CLEMSON UNIVERSITY
CENTER FOR HUMAN GENETICS

The Transcript

Newsletter of the Center for Human Genetics at Clemson University
Spring, 2022

From the Director

The great Brazilian novelist Paulo Cuelho wrote in his book *The Witch of Portobello* "Keep the bicycle moving, because if you stop pedaling, you will fall off." The Center for Human Genetics keeps pedaling without fear of falling off. The Center is growing, with new faculty, students, and staff. We welcome Alexis Stamatikos, Jessica Larsen, and David Feliciano as new Center for Human Genetics faculty. We are in the process of recruiting an additional colleague with expertise in epigenomics and we will be recruiting two additional faculty members in 2022.

Last Fall, we had our first successful virtual COBRE retreat with our pilot project leaders symposium. The spring of 2022 marks the initiation of the first Pilot Project Program of the Center of Biomedical Research Excellence in Human Genetics with awards to five investigators encompassing four university departments. Congratulations to Drs. Zhana Duren, Xinyi Li, Jessica Larsen, Liangjiang Wang and David Feliciano!

Zhana Duren will develop computational approaches to link open chromatin to gene expression from unpaired samples by analyzing single cell RNAseq data and ATAC seq data. Xinyi Li will develop computational methods to associate genetic variation with variation in brain scan images for the early detection and management of Alzheimer's disease. Jessica Larsen will develop a polymer-based nanoparticle delivery system for encapsulation and targeting of Cas9 containing ribonucleoprotein particles that can cross the blood brain barrier to target specific brain regions for therapeutic gene editing. Liangjiang Wang will identify splice variants of long non-coding RNAs that can serve as diagnostic and prognostic biomarkers for triple negative breast cancer, the most severe form of metastatic breast cancer. David Feliciano will study the neuropathogenesis of tuberous sclerosis, a rare but serious brain disorder.

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 Center for Human Genetics welcomes two new research technicians, Sarah Macon and Patrick Freymuth, who will join the Mackay-Anholt laboratory, and we are looking forward to welcoming new students later this year. We also welcome John Poole, our newest bioinformatician and computer wizard, who has joined our bioinformatics team. Unfortunately, our administrative assistant, Heather Lynch, is leaving us to pursue her career advancement as a Digital Content Specialist at Central Carolina Technical College. We will miss her.

The Spring semester will continue our series of Distinguished Lectures in Human Genetics with Drs. James Lupski from Baylor College of Medicine, Daniel Weinberger from the Lieber Institute for Brain Development, and Wylie Burke from the University of Washington. We are also looking forward to this year's celebration of Charles Darwin's birthday with Dr. Dolph Schluter from the University of British Columbia. This issue of The Transcript features synopses of last semester's distinguished speakers contributed by our graduate students, a tradition which we will continue.

We are all aware that pay lines at the NIH are at historically low levels leaving much excellent research proposals without funding. However, I am confident in the talent and persistence of our faculty and I have no doubt that we can navigate the turbulent waters successfully.

I would like to thank all members of the Center for Human Genetics, both at the Greenwood campus and the Clemson campus, faculty, students, and staff members, for continuing to be part of a collegial and supportive environment. Together, we are the Clemson family!

The First Annual Project Leaders Symposium of the Center for Biomedical Research Excellence (COBRE) in Human Genetics

by Rebecca A. MacPherson

On November 16th, project leaders of the COBRE in Human Genetics presented their work at a symposium as part of the 1st Annual COBRE in Human Genetics fall retreat. In addition to research funding, the COBRE also includes support for professional development and mentoring, two focuses of the annual retreat. This symposium provided project leaders an opportunity to share their work with the broader scientific community. Each talk garnered thoughtful questions from the over 60 people in attendance and highlighted the excellent work taking place within the Clemson Center for Human Genetics and the Greenwood Genetic Center.

The symposium began with a talk by Dr. Andrei Alexandrov, titled "Forward Genetic Analysis of Human Nuclear Long Non-Coding RNAs". Alexandrov is working to identify regulatory pathways acting on long non-coding RNAs in the nucleus using human cell lines. Previously, the Alexandrov lab developed ultra-high throughput technology to interrogate the genetic underpinnings of biogenesis and maturation of the cancer-associated lncRNA MALAT1. With this technology, Alexandrov is investigating pathways that may be critical for other disease-associated long non-coding RNAs, including PAN, which is required for pathogenesis of Kaposi's sarcoma-associated herpesvirus, and MEN-β, which is involved in neuronal differentiation. In the future, genes within regulatory networks identified by the Alexandrov lab can be used as potential targets for anti-cancer and anti-viral therapies.

