

SEX AS A BIOLOGICAL VARIABLE *Workshop*



October 26-27, 2017

Location:

NIH Main Campus
John Edward Porter Neuroscience Research Center
Building 35, Rooms 610/620/630 (and the Atrium)
35 Convent Drive, Bethesda, MD 20892

Keynote Speaker:

Dr. Virginia Miller, Ph.D.
Professor of Surgery and Physiology
College of Medicine, Mayo Clinic
“Sex-specific Risk For Cardiovascular Dysfunction
And Cognitive Decline”

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AGENDA

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National Institutes of Health
Main Campus
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Building 35, Rooms 610/620/630 (and the Atrium)
35 Convent Drive, Bethesda, MD 20892

AGENDA

DAY 1: Thursday, October 26, 2017

Please click the following link to participate virtually on Day 1: <https://videocast.nih.gov/summary.asp?live=26050&bhcp=1>

8:00 AM

Registration Check-In

8:30 AM - 8:45 AM

Introduction

Janine A. Clayton, MD, Director, Office of Research on Women's Health (ORWH), NIH
Elizabeth (Betsy) Wilder, PhD, Director, Office of Strategic Coordination (OSC)—The Common Fund, NIH

8:45 AM - 9:30 AM

Keynote Address: Sex-Specific Risk for Cardiovascular Dysfunction and Cognitive Decline

Keynote Presenter: *Virginia M. Miller, PhD*, Mayo Clinic, Rochester, MN

9:30 AM - 11:00 AM

SESSION 1: Sex Differences in Brain Function and Behavior

Moderator: *Paul Barrett, PhD*, OSC, NIH

9:35 AM - 9:55 AM

The Influence of Metabolic Syndrome and Sex on the DNA Methylome in Schizophrenia

Presenter: *Kyle J. Burghardt, PharmD*, Wayne State University, Detroit, MI

9:55 AM - 10:15 AM

Sex-Specific Metabolic and Epigenetic Changes in Primary Fibroblasts from Patients with Alzheimer's Disease

Presenter: *Eugenia Trushina, PhD*, Mayo Clinic, Rochester, MN

10:15 AM - 10:35 AM

Sex Differences in Brain Gut Interactions

Presenter: *Emeran A. Mayer, MD*, University of California, Los Angeles, CA

10:35 AM - 10:55 AM

Incorporating Sex as a Biological Variable in Understanding Brain Development

Presenter: *Margaret McCarthy, PhD*, University of Maryland School of Medicine, Baltimore, MD

10:55 AM - 11:15 AM

Networking Break

11:15 AM - 12:45 PM **SESSION 2: How Sex Impacts Our Interaction with External Influences**

Moderator: *Jennifer L. Troyer, PhD*, National Human Genome Research Institute (NHGRI), NIH

11:20 AM - 11:40 AM **Sex Stratification for Obesity and Related Traits in African Populations—H3Africa AWI-Gen Study**

Presenter: *Michèle Ramsay, PhD*, University of the Witwatersrand, Johannesburg

11:40 AM - 12:00 PM **Sex Differences in Pain Management**

Presenter: *Anne Z. Murphy, PhD*, Georgia State University, Atlanta, GA

12:00 PM - 12:20 PM **Sex Differences in Vaccine-Induced Immune Responses**

Presenter: *Nicole E. Basta, PhD*, University of Minnesota, Minneapolis, MN

12:20 PM - 12:35 PM ***Sex Differences in the Endocannabinoid System Determines Differences in Juvenile Rough-and-Tumble Play Behavior and Correlating Differences in Phagoptosis by Microglia in the Developing Amygdala**

Kathryn J. Argue, PhD, University of Maryland School of Medicine, Baltimore, MD

12:35 PM - 1:35 PM

Networking Lunch Break

(NIH will not provide any food or drinks, please make your own arrangements.)

1:45 AM - 3:00 PM **SESSION 3: Sex Differences in Animal Models**

Moderator: *Colin Fletcher, PhD*, NHGRI, NIH

1:50 PM - 2:10 PM **Sex in Perinatal Brain Damage: Why Can't Boys be More like Girls?**

Presenter: *Anna Penn, MD, PhD*, Children's National Medical Center, Washington, DC

2:10 PM - 2:30 PM **Control of Autoimmunity by Genes, Sex and the Microbiome**

Presenter: *Jayne Danska, PhD*, The Hospital for Sick Children, Toronto

2:30 PM - 2:50 PM **Sex and Mouse Brain Anatomy during Health and Treatment**

Presenter: *Brian Nieman, PhD*, The Hospital for Sick Children, Toronto

2:50 PM - 3:05 PM ***Estrogen Contributes to Sex Differences in M2-Polarization during Asthma**

Presenter: *Aleksander Keselman, PhD*, Johns Hopkins University, Baltimore, MD

3:05 PM - 3:15 PM

Group Photo of Speakers with the Workshop Organizers

3:05 PM - 3:30 PM

Networking Break

(Poster presenters finalize set-up for Poster Session)

3:30 PM - 5:30 PM **SESSION 4: Poster Session**

Moderators: *Rajeev K. Agarwal, PhD*, ORWH, NIH, and *Paul Barrett, PhD*, OSC, NIH

5:30 PM

Day 1 of the Workshop Adjourns

*Abstracts selected for both Oral and Poster Presentation

DAY 2: Friday, October 27, 2017

Please click the following link to participate virtually on Day 2: <https://videocast.nih.gov/summary.asp?live=26054&bhcp=1>

- 8:00 AM **Registration Check-In**
- 8:30 AM - 10:00 AM** **SESSION 5: Sex Differences in Gene Expression**
Moderator: *Rajeev K. Agarwal, PhD*, ORWH, NIH
- 8:35 AM - 8:55 AM **UTI Complexity Results from Diversity at the Bacterial-Host Interface**
Presenter: *Scott Hultgren, PhD*, Washington University School of Medicine, St. Louis, MO
- 8:55 AM - 9:15 AM **Sex Differences in Pathogenesis of Urinary Tract Infection**
Presenter: *David A. Hunstad, MD*, Washington University School of Medicine, St. Louis, MO
- 9:15 AM - 9:35 AM **X, Drugs, Rock, an' Roles...**
Presenter: *Lauren A. Weiss, PhD*, University of California, San Francisco, CA
- 9:35 AM - 9:55 AM **Sex-Specific Genetic Architecture of the Human Transcriptome**
Presenter: *Barbara Stranger, PhD*, University of Chicago, Chicago, IL
- 9:55 AM - 10:15 AM **Networking Break**
- 10:15 AM - 11:00 AM** **SESSION 6: Panel Discussion on the Lessons Learned from Accounting for SABV**
Moderator: *Judy Hewitt, PhD*, National Institute of Allergy and Infectious Diseases (NIAID), NIH
- Panel Members:
Emeran A. Mayer, MD, University of California, Los Angeles, CA
Margaret M. McCarthy, PhD, University of Maryland School of Medicine, Baltimore, MD
Virginia M. Miller, PhD, Mayo Clinic, Rochester, MN
Brian Nieman, PhD, The Hospital for Sick Children, Toronto
Michèle Ramsay, PhD, University of the Witwatersrand, Johannesburg
- 11:15 AM - 11:30 AM **Closing Remarks**
James (Jim) M. Anderson, MD, PhD, Director, Division of Program Coordination, Planning, and Strategic Initiatives, Office of the Director (OD), NIH
- 11:30 AM **2017 SABV Workshop Adjourns**
- 11:45 AM - 12:30 PM **Round Table Discussion with Speakers [CLOSED SESSION—Open only to Invited Speakers, Moderators, NIH Leadership, and staff members of OSC and ORWH]**
- Interesting topics
 - Future of the field/needs
 - White paper plans to highlight symposium

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SPEAKER BIOGRAPHIES



SPEAKER BIOGRAPHIES

Nicole E. Basta, PhD is an Epidemiologist who combines expertise in epidemiology, biostatistics, immunology, population biology, ecology, and public health to design and evaluate preventive measures to reduce the burden of infectious diseases. Her research aims to quantify the impact of vaccines and vaccination programs, develop targeted vaccination strategies, increase vaccine acceptance, and improve vaccine uptake.

Kyle J. Burghardt, PharmD is an Assistant Professor of Pharmacy Practice at the Eugene Applebaum College of Pharmacy and Health Sciences at Wayne State University. After completing his PharmD at the University of Michigan he completed a 3-year postdoctoral fellowship in pharmacogenomics and psychiatry with Dr. Vicki Ellingrod. He currently directs a pharmacogenomics lab at Wayne State University and studies the skeletal muscle and adipose molecular mechanisms of antipsychotic-induced insulin resistance. He teaches several courses in the pharmacy curriculum including psychiatry therapeutics, applied kinetics, and pharmacogenomics.

Jayne Danska, PhD is a Senior Scientist in the Genetics and Genome Biology Program of The Hospital for Sick Children, and a Professor in the Departments of Immunology and Medical Biophysics at the University of Toronto Faculty of Medicine. Dr. Danska's research interest is on the genetic and immunological basis of type 1 diabetes and on molecular mechanisms of acute lymphoblastic leukemia in animal models and in humans. She is currently the lead investigator on a Genome Canada project in type 1 diabetes and co-investigator on two collaborative projects on lymphoblastic leukemia. Dr. Danska has chaired and served on grant panels including the National Cancer Institute of Canada, the Canadian Institutes of Health Research, the Canadian Diabetes Association, and the National Institutes of Health (USA). Dr. Danska is a recipient of the NCIC Scientist Award and the Premier's Research Excellence Award.

Scott Hultgren, PhD is the Helen Lehbrink Stoeber Professor of Molecular Microbiology and Director of the Center for Women's Infectious Disease Research at Washington University in St. Louis, and a member of the National Academy of Sciences. He earned his PhD at Northwestern University and did postdoctoral training at Umeå University in Sweden.

David A. Hunstad, MD is Associate Professor and Chief of the Division of Pediatric Infectious Diseases at Washington University in St. Louis. Dr. Hunstad is an NIH-funded pediatric physician-scientist studying pathogenesis of Gram-negative bacterial infections and has contributed to the development of new mouse models for the study of sex differences in pathogenesis and host response to urinary tract infections.

Emeran A. Mayer, MD is a Professor of Medicine and directs the ORWH Specialized Center for Neurovisceral Sciences & Women's Health at the University of California, Los Angeles. He is a pioneer and a leading researcher in the role of mind-brain-gut interactions in health and chronic disease, with a particular interest in sex-related differences.

Margaret M. McCarthy, PhD received a PhD from the Institute of Animal Behavior at Rutgers University, did postdoctoral training at Rockefeller University, and was a National Research Council Fellow at NIAA before joining the faculty of the University of Maryland School of Medicine in 1993. She was a professor in the Department of Physiology before becoming the Chair of the Department of Pharmacology in 2011. She has received numerous awards and recognition for her mentoring of graduate students. Dr. McCarthy has a longstanding interest in the cellular mechanisms that establish sex differences in the brain. She uses a combined behavioral and mechanistic approach in the laboratory rat to understand both normal brain development and how these processes might go selectively awry in males versus females. She is currently President of the Organization for the Study of Sex Differences, Associate Editor of *Hormones and Behavior*, and serves on the Advisory Board of eNeuro. She also serves on the Board of Scientific Councilors of the National Institute of Mental Health and was named one of Maryland's Top 100 Women in 2009.

Virginia M. Miller, PhD is Professor of Surgery and Physiology and Director of the Women's Health Research Center at the Mayo Clinic. She was President of the Organization for the Study of Sex Differences, has received numerous awards, and actively advocates for the inclusion of sex and gender variables in basic and clinical research and medical curricula.

Anne Z. Murphy, PhD is an Associate Professor in the Neuroscience Institute at Georgia State University. Her NIH-funded research focuses on the impact of sex and age on pain and opiate responsiveness as well as the long-term consequences of early life experience on adult pain, stress, and immune response.

Brian Nieman, PhD is currently a Senior Scientist at the Mouse Imaging Centre of The Hospital for Sick Children and a Level 2 Investigator with the Ontario Institute for Cancer Research. He obtained his PhD at the University of Toronto and completed postdoctoral work at the New York University School of Medicine.

Anna Penn, MD, PhD is an Associate Professor, clinical neonatologist, and neuroscientist at Children's National Medical Center in Washington, DC. She directs the Board of Visitors Perinatal Neuroprotection Program and runs a research program focused on preterm brain injury in relation to placental function.

