Toxicon* **234**: 107280.

Cheng R, Zhou S, K C R, Lizarazo S, Mouli L, Jayanth A, **Liu Q** and Van Bortle K. 2023. A combinatorial regulatory platform determines expression of RNA Polymerase III subunit RPC7α (*POLR3G*) in cancer. *Cancers (Basel)* **15**: 4995.

Claus LR, Chen C, Stallworth J, Turner JL, Slaats GG, Hawks AL, Mabillard H, Senum SR, Srikanth S, **Flanagan-Steet H**, Louie RJ, Silver J, Lerner-Ellis J, Morel C, Mighton C, Sleutels F, van Slegtenhorst M, van Ham T, Brooks AS, Dorresteijn EM, Barakat TS, Dahan K, Demoulin N, Goffin EJ, Olinger E; Genomics England Research Consortium; Larsen M, Hertz JM, Lilien MR, Obeidová L, Seeman T, Stone HK, Kerecuk L, Gurgu M, Yousef Yengej FA, Ammerlaan CME, Rookmaaker MB, Hanna C, Rogers RC, Duran K, Peters E, Sayer JA, van Haaften G, Harris PC, Ling K, **Mason JM**, van Erde AM and **Steet R**. 2023. Certain heterozygous variants in the kinase domain of the serine/threonine kinase NEK8 can cause an autosomal dominant form of polycystic kidney disease. *Kidney Int* **104**: 995-1007.

**Dasgupta S**, Simonich MT and Tanguay RL. 2024. Developmental toxicity assessment using zebrafish-based high-throughput screening. *Methods Mol Biol* **2707**: 71-82.

**Feliciano DM**. 2023. Do neural stem cell extracellular vesicles help evade depression? *Neuroscience*. **2023**: S0306-4522(23)00346-9.

Gettings JR, **McMahan CS**, Cleveland CA, Varela-Stokes A, Hubbard K, Hamer SA, Walden HS and Yabsley MJ. 2023. Association between vector-borne pathogen seroprevalence in shelter-housed and owned dog populations in the contiguous United States of America. *Parasit Vectors* **16**: 405.

Harb JF, Christensen CL, Kan SH, Rha AK, Andrade-Heckman P, Pollard L, **Steet R**, Huang JY and Wang RY. 2023. Base editing corrects the common Salla disease *SLC17A5* c.115C>T variant. *Mol Ther Nucleic Acids* **34**: 102022.

Hickman AR, Selee B, Pauly R, Husain B, Hang Y and **Feltus FA**. 2023. Discovery of eQTL alleles associated with autism spectrum disorder: A case-control study. *J Autism Dev Disord* **53**: 3595-3612.

Hofmeister NR, Stuart KC, Warren WC, Werner SJ, Bateson M, Ball GF, Buchanan KL, Burt DW, Cardilini APA, Cassey P, De Meyer T, **George J**, Meddle SL, Rowland HM, Sherman CDH, Sherwin WB, Vanden Berghe W, Rollins LA, **Clayton DF**. 2023. Concurrent invasions of European starlings in Australia and North America reveal population-specific differentiation in shared genomic regions. *Mol Ecol* **2023**:10.1111/mec.17195.

**MacPherson RA, Shankar V, Anholt RRH and Mackay TFC**. 2023. Genetic and genomic analyses of *Drosophila melanogaster* models of chromatin modification disorders. *Genetics* **224**: iyad061.

Naveed S, Gandhi N, Billings G, Jones Z, Campbell BT, Jones M and **Rustgi S**. 2023. Alterations in growth habit to channel end-of-season perennial reserves towards increased yield and reduced regrowth after defoliation in upland cotton (*Gossypium hirsutum* L.). *Int J Mol Sci* **24**: 14174.

Oladosu O, Esobi IC, Powell RR, Bruce T and **Stamatikos A**. 2023. Dissecting the impact of vascular smooth muscle cell *ABCA1* versus *ABCG1* expression on cholesterol efflux and macrophage-like cell transdifferentiation: The role of SR-BI. *J Cardiovasc Dev Dis* **10**: 416.

Pandey P, Panday SK, Rimal P, Ancona N and **Alexov E**. 2023. Predicting the effect of single mutations on protein stability and binding with respect to types of mutations. *Int J Mol Sci* **24**: 12073.

Rand MD, Tennessen JM, **Mackay TFC** and **Anholt RRH**. 2023. Perspectives on the *Drosophila melanogaster* model for advances in toxicological science. *Curr Protoc* **3**: e870.

## Out and About

**Robert Anholt** was appointed Associate Editor of Frontiers in Genetics, Evolutionary and Population Genetics (specialty section of Frontiers in Genetics, Frontiers in Ecology and Evolution and Frontiers in Plant Science). He also served on two NIH study sections and as a reviewer for Peer Review Training applications for the Genetics Society of America. He moderated a panel discussion on “Publishing and Data Sharing” on the annual Ethics Day at Clemson University.

**Spencer Hatfield** presented a ‘Science on Tap’ Community Lecture on cocaine addiction at Howards on Main in Greenwood, SC.

**Trudy Mackay** and **Robert Anholt** attended the biannual meeting of the American Philosophical Society in Philadelphia, PA.

**Fabio Morgante** attended the CM Statistics meeting in Berlin, Germany.

**John Poole** attended the SC23 International Conference for High Performance Computing, Networking, Storage and Analysis in Denver, CO.

**Bibhu Simkhada** represented Clemson University’s College of Science and Center for Human Genetics at the Annual Biomedical Research Conference for Minoritized Scientists in Phoenix, Arizona.

**Bibhu Simkhada, Kristin Bussey, Rebecca Bishop, Alp Ummet and Chunming Liu** presented posters at the annual meeting of the American Society for Human Genetics in Washington, DC. **Trudy Mackay, Robert Anholt and Rachel Lyman** also attended the conference.

**Zhana Duren, Naqing, Kaya Yuan, Hui Ma, Fengge Chang and Ishita Debnath** attended the Conference on Regulatory & Systems Genomics organized by the International Society of Computational Biology (ISCB) in Los Angeles, CA, and presented posters. **Ishita Debnath** also gave a flash talk.

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<https://scienceweb.clemson.edu/chg/>

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<https://iamatiger.clemson.edu/giving/giving-to-clemson?id=650eec53-bf25-45eb-8960-017c36738c06>