Next, Dr. Miriam Konkel presented a talk on "Transposable Elements, Genome Architecture and Gene Regulation". The research focus of the Konkel lab is to identify sources of and to better understand the spread of jumping genes, or transposable elements, within the human genome. Specifically, Konkel and her colleagues investigate *Alu* elements, the most active transposable element in humans, as about 1 in 20 live births features a new insertion, of an *Alu* element. Currently, the Konkel lab is examining expression of *Alu* elements and assessing the relationship between known *Alu* elements, finding that there are likely more subfamilies of *Alu* elements than previously appreciated. Given that insertions and genomic rearrangements caused by *Alu* elements are associated with numerous genetic disorders, including cancer, Konkel's work on the spread of *Alu* elements is important for a broader understanding of human disease.

The third speaker of the day was Dr. Heather Flanagan-Steet from the Greenwood Genetic Center. Her presentation was titled "Mechanisms of Disease Pathogenesis in Congenital Disorders of Glycosylation". Flanagan Steet uses the zebrafish to model congenital disorders of glycosylation (CDGs), rare genetic diseases caused by defects in placement of sugar molecules onto proteins, nucleic acids, and lipids for proper function. The Flanagan-Steet lab studies PMM2-CDG, the most common CDG, and is working to understand how specific enzymes (previously implicated due to observed changes in cartilage and neuromuscular traits in the PMM2-CDG fish) contribute to PMM2-CDG. Flanagan-Steet's group is also working to understand the mechanism of a successful PMM2-CDG pharmaceutical intervention while taking a systems approach to identify pathways and genes that may influence PMM2-CDG disease severity.

Dr. Fabio Morgante gave the concluding talk of the symposium, titled "Genomic Prediction of Human Disease". The Morgante lab works to develop better computational methods for predicting phenotypes (traits) from genotypes (genetics). These efforts are important for improving precision medicine, yet remain challenging, as multiple phenotypes may share genetic components and genotype-phenotype relationships are highly complex. The Morgante lab has developed new self-tuning Bayesian multivariate regression methods (*e.g. mr.mash*) for phenotype prediction that rely upon flexible priors and joint modeling of multiple phenotypes. Compared to other models, *mr.mash* generally produced more accurate and efficient predictions across multiple conditions within simulated and real datasets. We are looking forward to progress from these research projects for the 2022 COBRE Project Leaders symposium.

Rebecca MacPherson is a graduate student in the Center for Human Genetics at Clemson University.

Rules of Engagement: Molecular Arms Races between Host and Viral Genomes

by Austin Herbert

On Friday, October 8th, 2021, Dr. Harmit Malik, Professor and Associate Director of the Fred Hutchinson Research Center gave a seminar in the distinguished speaker series of Clemson University's Center for Human Genetics titled *"Rules of engagement: molecular arms races between host and viral genomes"*. One topic studied by Malik is the coevolution of interactions between viral and host anti-viral proteins in primates. He describes these interactions as a classic example of the "Red Queen hypothesis". This hypothesis was coined from the following statement by the Red Queen, a fictional character in Lewis Carroll's *"Through the Looking Glass"*, she states "Now, here, you see, it takes all the running you can do, to keep in the same place. If you want to get somewhere else, you must run at least twice as fast as that!". In biology, the Red Queen hypothesis is often defined as the constant adaptation and evolution of advantageous traits in one species in response to the ever-evolving competing species in the same ecosystem. Studying host-viral interactions at the molecular level is quintessential for understanding the evolution of zoonotic viruses and how host and viral genotype manifest as differences in viral pathogenicity between species.

Dr. Harmit Malik

Malik earned his Bachelor's degree in technology and chemical engineering at the Indian Institute of Technology, and his Ph.D. in biology at the University of Rochester, NY. He started his work at the Fred Hutchinson Cancer Research Center in 1999 studying the evolution of centromeres and centromeric proteins; he soon after started his own lab at the same research center in 2003. Malik currently uses three model systems, primates, yeast, and *Drosophila*, and centers his research around understanding innate and intrinsic immunity against viruses in primates, the role of centromeres in chromosomal segregation, and mobile genetic elements in *Drosophila*. He received the Early Career Scientist award of the Howard Hughes Medical Institute in 2009, and in 2017 was awarded the Eli Lilly Prize in microbiology, the most prestigious award of the American Society of Microbiology.