Michèle Ramsay, PhD is the Director of the Sydney Brenner Institute for Molecular Bioscience (SBIMB) and Professor in the Division of Human Genetics, University of the Witwatersrand, Johannesburg, South Africa. Research interests include African population diversity and its role in disease susceptibility. Dr. Ramsay also serves as the Principal Investigator of H3Africa Collaborative Center (AWI-Gen), President of the African Society of Human Genetics, supervisor, and mentor.

Barbara Stranger, PhD develops effective analytic approaches for large-scale analysis of human genomics data, particularly transcriptome and genetic variation data, in the context of health and disease. She applies systems biology methodologies to integrate data of different types to inform biology of complex traits.

Eugenia Trushina, PhD is Associate Professor in the Department of Neurology at the Mayo Clinic. Her interests include the role that mitochondria and cellular metabolism play in neurodegenerative processes related to Huntington's, Alzheimer's, and Parkinson's diseases, chemotherapy-induced peripheral neuropathy, multiple sclerosis, and neurodegeneration caused by environmental toxicants.

Lauren A. Weiss, PhD is a human geneticist with a strong interest in complex genetic mechanisms of autism spectrum disorders. She has a BS in human genetics from the University of Michigan, where she first developed her interest in autism genetics studying sodium channel subunit genes with Dr. Miriam Meisler. She earned a PhD in human genetics from The University of Chicago, where she first became interested in the role of gene-sex interaction in quantitative trait genetics with Drs. Carole Ober and Edwin H. Cook, Jr. She did a postdoctoral fellowship in psychiatric genetics jointly at the Massachusetts Institute of Technology and Harvard University with Drs. Pamela Sklar and Mark Daly, before joining the faculty at the University of California, San Francisco in 2008. Her laboratory in the Department of Psychiatry, the Institute for Human Genetics, and the Weill Institute for the Neurosciences utilizes analytical approaches and human iPSC models to understand the role of gene-sex interaction, gene-environment interaction, gene-gene interaction, and gene-phenotype relationships in autism and related traits.

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MODERATOR BIOGRAPHIES

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MODERATOR BIOGRAPHIES

Rajeev K. Agarwal, PhD is a Senior Research Program Officer in the Office of Research on Women's Health (ORWH) at the NIH. He manages two flagship programs: Specialized Centers of Research (SCOR), a P50 program related to research on sex differences; and an Administrative Supplement program for research on sex/gender influences. He is also a Project Scientist in Environmental influences in the Child Health Outcomes (ECHO) program and represents ORWH in two Common Fund Programs: Human Health and Heredity in Africa (H3Africa); and Knockout Mouse Phenotyping (KOMP). Before joining ORWH, he was a Program Director in the Division of Cancer Treatment and Diagnosis (DCTD) at the National Cancer Institute, overseeing a P50 cancer research portfolio known as the Specialized Programs of Research Excellence (SPOREs). Dr. Agarwal has knowledge and expertise in immunology, molecular biology, autoimmune diseases, infectious diseases, biomarkers, and cancer research. He also has extensive experience as a grant administrator on complex multicomponent research projects related to inter/multidisciplinary basic, clinical, and translational research.

Paul Barrett, PhD joined the Common Fund in the Office of Strategic Coordination (OSC) in 2016 as a Health Specialist. After receiving a BS in chemistry from SUNY Binghamton, Dr. Barrett worked as a laboratory technician at SUNY Stony Brook in a clinical research laboratory looking for novel, non-invasive biomarkers in bladder, prostate, and kidney cancers from human patients. Dr. Barrett continued his research career at Vanderbilt University, where he obtained a PhD in biochemistry. His work utilized nuclear magnetic resonance spectroscopy and other biochemical and biophysical assays to study the structure and function of membrane proteins and lipids in Alzheimer's disease. He then served as a Post-Doctoral Associate at the University of Pittsburgh, where he investigated how protein/protein interactions disrupt mitochondrial function and contribute to neuronal death in Parkinson's disease using in vitro and in vivo model systems. Dr. Barrett now works as a policy, planning, communication, and evaluation specialist for several NIH Common Fund programs.

Colin Fletcher, PhD supervises the Knockout Mouse Project (KOMP). KOMP aims to produce and phenotype a comprehensive collection of mouse KO strains, including cataloging sexual dimorphism. Dr. Fletcher received an AB from Dartmouth University and a PhD from the Rockefeller Institute. He has been working in the field of mouse genetics since 1989.

Judy Hewitt, PhD is Chief of the Research Resources Section in the Office of Biodefense, Research Resources, and Translational Research at the National Institute of Allergy and Infectious Diseases. Her group provides reagents, animal models, and screening for infectious diseases. In 2014-15, she served as point person for NIH's extramural policy to enhance reproducibility through rigor and transparency.

Jennifer L. Troyer, PhD is the Program Director at the National Human Genome Research Institute. As part of a team, she administers Human Health and Heredity in Africa (H3Africa), a Common Fund (trans-NIH) initiative that facilitates application of contemporary research approaches to the study of genomics and environmental determinants of common diseases with the goal of improving the health of African populations. Her research has ranged from cats to lions to humans, but is primarily focused on genetic variations in the virus and host that alter the outcome of infection. She has experience in leading genome-wide association studies and has participated in international consortium efforts to identify host restriction factors for HIV/AIDS.



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ORAL PRESENTATION ABSTRACTS



ORAL PRESENTATION ABSTRACTS

***Sex Differences in the Endocannabinoid System Determines Differences in Juvenile Rough-and-Tumble Play Behavior and Correlating Differences in Phagoptosis by Microglia in the Developing Amygdala**

Kathryn Jean Argue, PhD
University of Maryland School of Medicine

The amygdala is a sexually dimorphic brain region that is critical for the expression of social behavior. In rats, the medial amygdala mediates the sex difference in juvenile rat play behavior. During early postnatal development, the male amygdala has a higher endocannabinoid (ECB) tone and contains fewer newborn cells than females, which correlates to increased levels of juvenile rough-and-tumble play. Masculinization of the female developing amygdala through agonism of the CB1 and CB2 ECB receptors, decreases the number of newborn cells and increases subsequent juvenile rough-and-tumble play (Krebs-Kraft et al. 2010 and Argue et al. 2017). We now report that the higher ECB tone in males is due to the actions of testosterone. Treating neonatal females with exogenous testosterone was sufficient to increase the ECB tone and juvenile rough-and-tumble play. This testosterone-induced increase in female rough-and-tumble play could be ablated with neonatal administration of CB1 and CB2 receptor antagonists. Additionally, microglia, the resident immune cells of the brain, are more phagocytic in the amygdala of males during this postnatal window, suggesting a possible mechanism by which ECBs affect the number of newborn cells. We find that males have more phagocytic microglia between postnatal day 0 and 4, during which time they also have higher ECB tone than females. Increasing ECB tone in female pups by treatment with specific agonists for either CB1 or CB2 receptors increases the number of phagocytic microglia to that of males and results in a corresponding decrease in the number of newborn cells indicated by BrdU labeling. We hypothesize that microglia control the number of newborn cells in the postnatal rat amygdala by phagoptosing (targeted phagocytosis of viable cells) newborn cells in an ECB-dependent manner. Masculinizing female pups with testosterone increases the number of phagocytic microglia and decreases the number of BrdU+ cells. Immunohistochemical analysis together with confocal microscopy indicates phagocytic microglia contain markers of DNA as well as newly proliferated cells in their phagocytic cups. To directly implicate microglia phagoptosis we utilized a complement receptor 3 (CR3) function-blocking antibody to inhibit phagocytosis, which increased the number of BrdU+ cells in both males and females. Thus microglia actively control developmental sex differences in cell genesis and this correlates with sex differences in later life social behavior. We are currently investigating whether juvenile rough-and-tumble play behavior can be mediated through direct manipulation of the microglia during the neonatal period.

**This oral presentation will also be a poster presentation.*

Sex Differences in Vaccine-Induced Immune Responses

Nicole E. Basta, PhD
University of Minnesota

Understanding the distribution, causes, and consequences of heterogeneity in vaccine-induced immune responses is critical to ensuring that protective levels of immunity persist over time. Sex differences in immune responses following pneumococcal, influenza, measles, and other vaccinations have been reported and may contribute significantly to the variation observed. Yet, for many recently developed vaccines, little is known about whether sex differences are an important driver of variation in immune responses. We designed and implemented a longitudinal study, launched in 2012 in Bamako, Mali, to evaluate changes in immune responses following the introduction of the new meningococcal A polysaccharide-tetanus toxoid conjugate vaccine (MenAfriVac). We investigated sex-specific differences in immune responses two years after vaccination and found that females had higher meningococcal A-specific serum bactericidal antibody titers, a marker of functional immunity and the accepted correlate of protection, in all age groups <18 years. In addition, we demonstrated that while MenAfriVac vaccination boosts tetanus toxoid IgG, a potentially important benefit in countries where maternal and neonatal tetanus remains a concern, sex-specific differences in tetanus immunity before and after MenAfriVac introduction suggest the need for additional, targeted vaccination. Our ongoing research is aimed at investigating the relationship between vaccine-induced immunity and nutritional status and markers of inflammation to determine whether these factors contribute to the sex differences observed.

The Influence of Metabolic Syndrome and Sex on the DNA Methylome in Schizophrenia

Kyle J. Burghardt, PharmD
Wayne State University Eugene Applebaum College of Pharmacy and Health Sciences

INTRODUCTION: The rate of metabolic syndrome within schizophrenia is 2-3 times higher compared to the general population, which may be partly due to the metabolic side effects of atypical antipsychotics (AAPs). The mechanism by which AAPs increase the risk of metabolic syndrome is not completely known; however, previous work suggests that the folate system, which is involved in the production of methyl groups used in DNA methylation, may be involved. Within this study, the DNA methylome was profiled to identify altered methylation associated with metabolic syndrome in a schizophrenia population and based on sex.

METHODS: Cross-sectional peripheral blood from schizophrenia subjects were utilized for DNA methylation analyses. Discovery analyses (n=49 males and n=47 females) were performed using an epigenome-wide analysis on the Illumina HumanMethylation450 BeadChip. The top hits were analyzed in an additional validation set (n=166) using the gold-standard of site-specific methylation pyrosequencing.

RESULTS: Differential methylation was found within the MAP3K13 gene in females and the CCDC8 gene in males. Significant differences in methylation were validated for the MAP3K13 genes, but not CCDC8, in the validation sample set.

CONCLUSIONS: Differential DNA methylation was identified based on metabolic syndrome, which was also specific to sex. Further prospective work is needed to determine whether DNA methylation could serve as a useful biomarker or treatment target for metabolic syndrome associated with antipsychotic use.

Control of Autoimmunity by Genes, Sex and the Microbiome

Jayne Danska, PhD
The Hospital for Sick Children

Immune-mediated diseases, including type 1 diabetes (T1D), multiple sclerosis, and inflammatory bowel disease, result from the interaction between genetic variants at many loci with poorly understood environmental factors. Over the past 60 years, multiple immune-mediated diseases have seen a dramatic rise in frequency. For example, T1D has increased by >500% in developed countries over this period. In addition to environmental risk factors, the incidences of many autoimmune syndromes display sex bias in incidence and severity, but the mechanisms of sexually dimorphic immune regulation are poorly understood. In the majority of rodent models of autoimmunity, only females are studied because they display more severe effects.

The non-obese diabetic (NOD) mouse displays spontaneous, immune-mediated pancreatic beta cell destruction causing diabetes with both a genetic and environmental etiology. Disease in the NOD mouse is twice as frequent in females compared to males and is also impacted by with hygiene status. By studying both sexes, our work has revealed mechanisms by which the gut microbial community (the microbiome) influences sex hormones, metabolites, and autoimmunity in the NOD model. A focus of our current effort is to dissect the sex chromosome and hormonal mechanisms that regulate the gut microbiome and the function of the gut-associated immune system.

UTI Complexity Results from Diversity at the Bacterial-Host Interface

Scott Hultgren, PhD
Washington University School of Medicine

Our studies blend multiple scientific disciplines elucidating bacterial and host mechanisms that determine the onset, course, and outcome of interactions between uropathogens and host tissues. We have illuminated bacterial mechanisms, intracellular lifestyles, and community behaviors, which play critical roles in urinary tract infection (UTI). We have shown that risk of UTI depends on specific pairing between diverse uropathogens and host susceptibility, with the outcome depending on both bacterial gene carriage and transcriptional responses, and complex host mucosal response networks, governed by disease history and host sex factors. Our work is changing the way UTIs are evaluated and is informing the design of novel vaccines and therapeutics.