Viral proteins and host anti-viral proteins have evolved to outcompete one another, where the viral proteins evolve to offer advantages for viral replication and survival, while the host anti-viral proteins evolve to provide adequate immune responses and defenses to these viral proteins. The presence of varying selection pressures between species subsequently alters amino acid sequences between human anti-viral proteins and their orthologues in closely related species. Such changes consequently affect anti-viral potency. To perform these studies, Malik harnesses a deep mutational scanning approach to determine how each amino acid potentially alters the efficacy of anti-viral proteins between species. In this technique, the effect of single missense mutations on the restriction efficacy of human and macaque HIV-1 antiviral protein TRIM5α are assessed via changes in fluorescence intensity using fluorescence activated cell sorting (FACS). Gain-of-function mutations reduce intensity of the fluorescence read-out and are ultimately binned for further enrichment and characterization of amino acid changes on viral restriction. Once again-of-function mutation is discovered at one locus, the stability of its viral restriction potency is investigated by altering other amino acids in this already mutated gene.

Malik has demonstrated that a single amino acid change in human TRIM5α readily increases levels of its HIV-1 restriction capacity in cell culture compared to those of the macaque TRIM5a. Furthermore, the levels of viral restriction in human TRIM5α mutant were relatively unaffected by the introduction of other missense mutations. Interestingly, whether these mutations occurred in highly conserved or rapidly evolving sites between the macaque and human TRIM5α gene had no significant effect on viral restriction. Thus, function of TRIM5a in both macaques and humans is robust and its evolutionarily landscape is largely unaffected by small changes in its amino acid sequence.

A combination of *in silico* sequence analysis, and *in vivo* techniques, provided Malik and his colleagues with the means to assess adaptive evolution through sequence mutation and to identify gain-of-function mutations that increase the fidelity of viral restriction in TRIM5α. Addressing the effects of evolutionarily derived amino acid changes among orthologous antiviral proteins contributes to a better understanding of how speciation and selection impact the arms race between virus and anti-viral proteins in different species. Moving forward, Malik and his team at the Fred Hutchinson Cancer Research Center continue to explore the genomic mechanisms responsible for genomic adaptations that offering competitive advantages to competing organisms.

Austin Herbert is a graduate student in the Center for Human Genetics at Clemson University.

Endogenous Retroviruses and Neurodegeneration

by J. Spencer Hatfield

On September 3, 2021, Dr. Avindra "Avi" Nath, Clinical Director of the Section of Infections of the Nervous System in the Division of Neuroimmunology and Neurovirology at the National Institute of Neurological Disorders and stroke, presented a seminar in the Distinguished Lectures in Human Genetics series at the Clemson University Center for Human Genetics titled "The role of endogenous retroviruses in neurodegeneration."

Nath received his MD from Christian Medical College in India in 1981. He then completed a neurology residency at the University of Texas, followed by multiple fellowships in neurovirology. Following faculty positions at the University of Manitoba, the University of Kentucky, and Johns Hopkins University, he joined the NIH as Clinical Director for the National Institute of Neurological Disorders and Stroke (NINDS). He is also the Director of the Translational Neuroscience Center and Chief of the Section of Infections of the Nervous System at NINDS.

I had the pleasure of speaking with him prior to his lecture. His story includes observations from his early clinical work on AIDS, tumultuous travels to Liberia during the most recent Ebola outbreak, compelling research on the human HERV-K virus, and investigations into the long-term neurological effects of COVID-19 infection.

Nath's interest in retroviruses began during his time running an AIDS clinic. He noticed that when one patient with both HIV infection and Amyotrophic Lateral Sclerosis (ALS) was treated with antiretrovirals, their ALS symptoms disappeared. This inspired him to investigate the effects of retroviruses on the nervous system, including development and neurodegeneration. However, no exogenous retroviruses were found to be directly associated with ALS. This prompted Nath to consider human endogenous retroviruses, commonly known as HERVs. These viruses comprise approximately 8% of the human genome and have historically been labeled "junk DNA". Contrary to this label, Nath's group found that expression of HERV-K in particular, which is the most recently acquired family of HERVs, is elevated in the brains of patients with ALS. Normally involved in early development of both the placenta and the embryo, expression of HERV-K in adult neurons results in neurotoxicity and subsequent neuron death. In the words of Nath, "the laws of nature are very simple: the very elements that put us together are the same ones to take us out". This discovery has prompted even more questions, and Nath and his group continues to decipher the mechanism of HERV-K induced neurodegeneration, searching for pathways that can be targeted by therapeutics to treat diseases like ALS.