Sex Differences in Pathogenesis of Urinary Tract Infection

David A. Hunstad, MD
Washington University School of Medicine

Preclinical modeling of urinary tract infections (UTIs) has been limited to females. We devised a new technique for bladder inoculation with uropathogenic *Escherichia coli* (UPEC) in both male and female mice, enabling first-ever studies of sex differences in UTI. Male mice display striking susceptibility to chronic cystitis, severe pyelonephritis, and renal abscess (rare in female mice); castration or genetic deficiency of the androgen receptor (AR) prevents severe UTI. In female mice, dihydrotestosterone treatment potentiates severe UTI, while the AR antagonist enzalutamide is protective. These data mirror reports of elevated UTI risk in women with polycystic ovary syndrome, a common hyperandrogenic state. Our work illuminates sex influences on urogenital infections and suggests androgen modulation as a therapeutic strategy in recalcitrant or recurrent UTI.

***Estrogen Contributes to Sex Differences in M2-Polarization During Asthma**

Aleksander Keselman, PhD
Johns Hopkins University

Asthma exhibits sex differences, affecting mostly boys in childhood and women in adulthood. Alveolar macrophages have emerged as major mediators of allergic lung inflammation. We hypothesized that estrogen enhances the M2 polarization of alveolar macrophages to promote asthma. We found M2-gene expression to be elevated in alveolar and bone-marrow derived macrophages (BMMs) from female mice after challenge with allergen and stimulation with IL-4, respectively. Pretreatment of female BMMs with estrogen receptor ligands enhanced IL-4-induced M2-gene expression. Ovariectomized and LysMCRE ERa flox/flox mice exhibited impaired M2 responses after challenge with OVA. Together these data suggest that sex and hormonal factors contribute to sex differences in macrophage responses during asthma.

**This oral presentation will also be a poster presentation.*

Sex Differences in Brain Gut Microbiome Interactions

Emeran A. Mayer, MD
University of California, Los Angeles

Even though brain gut microbiome (BGM) interactions play a crucial role in the homeostasis of the organism, few studies have addressed sex-related differences in these interactions. To test this general hypothesis, we performed several studies aimed to characterize BGM interactions in subjects with chronic visceral pain (irritable bowel syndrome [IBS]), with obesity and in healthy controls. Behavioral data, multimodal brain imaging, and microbiome data were obtained in the three groups, and analyses of brain network connectivity, gut microbial community structure, and microbial metabolites were performed. Female IBS subjects showed lower sympathetic nervous system responses, lower HPA axis responses, and greater perceptual responses to an aversive visceral stimulus, compared to males. At the brain level, females showed more prominent sensorimotor structural and functional alterations, while males showed more prominent changes in the salience network. At the microbiome level, significant differences in

plasma levels of several microbial-derived tryptophan metabolites were observed. In obese subjects, significant sex-related differences were seen in anatomical and functional network connectivity involving cortical and subcortical regions, in addition to differences in gut microbial metabolites. The observed sex-related differences within the BGM axis may have implications for personalized treatment approaches.

Incorporating Sex as a Biological Variable in Understanding Brain Development

Margaret McCarthy, PhD
University of Maryland School of Medicine

Most people associate sex differences in brain and behavior with adult circulating hormones that can vary dramatically between males and females. But many adult sex differences are actually established early in development, beginning in utero and extending post-natally, as a result of elevated androgens in males that derive from the fetal testis. This early life programming has enduring effects that are often not manifest until the animal matures and is capable of exhibiting complex motivated and cognitive behaviors. Parsing out the contribution of biology to sex differences in adult behavior is complicated by the impacts of environment and experience, which have accumulated during the maturational period. Study of the immature brain provides an opportunity to more tightly control extraneous variables and thereby more cleanly delineate the impact of nature versus nurture. Our research program has focused on the molecular mechanisms by which early hormone exposure in males differentiates the neuroanatomical and genomic substrate. We have identified novel sources of modulation of synaptogenesis, synaptic pruning, cell genesis, and differentiation. These include unexpected roles for prostaglandins, endocannabinoids, microglia, and other immune cells. We have also found a profound effect of steroid exposure early in life on the epigenome of the brain and speculate this is a mechanism by which sex differences are established and endure. Our work highlights latent variables that may increase the risk of males to developmental neuropsychiatric and neurodevelopmental disorders while protecting females from the same. Supported by R01 DA039062 and R01MH052716.

Sex-Specific Risk for Cardiovascular Dysfunction and Cognitive Decline

Virginia M. Miller, PhD
Mayo Clinic

Women who experience a preeclamptic pregnancy had greater carotid intima-media thickness (CIMT), coronary artery calcification, lower cerebrovascular reactivity, and greater cognitive impairment and cortical atrophy than women who experienced normotensive pregnancies. In healthy, post-menopausal women neither transdermal 17β -estradiol nor oral conjugated equine estrogen affected CIMT during or within 3 years following the menopausal hormone treatment (MHT). Treatment effects were influenced by genetic variants associated within genes of the innate immune system, estrogen metabolism, and APO ϵ 4. Understanding the impact of pregnancy outcomes on risk of chronic diseases of aging and the therapeutic potential of MHT to alleviate them helps to inform personalized care of women as they age.

Sex Differences in Pain Management

Anne Z. Murphy, PhD
Georgia State University

Chronic pain is one of the most commonly reported health problems in the United States, affecting approximately 25% of the population. As clinicians advance toward more individualized treatment strategies for pain, the importance of biological sex is increasingly clear. Women have a higher incidence rate of chronic pain conditions, and in particular, those that include an inflammatory component, such as fibromyalgia and osteoarthritis. Morphine has been and continues to be one of the most effective drugs for the treatment of pain. However, preclinical studies have repeatedly demonstrated that morphine is a more effective analgesic in males than in females. Clinical studies examining sex differences in analgesia are more varied. This talk will discuss the various central mechanisms that contribute to the dimorphic effects of morphine.

Sex and Mouse Brain Anatomy during Health and Treatment

Brian Nieman, PhD
The Hospital for Sick Children

The brain undergoes extensive and prolonged development after birth with subtle anatomical differences between males and females. Events that affect this development often also show sex dependence. Children treated for cancer at an early age frequently experience lasting cognitive or behavioral difficulties, with females more susceptible than males. Using three-dimensional imaging to assess anatomy, we have similarly demonstrated sexual dimorphism in brain structure and regional volume changes in the brain after cancer treatment using a mouse model. The role of inflammatory response after cranial radiation, in particular, was investigated and shown to vary by sex.

Sex in Perinatal Brain Damage: Why Can't Boys Be More Like Girls?

Anna Penn, MD, PhD
Children's National Medical Center

Ten percent of all infants in the United States are born preterm, and more extremely preterm infants are being saved, so there is an increasing need to understand the factors contributing to brain damage in these infants. Despite improvements in survival, survivors remain at high risk for cerebral palsy and mental retardation, learning disability, and later psychiatric illness. Even in the absence of obvious structural brain injury, a significant number of infants born very preterm have cognitive deficits and behavioral problems. *Males are at particularly high risk.* A key contributor to this damage may be loss of placental hormones, including sex steroids and other hormones. To understand the placental hormone contribution to perinatal brain injury, and specifically to increased male vulnerability, we are pursuing investigations that range from the development of novel mouse models to use of human infant data. Our overall goal is to understand hormones that contribute to normal neurodevelopment, the effects of their loss following premature birth, and their potential as protective agents.

Sex Stratification for Obesity and Related Traits in African Populations—H3Africa AWI-Gen Study

Michèle Ramsay, PhD
University of the Witwatersrand, Johannesburg

The health and epidemiological transition across Africa is evident in the rising tide of obesity and its impact on hypertension and diabetes. Unlike the developed world, the sex disparity is marked in Africa, with women disproportionately affected. In a multi-center African study across four countries, obesity (BMI \geq 30) was highest (67%) among women in urban South Africa (42% men) and lowest in rural Burkina Faso (2.2% in women; 1.8% in men), where 31% of women (17% men) are underweight (BMI $<$ 18.5). These differences result from complex interactions between fixed (sex, age, genetic variation, biology) and modifiable (behavior, sociodemographic) risk factors. We need effective interventions to ameliorate the impact of obesity on population health.

Sex-Specific Genetic Architecture of the Human Transcriptome

Barbara Stranger, PhD
University of Chicago

Gene regulation may contribute to sexually dimorphic complex traits. We have characterized the role of sex in inter-individual transcriptional variation in tissues of the Genotype-Tissue Expression (GTEx) Project. Our goals are to (a) identify genes and regulatory networks differentially expressed (DE) between sexes, (b) identify and characterize sexually dimorphic expression quantitative trait loci (eQTLs), (c) pinpoint differences in sex-biased eQTL profiles between autosomal and sex chromosomes, and (d) determine the variation of sex biases across tissues and their contribution to disease. We present highlights of this ongoing work.

Sex-Specific Metabolic and Epigenetic Changes in Primary Fibroblasts from Patients with Alzheimer's Disease

Eugenia Trushina, PhD
Mayo Clinic

Complex changes that occur in Alzheimer's disease (AD) patients and the lack of understanding of early disease mechanisms have hindered the development of efficacious therapeutic interventions. Systems biology offers an outstanding opportunity to monitor changes involved in AD development in multiple functionally connected pathways using readily available biofluids or primary cells such as fibroblasts. We applied non-targeted and targeted metabolomics, stable isotope tracers, and next generation sequencing to establish to what extent metabolic and epigenetic changes in fibroblasts from late onset AD patients recapitulate alterations established previously in CSF, plasma, and postmortem brain tissue from individuals with mild cognitive impairment and AD. Our data revealed sex- and disease-specific signatures validating the use of human fibroblasts to study AD mechanisms.

X, Drugs, Rock, an' Roles...

Lauren A. Weiss, PhD
University of California, San Francisco

Sexual dimorphism in common complex genetic disease is extensive; the male predominance of autism spectrum disorders is one example of a longtime observation that remains largely unexplained. Possible genetic mechanisms include a disproportionate role for sex chromosomes, a substantial role for hormones (potentially via effects on gene expression), or different thresholds determining sufficient genetic liability. In addition, it is unknown whether the sex differences in autism are specific to its behavioral or neurodevelopmental features or will be representative of general sexual dimorphism in biology. We designed a study to evaluate evidence supporting these hypotheses utilizing common polymorphism genome-wide association study data.





ABSTRACTS
for
POSTER
SESSION



ABSTRACTS for POSTER SESSION

***POSTER #1: Sex Differences in the Endocannabinoid System Determines Differences in Juvenile Rough-and-Tumble Play Behavior and Correlating Differences in Phagoptosis by Microglia in the Developing Amygdala**

Abstract submitter: Kathryn Jean Argue

Kathryn Jean Argue¹; Jonathan W. VanRyzin¹; Margaret M McCarthy¹

¹University of Maryland School of Medicine

The amygdala is a sexually dimorphic brain region that is critical for the expression of social behavior. In rats, the medial amygdala mediates the sex difference in juvenile rat play behavior. During early postnatal development, the male amygdala has a higher endocannabinoid (ECB) tone and contains fewer newborn cells than females, which correlates to increased levels of juvenile rough-and-tumble play. Masculinization of the female developing amygdala through agonism of the CB1 and CB2 ECB receptors, decreases the number of newborn cells and increases subsequent juvenile rough-and-tumble play (Krebs-Kraft et al. 2010 and Argue et al. 2017). We now report that the higher ECB tone in males is due to the actions of testosterone. Treating neonatal females with exogenous testosterone was sufficient to increase the ECB tone and juvenile rough-and-tumble play. This testosterone-induced increase in female rough-and-tumble play could be ablated with neonatal administration of CB1 and CB2 receptor antagonists. Additionally, microglia, the resident immune cells of the brain, are more phagocytic in the amygdala of males during this postnatal window, suggesting a possible mechanism by which ECBs affect the number of newborn cells. We find that males have more phagocytic microglia between postnatal day 0 and 4, during which time they also have higher ECB tone than females. Increasing ECB tone in female pups by treatment with specific agonists for either CB1 or CB2 receptors increases the number of phagocytic microglia to that of males and results in a corresponding decrease in the number of newborn cells indicated by BrdU labeling. We hypothesize that microglia control the number of newborn cells in the postnatal rat amygdala by phagocytosing (targeted phagocytosis of viable cells) newborn cells in an ECB-dependent manner. Masculinizing female pups with testosterone increases the number of phagocytic microglia and decreases the number of BrdU+ cells. Immunohistochemical analysis together with confocal microscopy indicates phagocytic microglia contain markers of DNA as well as newly proliferated cells in their phagocytic cups. To directly implicate microglia phagoptosis we utilized a complement receptor 3 (CR3) function-blocking antibody to inhibit phagocytosis, which increased the number of BrdU+ cells in both males and females. Thus microglia actively control developmental sex differences in cell genesis and this correlates with sex differences in later life social behavior. We are currently investigating whether juvenile rough-and-tumble play behavior can be mediated through direct manipulation of the microglia during the neonatal period.