Dr. Avindra Nath

The recent global SARS CoV-2 pandemic has shifted the focus of Nath and NINDS to the potential long-term neurological effects of COVID-19 infection. Nath explains that the realization that viruses such as Ebola and COVID-19 produce neurological effects is often too late for patients suffering from the downstream effects of the disease. Therefore, researchers must actively investigate the effects of the virus on the brain to develop methods of treatment. Nath's group has already described expression of the COVID-19 virus in the hippocampal neurons of patients, including infants. He says that "the myth that COVID does not affect kids is completely wrong". In fact, his group has found that neurological symptoms of COVID-19 infection are more common in youth, indicating that the phenomenon of Long-Haul COVID may severely impact the younger generations. Nath and NINDS have proposed a longitudinal study of 1000 adults presenting with recent acute infection to investigate the long-term syndromes associated with COVID-19. Avi Nath remains on the forefront of viral research, and his translational perspective is instrumental to medical care. For, in his own words, "There is no clinical advancement without basic science".

J. Spencer Hatfield is a graduate student in the Center for Human Genetics at Clemson University.

Genomic Evolution and Adaptation in Africa

by Debarati Majumdar

Dr. Sarah Tishkoff

Dr. Sarah Tishkoff, the David and Lyn Silfen University Professor in Genetics and Biology at the University of Pennsylvania from the University of Pennsylvania, presented a wonderful lecture on her research on genomic and phenotypic variation between different African tribes. Studies on genetic variation in Africa is of particular interest because all modern human fossils were discovered in Africa, from where people have migrated across the world. The other motivation for her research on African populations was differences in disease risk and health disparities in the United States. For example, hypertension is more common in people with African ancestry compared to other groups in the US. Genome wide association studies performed in 2019 showed that there is 2% African ancestry in the US. Polygenic risk studies based on subjects from European ancestries, however, perform poorly for disease risk prediction in east Asian and African populations.

Tishkoff and her team traveled across Africa to assess phenotypic diversity. They collected blood samples and gathered information about cardiovascular, lung and blood phenotypes, metabolic function, and infectious disease status used integrative evolutionary genomic analysis, including genomics, proteomics, epigenomics, transcriptomics, microbiome, and metabolomics analyses. They performed high coverage whole genome sequencing in 94 African individuals from 44 African populations. They also considered environmental factors like diet, exposure to toxins and pathogens and geography. Inferences were made about the population sizes, ancestry, and population divergence times over the years. Their studies showed that Africa has a huge diversity in ancestry.

Tishkoff's team collected measurements of waist, hip, and knee length, as well as BMI, grip, blood pressure, and lipids levels from about 2000 individuals from various tribes in Africa who were either hunter-gatherers, herders, or farmers. Anthropometric data show that the shortest people are the hunter-gatherers, and tallest being mostly the herders; the people with lowest BMI are herders and with highest BMI are farmers; waist circumference is seen to be lowest in hunter gatherers and highest for farmers; body fat is lowest for hunter gathers and highest for herders. C-peptide, an indicator of insulin levels, is lowest in hunter gatherers and highest in farmers. Estimates of heritability, a measurement of the fraction of phenotypic variation that is due to genetic variation, were high for skin color, but lower for pulse rate, blood pressure, oxygen and hemoglobin levels. Lipid analysis showed that farmers have higher levels of free fatty acids and herders have higher levels of triglycerides. Skin color is highly correlated with UV exposure. Migration out of Africa to high latitudes led to the development of pale skin and to lower latitudes of dark skin. To identify melanin levels, light was shined on the subject's arm and the wavelength of the reflected light was measured. The Botswana Sans tribe is the most lightly pigmented population and the Ethiopia Nilo-Saharan tribe are the most darkly pigmented people in Africa.

Genome-wide association analysis for skin pigmentation was performed on 1570 people and showed eight significant associations. The *SLC24A5* gene that imparts light skin colors in Europeans had the strongest association which suggests that there might have been a back migration to Africa. The second strongest association was seen with *MFSD12* which has previously been shown to have low levels of expression in depigmented skin of people with vitiligo. Mapping showed two independent associations - one within the *MFSD12* gene (associated with darker skin), and one in the enhancer (associated with lighter skin). Gene genealogy showed that ancestral allele is associated with light skin color. Another gene associated with variation in skin pigmentation, *DDB1* mediates DNA excision damage and when mutated gives rise to Xeroderma pigmentosum. Derived mutations are older than the age of modern humans. There might have been a convergent evolution of dark pigmentation in South Asia and Australo-Melanesia, but a haplotype study shows that this is due to out of Africa migration.