****This poster presentation has also been selected for oral presentation.***

POSTER #2: Lateral Habenula-Induced Inhibition of Midbrain Dopamine Neurons in Male and Female Rats

Abstract submitter: P. Leon Brown

P. Leon Brown¹; Dana Brady¹

¹University of Maryland School of Medicine

There are consistent sex differences in the progression from illicit drug use to abuse, potentially due to differences in dopaminergic circuits that mediate reward. Dopamine neurons are transiently inhibited by the lateral habenula, an epithalamic structure activated by aversive stimuli and previously shown to contain estrogen receptors. We test the hypothesis that lateral habenula inhibitory control over dopamine neurons in female rats is reduced relative to males. Preliminary findings demonstrate an elevated baseline firing rate of dopamine neurons in female rats, but no difference in firing regularity or pattern. Although a majority of dopamine neurons are inhibited in both sexes, the duration of inhibition is reduced in female rats. Such dampened inhibitory control may diminish the aversive components of drug use, resulting in increased abuse potential.

POSTER #3: Characterization of Idiopathic Bronchiectasis in Patients with and without Pulmonary Nontuberculous Mycobacterial Disease

Abstract submitter: S. Daniel-Wayman

J. A. Nguyen¹; S. Daniel-Wayman¹; D. R. Prevots¹; J. Adjemian¹; M. Daniels²; C. L. Daley³; M. Knowles²; K. N. Olivier¹

¹National Institutes of Health, ²University of North Carolina at Chapel Hill, ³National Jewish Health

Idiopathic bronchiectasis describes abnormal airway widening and stretching without a known cause, leading to mucus build-up, infection, and respiratory complications. Bronchiectasis has been reported more commonly in postmenopausal women with a tall, thin body morphotype and is associated with pectus abnormalities, scoliosis, and nontuberculous mycobacterial (NTM) pulmonary infection. The link between bronchiectasis, gender, and these traits remains unclear.

METHODS: The Genetic Disorders of Mucociliary Clearance Consortium idiopathic bronchiectasis study included 258 idiopathic bronchiectasis patients: 70 women with NTM, 64 women without NTM, 67 men with NTM, and 57 men without NTM. Patient evaluation included a detailed physical examination, chest radiograph, medical history, lung function measurements, and collection of serial respiratory samples.

RESULTS: Men with NTM were significantly older at diagnosis of bronchiectasis (median, IQR, 60, 51-68 years) than men without NTM (46, 24-60 years, $p < 0.001$). While women with NTM were older at diagnosis of bronchiectasis (55, 48-64 years) than women without NTM (45, 26-62 years); this difference was not significant. Scoliosis was significantly more prevalent in men with NTM (45%) than in men without NTM (21%, $p = 0.004$), but no difference was seen among women. Pectus deformities were found in a similar small percentage across all strata.

CONCLUSIONS: This study supports past findings on morphotypic traits in bronchiectasis and NTM patients, including older age of diagnosis, and high incidence of scoliosis and suggests potential differences in these associations based on gender and on the presence or absence of NTM.

POSTER #4: Female and Male Differences in the Human Plasma Metabolome at Baseline and in Response to Insufficient Sleep

Abstract submitter: Christopher Depner

Christopher Depner¹; Rachel Markwald¹; Charmion Cruickshank-Quinn²; Kevin Quinn²; Nichole Reisdorph²; Kenneth Wright²

¹University of Colorado Boulder, ²University of Colorado Anschutz Medical Campus

We previously reported sex differences in energy expenditure, food intake, and weight gain during insufficient sleep. Here, we investigated the plasma metabolome using a cross-over laboratory study (8 females/8 males) consisting of 3 baseline days (9h sleep opportunities) followed by 5 days insufficient (5h sleep opportunities) and adequate sleep (9h sleep opportunities) with *ab libitum* food intake. Across all conditions, 112 metabolites showed sex differences and an additional 209 metabolites showed sex differences during insufficient sleep. For females and males, 28 and 13 metabolites, respectively, were uniquely different during insufficient sleep versus both baseline and adequate sleep. Findings highlight the need to include sex as a variable in metabolomics research and for understanding individual differences in responses to insufficient sleep.

POSTER #5: The Gestational Foundation of Sex Differences in Development and Vulnerability

Abstract submitter: Janet DiPietro

Janet DiPietro¹; Kristin Voegtline¹

¹Johns Hopkins Bloomberg School of Public Health

Despite long-standing interest in the role of sex on human development, the functional consequences of fetal sex on early development are not well understood. Here we explore the gestational origins of sex as a moderator of development. In accordance with the focus of this special issue, we examine evidence for a sex differential in vulnerability to prenatal and perinatal risks. Exposures evaluated include those present in the external environment (e.g., lead, pesticides), those introduced by maternal behaviors (e.g., alcohol, opioid use), and those resulting from an adverse intrauterine environment (e.g., preterm birth). We also provide current knowledge on the degree to which sex differences in fetal neurobehavioral development (i.e., cardiac and motor patterns) are present prior to birth. Also considered are contemporaneous and persistent sex of fetus effects on the pregnant woman. Converging evidence confirms that infant and early childhood developmental outcomes of male fetuses exposed to prenatal and perinatal adversities are more highly impaired than those of female fetuses. In certain circumstances, male fetuses are both more frequently exposed to early adversities and more affected by them when exposed than are female fetuses. The mechanisms through which biological sex imparts vulnerability or protection on the developing nervous system are largely unknown. We consider models that implicate variation in maturation, placental functioning, and the neuroendocrine milieu as potential contributors. Many studies use sex as a control variable,

some analyze and report main effects for sex, but those that report interaction terms for sex are scarce. As a result, the true scope of sex differences in vulnerability is unknown.

POSTER #6: Estrous Cycle Differences in Abdominal Sensitivity at Baseline and After Colitis in a Mouse Model of Chronic Visceral Pain

Abstract submitter: Santiago Martinez Gonzalez

Santiago Martinez Gonzalez¹; Yarimar Carrasquillo¹

¹National Institutes of Health

Chronic visceral pain represents a major clinical complaint, with women disproportionately affected. Despite the female prevalence in visceral pain, most preclinical studies in rodents have focused on males due to the potential variability that the estrous cycle stage might add when studying female subjects. As such, systematic evaluation of potential differences between sexes and/or across the estrous cycle in rodent models of visceral pain are warranted. The present study measured abdominal sensitivity using Von Frey filaments in males and in females across the different stages of the estrous cycle at baseline and in the dextran-sodium sulfate (DSS) model of colitis-induced visceral pain. Our results demonstrate that baseline abdominal sensitivity is similar in males and females in estrous and metestrous stages. However, females in the proestrous stage displayed significantly lower abdominal sensitivity than males or females at all other stages, which is consistent with clinical studies. In contrast, females in the diestrous stage displayed significant abdominal hypersensitivity, compared to males or females at all other stages. Following colitis, both male and female subjects exhibited robust referred abdominal hypersensitivity, which was similar in females across the different estrous cycle stages. Altogether, our results demonstrate that the estrous cycle stage is an important biological variable to be considered when studying baseline abdominal sensitivity in females. On the other hand, referred abdominal sensitivity in the DSS model of visceral pain is comparable in females across all stages, suggesting that the estrous cycle stage is not an important biological variable in this model.

POSTER #7: Sex Differences in the Association of Body Mass Index (BMI) with the Anatomical Architecture of Extended Reward Network Regions

Abstract submitter: Arpana Gupta

Arpana Gupta¹; Emeran A. Mayer¹; Kareem Hamadani¹; Mher Alaverdyan¹; Kirsten Tillisch¹; Claudia P. Sanmiguel¹; Jennifer S. Labus¹

¹University of California, Los Angeles

Alterations in key brain regions of the extended reward network have been linked to abnormal ingestive behaviors in obesity. Network analysis using graph theory allows the characterization of disease-specific alterations in the architecture of large-scale brain networks. Several measures are used to characterize the connectedness of a particular region to another region, including degree and local efficiency. These measures are associated with greater efficiency in transferring information

between regions.

AIMS: We hypothesized that body mass index (BMI) is associated with differences in degree and local efficiency of key regions comprising the extended reward circuit.

METHODS: White matter was measured in 120 healthy subjects. Regional parcellation was conducted using Freesurfer, and resulted in 74 bilateral cortical and 7 subcortical structures, including the cerebellum. White matter connectivity was estimated using DTI fiber tractography and Runge-Kutta algorithm. Controlling for the main effects of age, the general linear model was applied on connections that measured projections from regions associated with the extended reward network. Customized contrasts were used to assess for disease and sex differences. The resulting p-values were corrected for multiple comparisons. Significance was set at $q < 0.05$.

RESULTS: There were 57 lean (34 females), 47 overweight (18 females), and 16 obese (8 females) individuals. *Degree:* BMI was positively associated with degree of left thalamus ($\beta = 1.14$), left caudate ($\beta = .67$), and right nucleus accumbens ($\beta = .83$), but negatively associated with right ventromedial prefrontal cortex ($\beta = -.62$). There was a significant interaction effect for right dorsolateral prefrontal cortex ($\beta = -1.39$) with males and not females; and a significant interaction for right nucleus accumbens ($\beta = -.66$). *Local efficiency:* BMI was positively associated with local efficiency for right amygdala ($\beta = .009$) and left nucleus accumbens ($\beta = .008$), but negatively associated with right anterior insula ($\beta = -.006$), and right ventromedial prefrontal cortex ($\beta = -.007$), with females but not males. There was a significant interaction for left hippocampus ($\beta = -.005$) with males but not females.

DISCUSSION: The anatomical network architecture of regions within the reward network is associated with BMI in both male and female healthy subjects. Higher BMI and being female was associated with more local and regional communication between regions involved in dopamine signaling, and less information propagation was observed in the cognitive frontal regions. In males, the opposite pattern was observed. These findings are consistent with the reward deficiency syndrome hypothesis that suggests lower dopamine levels make them less sensitive to reward stimuli and more prone to food intake in order to compensate for this disorder.

POSTER #8: Sex Differences in Alzheimer's disease: Using Optogenetics and Neurochemical Assays to Understand and Rescue Cognitive Decline

Abstract submitter: Holly Hunsberger

Holly Hunsberger¹; Jennifer Perusini¹; Christine Denny¹

¹Columbia University

Recent evidence from our lab and others suggest that memory retrieval, rather than memory encoding, is impaired in Alzheimer's mice. Our lab previously created the ArcCreER^{T2} mice to permanently tag neurons following learning. We bred this line with the APP/PS1 (an AD model) mouse line and have reported deficits in long-term memory and social memory, which correlated with impaired memory traces in the hippocampus. To rescue this cognitive decline, we used optogenetic stimulation in the hippocampus and were able to recover lost memories in male AD mice. Interestingly, our preliminary data indicate that AD female mice develop memory deficits at an earlier age compared to AD males and that this impairment is comorbid with anxiety-like behavior.

Understanding these different mechanisms can lead to more personalized treatment options for males and females.

POSTER #9: Gender Stratification of the Experience of Violence, Depression, and Immune Function in African American Young Adults

Abstract submitter: Latifa Jackson

Latifa Jackson¹; Max Shestov¹; Forough Saadatmand¹

¹Howard University

Violence is more prevalent in African American communities than in other American communities. This has impacts not only on criminal justice interventions, but also on the physical and mental health of these communities, including their risk for acquiring life-threatening diseases. While many studies have focused on the effects of violence on African American males, we wanted to understand the relative gender effects of violence. Introducing gender biases associated with exposure to violence, depression, immune function, and risks to HIV/AIDS is an important step in understanding how young women perceive and internalize societal violence directed toward them. We study a cohort of 557 young African American adults aged 18-25 years old (females N=274, males N=283) from the Washington, DC, area with varying experiences of violence exposure. We used sociological, epidemiological, mental health, bioinformatics, and quantitative genetics approaches to build a predictive portrait of the effects of violence on African American health. We find that women are twice as likely to be a victim of sexual, verbal, and gender-based violence as their male counterparts. This is not the case for community-related violence such as that perpetrated by gangs. A cortisol stress response marker is modestly correlated to increased perceived victimization, with women having higher overall concentrations of cortisol. The results suggest that violence may be a contributing factor in negative health outcomes and therefore should be the target of both criminal justice and public health interventions to decrease societal violence and to increase immunological health and mental states.

POSTER #10: Sex Differences in Early Risk Factors for Cocaine Seeking in Adolescence and Adulthood

Abstract submitter: Chloe J. Jordan¹

¹National Institutes of Health

Drug abuse before age 14 doubles the likelihood of substance use disorder, yet few studies have focused on identifying risk factors for drug use in young subjects. We screened juvenile male and female rats for (1) sucrose preferences, (2) novelty responses, and (3) working memory, followed by cocaine self-administration starting in early or late adolescence. Stepwise regression revealed sucrose preferences were associated with cocaine seeking in males ($R^2=0.54$, $p<0.01$), but not females. In contrast, novelty-induced activity was associated with cocaine seeking in females ($R^2=0.48$, $p<0.02$), but not males. Novelty responses also predicted motivation to earn cocaine ($R^2=0.58$) and relapse in females ($R^2=0.73$, $p<0.05$). Lastly, poor working memory predicted relapse in

both males ($R^2= 0.44$) and females ($R^2=0.63$, $p<0.05$), suggesting working memory may be a risk factor for both sexes.

POSTER #11: Assessing the Impact of the Knockout Mouse Phenotyping Program (KOMP²) on the Biomedical Research Enterprise

Abstract submitter: Sheethal Jose

Sheethal Jose¹; Ya-Ling Li¹; Christine Change¹; Rebecca Lenzi¹; Oleg Mirochnitchenko¹; Colin Fletcher¹

¹National Institutes of Health

KOMP² is a Common Fund initiative that aims to generate a resource of mice containing a null (“knockout”) mutation in every gene in the mouse genome and to collect phenotype information for 3,000 mouse strains. This resource should enhance the ability of researchers to translate basic genetic research to applications relevant to human health. To study the impact of the KOMP² program on the scientific community, we identified more than 1,300 publications that used materials or data from KOMP. A bibliometric analysis of these publications was performed to evaluate the productivity and citation impact. Ultimately, we hope to identify research grants and publications that report findings on previously unexamined genes and measure the impact of KOMP².

POSTER #12: Ultrasonographic Median Nerve/Carpal Tunnel Ratio and Female/Male Differences in Carpal Tunnel Syndrome

Abstract submitter: Nanette C. Joyce

Scott Homer¹; Colleen Anthonisen²; Eduard Poltavskiy²; Heejung Bang²; Michael S. Cronan²; Jay J. Han MD³; Nanette C. Joyce²

¹University of Michigan Health System, ²University of California, Davis, ³University of California, Irvine

INTRO: Median nerve cross-sectional area (CSA) is commonly measured by ultrasound (US) in carpal tunnel syndrome (CTS). We evaluated whether median nerve/carpal tunnel CSA ratio could be the diagnostic parameter for CTS.

METHODS: Unilateral wrists from 130 electrodiagnostically confirmed CTS cases and 109 sex-matched controls underwent US measurement.

RESULTS: Nerve/tunnel CSA ratio, carpal tunnel and median nerve CSA had significant mean differences between cases and controls ($p<0.001$). Sex differences occurred for nerve/tunnel ratio, carpal tunnel CSA, and flexor tendon group CSA ($p<0.001$) but not for mean median nerve CSA (cases and controls).

DISCUSSION: Median nerve CSA was the best predictor for CTS without apparent sex difference. While nerve/tunnel CSA ratio is a suboptimal diagnostic parameter, it provides insight into the pathophysiology of CTS.

POSTER #13: Sex Differences in Response to a Targeted Kyphosis-Specific Exercise Program

Abstract submitter: Wendy B. Katzman

Wendy B. Katzman¹; Neeta Parimi²; Anne Schafer^{1,3}; Roger K. Long¹; Shirley Wong¹; Amy Gladin⁴; Nancy E. Lane⁵

¹University of California, San Francisco, ²San Francisco Coordinating Center, ³San Francisco Veterans Affairs Medical Center, ⁴Kaiser Permanente Northern California, ⁵University of California, Davis

Hyperkyphosis, an excessive anterior curvature in the thoracic spine, is associated with reduced health status in older adults. Hyperkyphosis is highly prevalent and more common in older women than men. There is no standard intervention to reduce age-related hyperkyphosis. Sex differences in response to a targeted intervention are not known.

We conducted a waitlist design randomized controlled trial to determine whether a targeted kyphosis specific exercise program improved Cobb angle of kyphosis, and whether the magnitude of change differed between men and women.

One hundred and twelve (112) participants aged ≥ 60 years with kyphosis ≥ 40 degrees were randomized to exercise or waitlist control. Group intervention was delivered by a physical therapist for 1 hour twice a week for 3 months. Controls received the intervention after 3 months. Primary outcome was change in Cobb angle measured from standing lateral spine radiographs. Secondary outcomes included change in kyphometer-measured kyphosis, physical function, and quality of life. Groups were combined, and ANOVA was used to test sex by time interaction to evaluate treatment effects in men and women.

Participants (67 women, 45 men) were 70.0 ± 6.2 years with baseline Cobb 55.6 ± 12.1 degrees. There were no between group differences at baseline; however, men had higher kyphometer-measured kyphosis. There was no significant between group difference in change in Cobb after intervention ($p=0.09$), but kyphometer-measured kyphosis differed by 4.8 degrees ($p<0.001$). There was no significant interaction between sex and change in Cobb after intervention ($p=0.67$).

A 3-month targeted exercise program reduced kyphometer but not radiographic-measured kyphosis. Despite sex differences in baseline kyphosis, sex did not affect treatment response.

***POSTER #14: Estrogen Contributes to Sex Differences in M2-Polarization During Asthma**

Abstract submitter: Aleksander Keselman

Aleksander Keselman¹; Jessie X. Fang¹; Preston White¹; Nicola Heller¹

¹Johns Hopkins University

Asthma exhibits sex differences, affecting mostly boys in childhood and women in adulthood. Alveolar macrophages have emerged as major mediators of allergic lung inflammation. We hypothesized that estrogen enhances the M2 polarization of alveolar macrophages to promote asthma. We found M2-gene expression to be elevated in alveolar and bone-marrow derived macrophages (BMMs) from female mice after challenge with allergen and stimulation with IL-4, respectively. Pretreatment of female BMMs with estrogen receptor ligands enhanced IL-4-induced M2-gene expression. Ovariectomized and LysMCRE E_Rα flox/flox mice exhibited impaired M2 responses after challenge with OVA. Together these data suggest that sex and hormonal factors contribute to sex differences in macrophage responses during asthma.

**This poster presentation has also been selected for oral presentation.*

POSTER #15: Sex Commonalities and Differences in Body Mass Index (BMI)-Related Alterations in Intrinsic Brain Activity and Connectivity

Abstract submitter: Lisa A. Kilpatrick

Lisa A. Kilpatrick¹; Emeran A. Mayer¹; Jennifer S. Labus¹; Kirsten Tillisch¹; Bruce Naliboff¹; Claudia P. Sanmiguel¹; Angeles Arpana Gupta¹

¹University of California, Los Angeles

We evaluated sex-differences in BMI-related intrinsic activity/connectivity of the brain's reward networks using partial least squares analyses. In both men (n=43) and women (n=43), increased BMI was associated with increased slow-5 activity in left globus pallidus (GP) and substantia nigra. In women only, increased BMI was associated with increased slow-4 activity in right GP and bilateral putamen, and reduced slow-5 connectivity between left GP/putamen and regions involved in emotion and cortical regulation. This sex difference in BMI-related connectivity may be related to observed sex differences in emotional eating and suggests the importance of personalized treatments for obesity that consider the sex of the affected individual.

POSTER #16: Sex Dimorphic Regulation of Osteoprogenitor Progesterone in Bone Stromal Cells

Abstract submitter: Nancy Lane

Alexander Kot¹; Nancy Lane¹; Wei Yao¹; Zhengdong Zhong¹; Yu-An Evan Lay¹

¹University of California, Davis

Increasing peak bone mass is a promising strategy to prevent osteoporosis. A mouse model of progesterone receptor (cPRKO) ablation selectively in mesenchymal stromal cells (MSCs) increased bone mass through a sex-dependent mechanism. Here we employed RNA sequencing analysis to evaluate sex-dependent differences in gene transcription of MSCs of wild type (WT) and cPRKO mice. cPRKO MSCs had marked sex hormone-dependent changes in gene transcription in male mice as compared to WT controls. These transcriptional differences revealed dysregulation in pathways involving immunomodulation, osteoclasts, bone anabolism, extracellular matrix interaction, and matrix interaction. These results identified many potential mechanisms that may explain our observed high bone mass phenotype with sex differences when PR was selectively deleted in the MSCs.

POSTER #17: MicroRNA-19b Acts as a Sex-Dependent Regulatory Hub for Posttraumatic Stress and Chronic Widespread Pain Development Following Trauma Exposure

Abstract submitter: Sarah Linnstaedt

Sarah Linnstaedt¹; Cathleen Rueckeis¹; Kyle Riker¹; Shan Yu¹; Samuel McLean¹

¹University of North Carolina at Chapel Hill

Posttraumatic stress (PTS) and chronic widespread pain (CWP) are frequent co-morbid sequelae of trauma that occur at different rates in men and women. Sex-dependent molecular mediators of PTS and CWP are poorly understood. We examined whether the stress and pain associated microRNA, miR-19b, might contribute to sex-dependent differences in vulnerability to these outcomes. We found a sex-dependent relationship between initial miR-19b expression levels and both PTS development (OR=1.41, p=0.039) and CWP development (OR=1.46, p=0.031) 6 months following motor vehicle collision trauma (n=178). Sex-dependent expression of miR-19b was also observed in two animal models. The potential importance of miR-19b to PTS/CWP pathogenesis is supported by further analyses indicating that miR-19b sex-dependently regulates a number of PTS and CWP associated transcripts.

POSTER #18: Women Experience Greater Chronic Pain Severity Following Major Thermal Burn Injury

Abstract submitter: Matthew Mauck

Matthew Mauck¹; Christopher Sefton¹; Samuel McLean¹

¹University of North Carolina

Women experience greater chronic pain severity compared to men across a number of chronic pain conditions including widespread pain, chronic abdominal pain, and persistent post-surgical pain. However, no studies have examined sex differences in chronic pain severity following Major Thermal Burn Injury (MThBI), a trauma in which female survivors are known to have worse outcomes. Therefore, we hypothesized that women would experience greater chronic pain severity than men in the aftermath of MThBI. We report the results of an observation cohort study (n=96) that prospectively evaluated chronic pain severity over 1 year following MThBI. These results indicate that women experience a greater burden of chronic pain following MThBI compared to men.

POSTER #19: Sex Differences in Placental Epigenetic Aging

Abstract submitter: Catherine Monk

Catherine Monk^{1,2}; Fabien Delahaye³; Frances Champagne⁴; Seonjoo Lee, PhD¹; Tianshu Feng, MA¹; Ronald Wapner¹

¹Columbia University Medical Center, ²New York State Psychiatric Institute, ³Albert Einstein College of Medicine, ⁴University of Texas at Austin

The study of prenatal exposures affecting long-term health trajectories has long identified sex differences in illness risk (Braithwaite et al., 2017). Sex differences in the placenta, originating in sex chromosomes, in part undergirds this sex-specific variation (Bale, 2016). Placental DNA methylation (DNAm) is used to calculate epigenetic age, the predicted age by DNAm in relation to the chronological gestational age (GA) at birth; older placental epigenetic age is found in preeclamptic pregnancies (Mayne et al., 2017). Using Mayne's (2017) DNAm approach in two studies of healthy pregnant women (n=192 and n=107) with chronological GA 36-42 and 33-41 weeks we found females versus males had stronger associations between placenta DNAm and chronological GA (.21 versus .11; .39 versus .01). In one sample, maternal education and 1st and 2nd trimester cortisol diurnal slope were inversely associated with placenta DNAm in the males ($r=-.27$ and $r=-.44$, $r=-.33$, all $ps<.05$) but not females ($r=-.13$ and $r=-.24$, $r=.06$, all $ps>.10$). Consistent with male vulnerability to preterm birth and early-appearing neurodevelopmental disorders, these data indicate that for women carrying males, lifestyle factors may be associated with placental immaturity relative to birth age, with possible implications for health outcomes.

POSTER #20: Reporting of Sex and Gender in Clinical Trial Protocols and Published Results

Abstract submitter: Thiyagu Rajakannan

Thiyagu Rajakannan¹; Kevin M. Fain¹; Rebecca J. Williams¹; Tony Tse¹; Deborah A. Zarin¹

¹National Institutes of Health

Background: Biomedical research funders and journals have increasingly focused on the importance of assessing and reporting the effect of sex (biological factors) and gender (sociocultural factors) on health outcomes in clinical studies.