Debarati Majumdar is a graduate student in the Center for Human Genetics at Clemson University.

A Conversation with Julia George

Could you briefly describe your research program?

My lab is studying how prenatal communication from parent to offspring can alter the trajectory of development. This focus arises out of several decades of research on how songbirds communicate important information through vocal signals. In 2016 it was discovered that zebra finches, in their natural habitat in Australia, produce “heat calls” when they are incubating eggs, when the weather is hot. These heat calls serve as a sort of weather forecast for developing embryos, who cannot directly sense the weather while they are being incubated by their parents. After hatching, the chicks who were exposed to the heat calls from their parents are better adapted to hot weather. In my current research I want to understand 1) how embryos perceive this auditory cue and then 2) how it is translated into a physiological reprogramming.

Can you explain why you chose zebra finches to serve as a model organism in your lab?

Zebra finches are a great model for prenatal studies because embryonic development is happening in the egg. This makes the system very accessible to experimental manipulation. For example, we can artificially incubate the eggs and expose them to different sounds. We have much more control than if we were studying mammals, where the embryo develops inside the mother. Zebra finches are already a well-established experimental system for neurobiology and behavior, because of their exquisite capacity for vocal learning. We know a lot already about the brain circuits that underlie perception, learning, and production of birdsong. And zebra finches are highly social species, so they adapt well to living in groups in an aviary environment.

Why did you choose to come to Clemson University?

I chose to come to Clemson without ever setting foot on campus. Due to pandemic restrictions on travel (I moved from the UK) my interviews were conducted entirely via Zoom. I relied a lot on the reputation of Clemson and on my remote interactions with faculty and staff to make my decision. My colleagues at other institutions view Clemson as a rising force in research, and I was excited to be a part of that. Clemson has excellent students, and historically I have relied on strong graduate and undergraduate students to power my research. And the faculty and staff were and continue to be welcoming and helpful.

Briefly describe your collaboration efforts with the CHG as a faculty member on the main campus.

I am especially interested in collaborating on single-cell RNASeq experiments to detect the earliest responses to

heat calls in embryos. These studies are still in the planning stage, but I think there is a lot of potential there.

What is the most rewarding part of genetics research?

My favorite part of research is working with other people as part of a team. Group discussions can really stimulate creativity, and we can accomplish so much when we work together. I also think it’s fun to see our research published, but by the time we reach that point on a project, we are usually on to the next thing so it can be a bit anti-climactic!

Tell us about an interesting discovery or breakthrough in your lab.

We were studying neural plasticity related to song learning in zebra finches and discovered a gene with a unique pattern of expression in the song control circuit. We hypothesized that this gene (synuclein) would have a fundamental role in the function of the nervous system. In parallel, the same gene was found to be a component of pathological clumps (Lewy bodies) in Parkinson’s disease. We went on to show that synuclein is an abundant protein in the normal brain which plays a prominent role in normal vesicle trafficking. I think our work has encouraged researchers to consider how Parkinson’s disease might result from the disruption of normal cellular processes in neurons rather than focusing exclusively on protein aggregation.

What is your advice for young genetics investigators?

My advice is to really pay attention so-called “failed” experiments. Sometimes you may repeat an experiment again and again without getting the expected result. Consider that it may be telling you something novel! Be open to the unexpected!

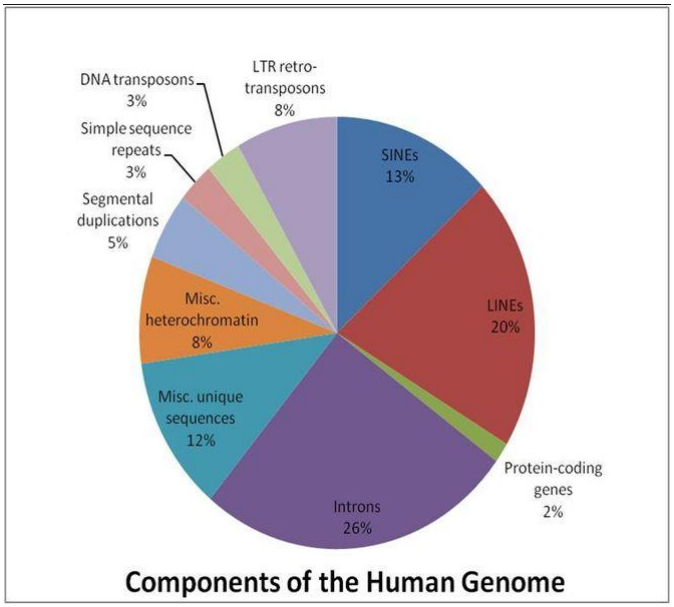
Viewpoint

The Genomic Trash Can: Is it Junk?