Objective: To assess publicly available clinical study protocols and corresponding published studies to analyze how “sex” and “gender” information was incorporated in study design and reported.

Study Design: We identified a convenience sample of 80 articles from *NEJM* and *JAMA* published in 2014/2015 for which full protocols were available online. We searched for and assessed use of the terms “sex” and “gender” in the protocol and corresponding publication.

Results: First, the terms “sex” and “gender” were not defined in any protocol or publication. Second, 40% of trials (32/80) used both terms interchangeably within the protocol; 28 of these used “sex” only and 4 used neither term in the corresponding publication. Third, the term “gender” only was used in 29% (23/80) of protocols, but only one publication used this term.

Our data indicate imprecision in how “sex” and “gender” terms were used in study protocols, suggesting a lack of appreciation among researchers of these distinct concepts. Publications generally used only “sex,” implying that journals enforce specific and consistent terminology when reported. We note the generalizability of these findings may be limited. Also, we did not assess how the constructs were used in the research.

Conclusions: Our study supports the need for continuing efforts to standardize the concepts of “sex” and “gender” and ensure their appropriate use in biomedical research.

POSTER #21: Sex Chromosome Dosage Effects on Gene Expression in Humans

Abstract submitter: Armin Raznahan

Armin Raznahan¹; Neelroop Parikshak²; Judith Ross³; Jay Giedd⁴; Daniel Geschwind²

¹National Institutes of Health, ²University of California, Los Angeles, ³Thomas Jefferson University; ⁴University of California, San Diego

A fundamental question in the biology of sex-differences has eluded direct study in humans: how does sex chromosome dosage (SCD) shape genome function? To address this, we developed a systematic map of SCD effects on gene function by analyzing genome-wide expression data in humans with diverse sex chromosome aneuploidies (XO, XXX, XXY, XYY, XXYY). For sex chromosomes, we demonstrate a pattern of obligate dosage sensitivity among evolutionarily preserved X-Y homologs, and revise prevailing theoretical models for SCD compensation by

detecting X-linked genes whose expression increases with decreasing X- and/or Y-chromosome dosage. We further show that SCD-sensitive sex chromosome genes regulate specific co-expression networks of SCD-sensitive autosomal genes with critical cellular functions and a demonstrable potential to mediate previously documented SCD effects on disease. Our findings detail wide-ranging effects of SCD on genome function with implications for human phenotypic variation.

POSTER #22: Dopamine Transporter (DAT1) Gene Variation and Intravenous (IV) Alcohol Self-Administration in Non-Dependent Drinkers: Moderation by Sex

Abstract submitter: Bethany L. Stangl

Bethany L. Stangl¹; Courtney L. Vaughan¹; Hui Sun¹; Melanie L. Schwandt¹; Falk Lohoff¹; Vijay A. Ramchandani¹

¹National Institutes of Health

Genetic variation in the dopamine transporter (DAT1) has been associated with alcohol dependence. This study aimed to characterize the effect of DAT1 variation and the role of sex differences on self-administration of IV alcohol in 70 non-dependent drinkers.

Results revealed that male 10A homozygotes had greater alcohol self-administration compared to 10A females, while 9A males had lower self-administration than 9A females. Similarly, 10A males felt and wanted more alcohol compared to 10A females, while 9A females had higher subjective responses compared to 9A males.

These findings underscore the critical role of dopamine neurotransmission and the moderating role of sex in the motivation and consumption of alcohol in non-dependent drinkers.

POSTER #23: Sex-Stratified Analysis of Obsessive-Compulsive Disorder Reveals Minor Differences in Genetic Architecture

Abstract submitter: Barbara Stranger

Ekaterina A. Khramtsova^{1,6}; Raphael Heldman^{2,6}; Eske M. Derks^{3,6}; Dongmei Yu^{4,6}; Lea K. Davis^{5,6}

¹University of Chicago, ²University of California, Berkeley, ³Queensland Institute of Medical Research, ⁴Harvard Medical School, ⁵Vanderbilt University Medical Center, ⁶University of Chicago

Obsessive-compulsive disorder (OCD) exhibits sexual dimorphism in age of onset and clinical presentation. Here, we present the first genome-wide assessment of the sex-specific genetic architecture of OCD. Overall we find evidence for minor differences in the genetic architecture of OCD between the sexes. Specifically, we report that the genetic correlation is high between males and females, and heritability estimates do not differ between the sexes. Despite this, we observed differences in enrichment of functional annotations between variants with very different effects across sexes. This suggests that although we are underpowered to detect these minor differences at the individual variant or gene level, we observe evidence of sexually dimorphic biology underlying risk for OCD. These results hold promise for discoveries in future studies with larger sample sizes.

POSTER #24: Epidemiological and Experimental Evidence for Sex-Dependent Differences in the Outcome of Leishmania Infantum Infection

Abstract submitter: Elizabeth A. Turcotte

Elizabeth A. Turcotte¹; Nilda E. Rodriguez¹; Iraci D. Lima²; Upasna Gaur Dixit³; Ryan D. Lockard¹; Hemali Batra-Sharma³; Eliana L. Nascimento²; Selma M. B. Jeronimo^{2,4}; Mary E. Wilson^{3,5}

¹University of Northern Iowa, ²Federal University of Rio Grande do Norte, ³University of Iowa, ⁴National Institute of Science and Technology of Tropical Diseases, ⁵Veterans' Affairs Medical Center

Leishmania infantum causes visceral leishmaniasis (VL) in Brazil. We previously observed that VL is more common in males than females living in endemic neighborhoods, despite similar exposure. Using a larger sample, we document that VL is more common in males than females, but only after puberty. BALB/c and C57BL/6 mouse models confirmed that there is a biological basis for male susceptibility to symptomatic VL, showing higher parasite burdens in males than females. Female C57BL/6 mice generated more antigen-induced cytokines associated with curative responses (IFN- γ , IL-1 β). Males expressed higher levels of IL-10 and TNF, which are linked to exacerbated disease. Different parasite lines entered or survived at a higher rate in macrophages of male than female origin. These results suggest that males are inherently more susceptible to *L. infantum* than females, and that mice are a valid model to study this sex-dependent difference.

POSTER #25: Race and Sex Differences in Maternal Mediated Childhood Obesity (MMCO)

Abstract submitter: ClarLynda Williams-DeVane

ClarLynda Williams-DeVane¹; Michele Josey¹; Cathrine Hoyo²

¹North Carolina Central University, ²North Carolina State University

BACKGROUND: Childhood obesity is an epidemic in the United States, creating a medical burden of more than \$14 billion a year as of 2010. It is an epidemic that disproportionality effects minorities and those of lower socioeconomic status (SES). While diet and exercise interventions have had limited success, the etiology and complexity of childhood obesity and its disparities are not well understood. To address childhood obesity the Newborn Epigenetic Study (NESt) was initiated to prospectively test the influence of *in utero* environmental exposures on DNA methylation profiles in newborns. However, analyzing DNA methylation data requires the correct classification of obesity status in the child and the associated risk based on maternal body mass index (BMI).

OBJECTIVE: We hypothesize that the relationship between childhood obesity status and maternal BMI classification is both sex and race specific.

METHODS: Here, we calculate race- and sex-specific odds ratios of risk for children in NeSt becoming overweight or obesity by age 4. Further, we identify ideal cutoffs for maternal BMI utilizing Receiver Operating Characteristic (ROC) curves.

RESULTS: The odds ratios of obesity risk between Caucasian and African American as well as male and female children are significantly different. Further, the optimal maternal BMI cutoff for the prediction of children becoming overweight or obese by age 4 is significantly different in Caucasian and African American moms.

CONCLUSIONS: Ultimately, this work will lead to better understanding of the maternal influences of childhood obesity and more efficient analysis of DNA methylation data.

POSTER #26: Trajectories of Knee Bone Shape Change Are Associated with Sex and Osteoarthritis

Abstract submitter: Barton L. Wise

Barton L. Wise¹; Jingbo Niu²; Yuqing Zhang³; Felix Liu⁴; Joyce Pang⁵; John A. Lynch⁴; Nancy E. Lane¹

¹University of California, Davis, ²Baylor College of Medicine, ³Harvard University School of Medicine, ⁴University of California, San Francisco, ⁵University of New Mexico School of Medicine

Knee osteoarthritis (OA) is more common in women than men; however, the biological mechanisms for sex difference in knee OA are not well understood. The purpose of the present study was to describe knee bone shape changes over time and to examine whether sex or knee OA are associated with change of bone shape. We outlined distal femur and proximal tibia shape on radiographs of 473 knees chosen without regard to OA status in the Osteoarthritis Initiative at three time points and used group-based trajectory modeling to identify distinctive patterns of bone shape

change for each shape mode. The shapes of the distal femur and proximal tibia changed little over time but did divide into separate trajectory groups largely due to baseline differences that remained consistent across the years. The trajectory subgroups were associated with both sex and knee OA.

POSTER #27: Sex-Dependent, Osteoblast Stage-Specific Effects of Progesterone Receptor on Bone Acquisition

Abstract submitter: Wei Yao

Zhendong A. Zhong¹; Alexander Kot²; Yu-An E. Lay²; Hongliang Zhang²; Junjing Jia²; Nancy E. Lane²; Wei Yao²

¹Van Andel Research Institute, ² University of California, Davis

To define progesterone receptor (PR) in the regulation of sexual dimorphism in bone, we selectively deleted PR at different stages of osteoblast differentiation. We found that mice with PR conditional deletion in early osteoprogenitor cells developed greater trabecular bone volume, greater bone formation, increased number of mesenchymal stem cells, and greater osteogenic potential, particularly in males. PR deficiency during the period of rapid bone growth induced rapid trabecular bone loss in both the wild type and the PRcKO mice in both sexes. No differences in trabecular bone mass was observed when PR was deleted in mature osteoblasts. In conclusion, PR inactivation in early osteoprogenitor cells but not in mature osteoblasts influenced trabecular bone accrual in a sex-dependent manner. PR deletion in osteoblast lineage cells did not affect cortical bone mass.

SEX AS A BIOLOGICAL VARIABLE

October 26-27, 2017

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PARTICIPANTS LIST



PARTICIPANTS LIST

GENERAL ATTENDEES

Sean Agbor

National Institutes of Health
Building 10, 7S216
Bethesda, MD 20892
Phone: (301) 828-7338
Email: sean.agbor-enoh@nih.gov

Mark R. Alexander

Public Health Advisor
National Cancer Institute, NIH
9609 Medical Center Drive
Rockville, MD 20892
Phone: (240) 276-6869
Email: alexandm@mail.nih.gov

James (Jim) M. Anderson, MD, PhD

Director, Division of Program Coordination
Planning and Strategic Initiatives
Office of the Director, NIH
1 Center Drive, Room 260
Bethesda, MD 20892
Phone: (301) 402-9852
Email: james.anderson2@nih.gov

Donald R. Anderson, MD

Chief Executive Officer
Cardiology Associates
3558 Jefferson Street N, PMB 104, Suite 5
Lewisburg, WV 24901
Phone: (202) 294-7948
Email: donaldranderson2@aol.com

Kathryn Jean Argue, PhD

Postdoctoral Fellow
University of Maryland School of Medicine
655 W Baltimore Street, BRB 5-014
Baltimore, MD 21201
Phone: (410) 706-2654
Email: kargue@som.umaryland.edu

Matthew Arnegard, MSc, PhD

Science Writer
National Institutes of Health
6707 Democracy Boulevard
Bethesda, MD 20817
Phone: (301) 451-6878
Email: matthew.arnegard@nih.gov

Whitney Barfield

National Heart, Lung, and Blood Institute, NIH
6705 Rockledge Drive
Bethesda, MD 20816
Phone: (404) 273-6730
Email: whitney.barfield@nih.gov

Mireya Becerra-Díaz

Postdoctoral Fellow
Johns Hopkins School of Medicine
720 Rutland Avenue
Baltimore, MD 21205
Phone: (410) 955-1747
Email: mbecerr1@jhmi.edu

Aruna Behera

Scientific Review Officer
Center for Scientific Review, NIH
6701 Rockledge Drive
Bethesda, MD 20892
Phone: (301) 435-6809
Email: aruna.behera@nih.gov

Babak Behnam, MD, PhD

National Human Genome Research
Institute, NIH
5625 Fishers Lane, MSC 0901, Room 4N-15
Rockville, MD 20892
Phone: (407) 920-4420
Email: babak.behnam@nih.gov

Inna Belfer, MD, PhD

National Institutes of Health
6707 Democracy Boulevard
Bethesda, MD 20892
Phone: (301) 435-1573
Email: inna.belfer@nih.gov

Margaret F. Bevans, PhD

Associate Director, Clinical Research
National Institutes of Health
6707 Democracy Boulevard, Suite 404
Bethesda, MD 20817
Phone: (301) 496-3934
Email: mbevans@nih.gov

Kim Boggess

University of North Carolina at Chapel Hill
3010 Old Clinic Building
Chapel Hill, NC 27514
Phone: (919) 966-1601
Email: kboggess@med.unc.edu

David M. Bollweg

Program Specialist
Office of Strategic Coordination, OD, NIH
6001 Executive Boulevard
Rockville, MD 20852
Phone: (301) 451-3046
Email: david.bollweg@nih.gov

Marty Bond, MA

Chief of Public Affairs
National Institutes of Health
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20892
Phone: (301) 496-9006
Email: martha.bond@nih.gov

Ghada Bourjeily

Associate Professor of Medicine
Brown University
146 West River, Suite 11C
Providence, RI 02904
Phone: (401) 444-8664
Email: ghada_bourjeily@brown.edu

Beth Bowers

National Institute of Mental Health, NIH
6001 Executive Boulevard
Bethesda, MD 20892
Phone: (301) 443-3776
Email: bb222c@nih.gov

Jim Boyce

National Institute of Allergy and Infectious
Diseases, NIH
5601 Fishers Lane
Rockville, MD 20878
Phone: (555) 555-5555
Email: jim.boyce@nih.gov

Rachel Britt

Office of Strategic Coordination, OD, NIH
6001 Executive Boulevard, Room 8180T
Rockville, MD 20852
Phone: (301) 435-0968
Email: rachel.britt@nih.gov

Liliana Brown

National Institute of Allergy and Infectious
Diseases, NIH
5601 Fishers Lane
Rockville, MD 20892
Phone: (301) 496-1884
Email: liliana.brown@nih.gov

Paul L. Brown, PhD

Post-doctoral Fellow
University of Maryland School of Medicine
655 West Baltimore Street
Baltimore, MD 21201
Phone: (410) 402-6069
Email: lbrown@mprc.umaryland.edu

Gretchen Buckler, MD, MPH

Medical Officer
Office of Research on Women's Health, OD, NIH
6707 Democracy Boulevard
Bethesda, MD 20817
Phone: (301) 451-2730
Email: bucklerag@nih.gov

Bettina Buhring

National Institute of Mental Health, NIH
6001 Executive Boulevard
Rockville, MD 20852
Phone: (301) 443-1576
Email: bettina.buhring@nih.gov

Catherine Burnette, PhD, LMSW

Assistant Professor
Tulane University
127 Elk Place
New Orleans, LA 70112
Phone: (504) 862-3495
Email: cburnet3@tulane.edu

Karim Calis

National Institute of Child Health and Human
Development, NIH
17933 Ednor View Terrace
Ashton, MD 20861
Phone: (301) 928-2413
Email: kcalis@nih.gov

Rebecca Campo

National Heart, Lung, and Blood Institute, NIH
6701 Rockledge Drive
Bethesda, MD 20817
Phone: (301) 594-1047
Email: rebecca.campo@nih.gov

Victoria A. Cargill, MD, MSCE

Associate Director, Interdisciplinary Research
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20817
Phone: (301) 435-0971
Email: vc52x@nih.gov

Diana Chan

National Institute of Arthritis and Musculoskeletal
and Skin Diseases, NIH
10 Center Drive
Building 10, Room 12S251
Bethesda, MD 20892
Phone: (206) 753-9241
Email: dianchan@uw.edu

Preethi Chander

National Institute of Dental and Craniofacial
Research, NIH
6701 Democracy Boulevard
Bethesda, MD 20892
Phone: (301) 827-4620
Email: preethi.chander@nih.gov

Anshumali Chaudhari, PhD

Scientific Review Officer
Center for Scientific Review, NIH
6701 Rockledge Drive
Bethesda, MD 20892
Phone: (301) 435-1210
Email: chaudhaa@csr.nih.gov

Janine A. Clayton

Director
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard
Bethesda, MD 20817
Phone: (301) 402-1770
Email: janine.clayton@nih.gov

Richard Conroy

National Institutes of Health
6001 Executive Boulevard, Room 8180E
Bethesda, MD 20892
Phone: (301) 435-9008
Email: conroyri@mail.nih.gov

Stephanie Courchesne

Lead Health Science Policy Analyst
Office of Strategic Coordination, OD, NIH
6001 Executive Boulevard, Room 8190L
Rockville, MD 20852
Phone: (301) 451-3687
Email: stephanie.courchesne@nih.gov

Shelby Daniel-Wayman

National Institute of Allergy and Infectious
Diseases, NIH
Qrtrs 15B-1
8 West Drive, MSC 2665
Bethesda, MD 20892-2665
Phone: (301) 761-7785
Email: shelby.daniel-wayman@nih.gov

Simhan Danthi

Program Officer
National Heart, Lung, and Blood Institute, NIH
6701 Rockledge Drive
Bethesda, MD 20892
Phone: (301) 435-0513
Email: ndanthi@mail.nih.gov

Rebecca DelCarmen Wiggins, PhD

Program Officer
Office of Research on Women's Health, OD,
NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20892
Phone: (301) 273-8373
Email: rdelcarm@nih.gov

Roberto Delgado

Brigham and Women's Hospital, Harvard
Medical School
6001 Executive Boulevard, Suite 7210
Bethesda, MD 20892
Phone: (301) 443-4372
Email: roberto.delgado@nih.gov

Christopher M. Depner, PhD

Postdoctoral Fellow
University of Colorado Boulder
1725 Pleasant St. Clare Small, Room 114
Boulder, CO 80309
Phone: (303) 735-1923
Email: christopher.depner@colorado.edu

Leslie Derr

Office of Strategic Coordination, OD, NIH
6001 Executive Boulevard
Rockville, MD 20892
Phone: (301) 594-8174
Email: derrl@mail.nih.gov

Christine DesLauriers
Nurse Consultant
National Institute of Allergy and Infectious
Diseases, NIH
5601 Fishers Lane
Rockville, MD 20852
Phone: (301) 370-6334
Email: christine.deslauriers@nih.gov

Soumyadeep Dey
Research Fellow
National Institute of Diabetes and Digestive and
Kidney Diseases, NIH
10 Center Drive
Building 10, Room 9N321
Bethesda, MD 20874
Phone: (615) 426-4281
Email: shoumo.dey@nih.gov

Janet A. DiPietro
Professor; Vice Dean for Research and Faculty
Johns Hopkins Bloomberg School
of Public Health
615 N. Wolfe Street, W1033
Baltimore, MD 21205
Phone: (410) 750-9290
Email: jdipiet1@jhu.edu

Julie M. Eiserman, MA
Policy Analyst
Office of Human Subjects Research
Protections, NIH
10 Center Drive, Room 2C146
Bethesda, MD 20892-1154
Phone: (301) 402-8665
Email: julie.eiserman@nih.gov

Mariam Eljanne, PhD
Health Scientist Administrator
National Cancer Institute, NIH
9609 Medical Center Drive
Rockville, MD 20850
Phone: (240) 276-7607
Email: eljannem@mail.nih.gov

Sharon Elliot, PhD
Professor, Department of Surgery
University of Miami, Miller School of Medicine
600 NE 36th Street, PH20
Miami, FL 33137
Phone: (305) 807-3239
Email: selliot@med.miami.edu

Catherine Evans, PhD
Science Writer
Office of Communications and Health Education
National Institute of Dental and Craniofacial
Research, NIH
Building 31, Room 5B49
Bethesda, MD 20892
Phone: (301) 594-7557
Email: catherine.evans@nih.gov

Lisa Evans
Scientific Workforce Diversity Officer
National Institutes of Health
6705 Rockledge Drive, RKL 1 Room 5168
Bethesda, MD 20904
Phone: (301) 435-2689
Email: evansl@od.nih.gov

Andrew W. Frank-Wilson, PhD
Visiting Fellow
National Institute on Aging, NIH
7201 Wisconsin Avenue
Bethesda, MD 20814
Phone: (301) 496-6232
Email: andrew.frank-wilson@nih.gov

Carlos Eduardo Frigerio Domingues
Post-doctoral Scientist
National Institute on Deafness and Other
Communication Disorders, NIH
35A Convent Drive, Building 35A
Bethesda, MD 20892
Phone: (301) 594-1043
Email: carloseduardo.frigeriodomingues@nih.gov

Crina Frincu
National Institute of Dental and Craniofacial
Research, NIH
6701 Democracy Boulevard
Bethesda, MD 20892
Phone: (301) 594-0652
Email: crina.frincu@nih.gov

Nimesha Gerlus
Post-baccalaureate IRTA
National Institute of Mental Health, NIH
10 Center Drive
Bethesda, MD 20814
Phone: (301) 402-9353
Email: nimesha.gerlus@nih.gov

Anna Ghambaryan
Scientific Review Officer
National Institutes of Health
Rockville, MD 20892
Phone: (301) 443-4032
Email: anna.ghambaryan@nih.hhs.gov

Melissa Ghim, PhD

Health Scientist Administrator
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20892-5484
Phone: (301) 496-7853
Email: melissa.ghim@nih.gov

Ellen Gold, PhD

Professor
University of California, Davis
One Shields Avenue, Medical Science 1C
Davis, CA 94708
Phone: (530) 752-2446
Email: ebgold@ucdavis.edu

Elena K. Gorodetsky, MD, PhD

Health Scientist Administrator
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard
Bethesda, MD 20892
Phone: (301) 594-9004
Email: egorod@mail.nih.gov

J. Rafael Gorospe

Medical Officer
National Institute of General Medical
Sciences, NIH
45 Center Drive #2AS43P
Bethesda, MD 20835
Phone: (301) 435-0832
Email: gorospejr@mail.nih.gov

Faycal Guedj

Staff Scientist
National Institutes of Health
35A Convent Drive, Building 35A, Room 2F220
Bethesda, MD 20892
Phone: (617) 945-6729
Email: faycal.guedj@nih.gov

Fangjian Guo, MD, PhD

Assistant Professor
The University of Texas Medical Branch
301 University Boulevard
Galveston, TX 77550-0587
Phone: (409) 502-2532
Email: guofangjian@gmail.com

Arpana Gupta

UCLA Oppenheimer Center for Neurobiology of
Stress and Resilience
CHS 42-210
10833 Le Conte Avenue
Los Angeles, CA 90095-7378
Phone: (865) 406-0839
Email: agupta@mednet.ucla.edu

Sarthak Gupta, MD

Henry Metzger Scholar
National Institutes of Health
10 Center Drive, Room 12S257
Bethesda, MD 20892
Phone: (301) 443-8541
Email: sarthak.gupta@nih.gov

Jeannine M. Helm

National Institute of Dental and Craniofacial
Research, NIH
6701 Democracy Boulevard
Bethesda, MD 20892-4878
Phone: (301) 827-4629
Email: helmjm@nidcr.nih.gov

Rebecca Henry, PhD, RN

Program Director and AIDS Coordinator
National Institute of Nursing Research, NIH
6701 Democracy Boulevard
Bethesda, MD 20817
Phone: (240) 604-3669
Email: rebecca.henry@nih.gov

Tanya Hoodbhoy, PhD

Program Director
National Institute of General Medical
Sciences, NIH
45 Center Drive, MSC 6200
Bethesda, MD 20892
Phone: (301) 594-0943
Email: tanyah@mail.nih.gov

Zoe E. Huang

Scientific Review Official
National Library of Medicine, NIH
6705 Rockledge Drive, Room 301
Bethesda, MD 20817
Phone: (301) 594-4937
Email: huangz@nih.gov

Holly Hunsberger, PhD

Postdoctoral Fellow
Columbia University
5000 Broadway, #3C
New York, NY 10034
Phone: (609) 501-9533
Email: hh2694@cumc.columbia.edu

Chyren Hunter, PhD

Associate Director for Basic and
Translational Research
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Room 437
Bethesda, MD 20817
Phone: (301) 496-7849
Email: hunterc@nih.gov

Karen Huss, PhD

Health Scientist Administrator
National Institute of Nursing Research, NIH
8616 Aqueduct Road
Potomac, MD 20854
Phone: (301) 594-5970
Email: hussk@mail.nih.gov

Latifa Jackson

Howard University
2041 Georgia Avenue NW
Cancer Center Building, Room 615
Washington, DC 20060
Phone: (202) 806-4076
Email: latifa.jackson@howard.edu