by Robert Anholt

The human genome is large, consisting of 6,200 Mbp for the diploid genome. The total length of all our DNA in all our cells would be ~6000 times the distance from the earth to the moon. That's a lot of DNA. Our chromosomes in each cell arranged head to tail would form a 2-meter-long string. But only ~2% of the genome codes for protein; that is a measly 4 centimeter. The remaining 1.96 meter has long been considered junk, useless DNA that accumulates and persists during evolution thought to escape purifying selection. Of course, sequencing of the human genome revealed that much of this non-coding DNA consists of transposable elements and regulatory regions that encode non-coding RNAs. In fact, those non-coding elements may themselves be important substrates for evolutionary change. But even if we account for transposons and noncoding regulatory RNAs, there is still a fair amount of "junk" left. As the British molecular biologist, Nessa Carey, wrote in her book *Junk DNA: A Journey Through the Dark Matter of the Genome*: "The more sophisticated an organism, the higher the percentage of junk DNA it contains."

The average mutation rate has been estimated at 175 mutations per diploid genome per generation. It is reasonable to assume, given the mutational targets of the protein coding and non-coding compartments of the genome, that most of these mutations land amidst the "junk". Some of this "junk" might have emerged from inactivation of



duplicated genes, which are no longer functional but persist in the genome. This raises an interesting possibility: Could our "junk" DNA serve as a reservoir for the recruitment of active protein coding genes or regulators?

Frameshift mutations that generate premature stop codons can lead to inactive genes that segregate as pseudogenes in a population. Multigene families with functional redundancy might be especially susceptible. For example, about two thirds of the ~1,000 olfactory receptor genes in the human genome are inactive pseudogenes and it has been suggested that mutations that restore functional open reading frames can generate functional receptors, providing a mechanism for potential rapid adaptation from pseudogenes that are cryptically hidden in the genomic garbage can.

Is it conceivable that mutations of the "junk" could result in reactivation of previously silent DNA resulting in the emergence of active genes that are now subject to selection? Yes, mutations are few and the likelihood that any given mutation would result in activation of a functional component in the silent DNA which can spread through the population is low, but there is time… hundreds and thousands and even millions of years. Thus, it is conceivable that over evolutionary time our genomic junk is evolvable and more dynamic than we may realize.

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

Trudy Mackay and **Robert Anholt**, in collaboration with Maria De Luca (University of Alabama, Birmingham, AL), received a one-year $271,382 grant from the National Institute on Aging to study the genetic basis of lifespan and healthspan extension by ACE inhibition in Drosophila.

Trudy Mackay, **Robert Anholt** and **Richard Steet** received a two-year $170,000 grant from the Cure Sanfilippo Foundation to identify disease modifiers in Drosophila models of MPSIIIA.

Seminars

On Friday, **February 4**, at 2:30 pm **Dr. James Lupski**, the Cullen Foundation Endowed Chair in Molecular Genetics at Baylor College of Medicine, will present a seminar titled "Biology in balance: human diploid genome integrity, gene dosage & genomic medicine."

On Friday, **February 25**, at 2:30 pm **Dr. Daniel Weinberger**, CEO and Director of the Lieber Institute for Brain Development, professor of Psychiatry, Neurology, and Neuroscience at Johns Hopkins University School of Medicine, and clinical professor of Psychiatry and Neurology at George Washington University School of Medicine, will present a seminar titled "Genomic insights into the developmental origins of behavioral disorders."

On Friday, **April 15**, at 2:30 pm **Dr. Wylie Burke**, Professor Emeritus and former Chair of the Department of Bioethics and Humanities at the University of Washington, member of the Fred Hutchinson Cancer Research Center, founder of the University of Washington Center for Genomics and Healthcare Equality, and past president of the American Society of Human Genetics, will present a seminar titled "Partnering with Communities in Genomic Research."

Publications

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

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