Hadine Joffe, MD, MSc

Executive Director, Connors Center for
Women's Health and Gender Biology
Brigham and Women's Hospital
Harvard Medical School
75 Frances Street
Boston, MA 02115
Phone: (617) 732-4906
Email: hjoffe@bwh.harvard.edu

Nicole Johnston

National Institute on Drug Abuse, NIH
6001 Executive Boulevard, Room 4264
Rockville, MD 20852
Phone: (301) 443-3482
Email: nicole.johnston@nih.gov

Chloe J. Jordan

Post-doctoral Fellow
National Institute on Drug Abuse, NIH
12014 Chase Crossing Circle, #402
Rockville, MD 20852
Phone: (443) 740-2586
Email: chloe.jordan@nih.gov

Sheethal Jose

Scientific Program Analyst
National Human Genome Research
Institute, NIH
5635 Fishers Lane
Rockville, MD 20852
Phone: (301) 480-2314
Email: sheethal.jose@nih.gov

Pritty Joshi

Office of the Director, NIH
6705 Rockledge Drive, RKL 1 Room 5168
Bethesda, MD 20817
Phone: (301) 435-4949
Email: pritty.joshi@nih.gov

Nanette C. Joyce, DO, MAS

Associate Clinic Professor
University of California, Davis
4860 Y Street, Suite 3850
Sacramento, CA 95817
Phone: (916) 734-5291
Email: ncjoyce@ucdavis.edu

Wendy B. Katzman, PT, DPTSc, OCS

Professor Emeritus
University of California, San Francisco
1500 Owens Street, Suite 400
San Francisco, CA 94158
Phone: (415) 514-6776
Email: wendy.katzman@ucsf.edu

Candace L. Kerr

Program Officer
National Institute on Aging, NIH
7200 Wisconsin Avenue
Bethesda, MD 20892
Phone: (301) 827-4474
Email: candace.kerr@nih.gov

Aleksander Keselman, PhD

Post-doctoral Fellow
Johns Hopkins University
720 Rutland Avenue
Baltimore, MD 21205
Phone: (410) 955-1747
Email: akeselm1@jhmi.edu

Lisa A. Kilpatrick, PhD

Associate Researcher
University of California, Los Angeles
10833 Le Conte Avenue
CHS 42-210 MC 737818
Los Angeles, CA 90095
Phone: (310) 904-9781
Email: lisa@lisakilpatrick.com

Cheryl Kitt, PhD

Senior Science Advisor
National Institutes of Health
7605 Rockledge Drive
Bethesda, MD 20850
Phone: (301) 435-8403
Email: kittc@od.nih.gov

Karin L. Klingman, MD

Medical Officer
National Institute of Allergy and Infectious
Diseases, NIH
5601 Fishers Lane, MSC 9830
Rockville, MD 20852
Phone: (301) 825-4765
Email: kkl Klingman@niaid.nih.gov

Ronald A. Kohanski, PhD

Division Deputy Director
National Institute on Aging, NIH
Gateway Building, Suite 3N300
Bethesda, MD 20892
Phone: (301) 402-0836
Email: ronald.kohanski@nih.gov

Nazzarena Labo

National Cancer Institute, NIH
PO Box B
Frederick, MD 21702
Phone: (301) 846-5939
Email: labon@mail.nih.gov

Allison Lambert, MD, MHS

Clinical Instructor
University of Washington
603 W Sumner Avenue
Spokane, WA 99204
Phone: (917) 751-6975
Email: aalamb@uw.edu

Nancy E. Lane, MD

Distinguished Professor of Medicine
University of California, Davis
4625 Second Avenue, Suite 2006
Sacramento, CA 95817
Phone: (650) 703-4662
Email: nelane@ucdavis.edu

Jeeyoung Lee

National Institute of Diabetes and Digestive and
Kidney Diseases, NIH
10 Center Drive
Building 10, 9N317
Bethesda, MD 20814
Phone: (301) 496-2313
Email: jeeyoung.lee@nih.gov

Tamara Lewis Johnson

Chief, Women's Mental Health Program
Brigham and Women's Hospital
Harvard Medical School
6001 Executive Boulevard
Room 7209 MSC 9659
Rockville, MD 20892
Phone: (301) 594-7963
Email: tamara.lewisjohnson@nih.gov

Yuhong Lin

Staff Scientist
National Institute on Alcohol Abuse and
Alcoholism, NIH
5625 Fishers Lane, Room 3N07
Rockville, MD 20852
Phone: (301) 443-9031
Email: yulin@mail.nih.gov

Sarah D. Linnstaedt, PhD

Assistant Professor
University of North Carolina at Chapel Hill
109 Mason Farm Road
Taylor Hall, Room 350
Chapel Hill, NC 27517
Phone: (919) 576-6179
Email: sarah_linnstaedt@med.unc.edu

Liang Liu

Columbia University
1150 St. Nicholas Avenue
New York, NY 10032
Phone: (212) 851-4836
Email: ll2697@columbia.edu

Tania B. Lombo

National Institutes of Health
18016 Silver Leaf Drive
Gaithersburg, MD 20877
Phone: (301) 435-5736
Email: tania.lombo@nih.gov

Philip Lucas, PhD

Staff Scientist
National Institutes of Health
10 Center Drive, Room CRC 3-3288
Bethesda, MD 20892
Phone: (301) 435-3542
Email: pjucas@nih.gov

Christine Maric-Bilkan

National Heart, Lung, and Blood Institute, NIH
6701 Rockledge Drive
Bethesda, MD 20892
Phone: (301) 435-0486
Email: christine.maric-bilkan@nih.gov

Cheryl Marks, PhD

Associate Director
National Cancer Institute, NIH
9609 Medical Center Drive
Rockville, MD 20850
Phone: (240) 276-6217
Email: marksc@nih.gov

Santiago Martinez Gonzalez

National Center for Complementary and
Integrative Health, NIH
35 Convent Drive
Bethesda, MD 20892
Phone: (240) 671-2646
Email: santiago.martinezgonzalez@nih.gov

Bonnie J. Mathieson

Office of AIDS Research, OD, NIH
5601 Fishers Lane, MSC 9840
Rockville, MD 20892
Phone: (301) 402-3998
Email: mathiesb@od.nih.gov

Polly Matzinger

National Institute of Allergy and Infectious
Diseases, NIH
Twinbrook II, Room 100A
Rockville, MD 20852
Phone: (240) 444-2047
Email: pcm@helix.nih.gov

Matthew Mauck, MD, PhD

Assistant Professor
University of North Carolina
N2198 UNC Hospitals CB#7010
Chapel Hill, NC 27517
Phone: (414) 530-9344
Email: mmauck@aims.unc.edu;
matt_mauck@med.unc.edu

Carolyn A. Meske

Intern
Office of Planning, Analysis and Evaluation, NIH
31 Center Drive, MSC 2292
Bethesda, MD 20892
Phone: (301) 928-8375
Email: carolyn.meske@nih.gov

Catherine Monk, PhD

Columbia University Medical Center
622 W 168th Street, PH1540
New York, NY 10032
Phone: (646) 774-8941
Email: cem31@cumc.columbia.edu

Kathryn A. Morris, MPH

Health Science Policy Analyst
Office of Behavioral and Social Sciences
Research, NIH
31 Center Drive, Room B1C19
Bethesda, MD 20892
Phone: (301) 496-5594
Email: kathryn.morris@nih.gov

Gurueswar "Guru" Nagarajan

Post-doctoral Scientist
National Institute of Mental Health, NIH
35A Convent Drive 2D804A
Bethesda, MD 20892
Phone: (301) 594-2738
Email: gurueswar.nagarajan@nih.gov

Melissa Nagelin

Scientific Review Officer
National Heart, Lung, and Blood Institute, NIH
6701 Rockledge Drive, Room 7202
Bethesda, MD 20892
Phone: (301) 827-7951
Email: nagelinmh2@nhlbi.nih.gov

Chandra Nagineni

Scientist
National Cancer Institute, NIH
RBB, CCR
Bethesda, MD 20892
Phone: (301) 443-6490
Email: naginenic@mail.nih.gov

Kate Nagy

Deputy Director
Office of Planning, Analysis, and Evaluation, NIA
Building 31, Room 5C05
Bethesda, MD 22042
Phone: (301) 451-8396
Email: nagyk@mail.nih.gov

Concepcion R. Nierras, PhD

Program Leader
Children's National Medical Center
6001 Executive Boulevard, NSC Building
Room 8190H
Rockville, MD 20852
Phone: (301) 435-5840
Email: concepcion.nieras@nih.gov

Leia Novak, PhD

Scientific Program Manager
National Heart, Lung, and Blood Institute, NIH
6701 Rockledge Drive, Room 9143
Bethesda, MD 20892
Phone: (423) 306-3809
Email: Leia.novak@nih.gov

Mikhail Olfieriev
Research Associate
Hospital for Special Surgery
535 East 70th Street
New York, NY 10021
Phone: (212) 774-2514
Email: olferiev@mss.edu

Voula Osganian, MD,SCD
Program Director
National Institute of Diabetes and Digestive and
Kidney Diseases, NIH
6707 Democracy Boulevard
Bethesda, MD 20892
Phone: (301) 827-6939
Email: voula.osganian@nih.gov

Laura A. Pace, MD, PhD
Assistant Professor
University of Utah
30 N 1900 E, SOM 4R118
Salt Lake City, UT 84132
Phone: (801) 581-5679
Email: laura.pace@hsc.utah.edu

Christina E. Parisi
Intern
National Institute on Aging, NIH
Building 31, Room 5C05
Bethesda, MD 20892
Phone: (301) 827-1369
Email: christina.paris@nih.gov

Ronna Popkin
Health Specialist
National Institute of Child Health and Human
Development, NIH
6710B Rockledge Drive, Room 2209A
Bethesda, MD 20817
Phone: (301) 646-8379
Email: ronna.popkin@nih.gov

Rebecca Prevots, PhD
Epidemiologist
National Institute of Allergy and Infectious
Diseases, NIH
Building 15B-1
Bethesda, MD 20852
Phone: (301) 451-3058
Email: rprevots@niaid.nih.gov

Thiyagu Rajakannan
Research Fellow
ClinicalTrials.gov
38A, 7N712B, 8600 Rockville Pike
Bethesda, MD 20894
Phone: (301) 435-3157
Email: rajakann@mail.nih.gov

Ipolia Ramadan
National Institute of Neurological Disorders and
Stroke, NIH
6001 Executive Boulevard
Rockville, MD 20892
Phone: (301) 496-1431
Email: ramadanir@mail.nih.gov

Armin Raznahan, MD, PhD
Chief, Developmental Neurogenomics Unit
Brigham and Women's Hospital
Harvard Medical School
Room 4D19, Building 10
Bethesda, MD 29892
Phone: (301) 435-7927
Email: raznahan@mail.nih.gov

Elizabeth L. Read-Coinnole
Section Chief Cancer Etiology
National Cancer Institute, NIH
9609 Medical Center Drive
Rockville, MD 20850
Phone: (240) 276-6190
Email: bconole@mail.nih.gov

Sara Reardon
Reporter
Nature
968 National Press Building
529 14th St NW
Washington, DC 20045
Phone: (415) 902-2786
Email: sara.reardon@us.nature.com

Karen Regan, MS, RD
Nutritionist
National Institute of Diabetes and Digestive and
Kidney Diseases, NIH
2 Democracy Plaza, Room 623
6707 Democracy Boulevard
Bethesda, MD 20892-5461
Phone: (301) 435-6199
Email: kr184x@nih.gov

Saundra T. Rice
Project Manager
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20892
Phone: (301) 435-1040
Email: saundra.rice@nih.gov

Sonia Rosenfield

National Cancer Institute, NIH
1614 Tilton Drive
Silver Spring, MD 20902
Phone: (240) 781-3443
Email: sonia.rosenfield@nih.gov

Forough Saadatmand

Howard University
1840 7th Street NW
Washington, DC 20001
Phone: (202) 865-8348
Email: frough.saadatmand@howard.edu

Christen A. Sandoval, MSPH, CHES

Office of the Director, NIH
9000 Rockville Pike
Bethesda, MD 20892
Phone: (301) 435-7841
Email: christen.sandoval@nih.gov

Michael Sayre

Branch Chief
National Institute on Minority Health and Health
Disparities, NIH
6707 Democracy Boulevard, Suite 800
Bethesda, MD 20892
Phone: (301) 435-0962
Email: sayrem@mail.nih.gov

Irina Sazonova, PhD

Health Scientist Administrator
National Institute on Drug Abuse, NIH
6001 Executive Boulevard
Rockville, MD 20852
Phone: (617) 610-3145
Email: irina.sazonova@nih.gov

Katrina J. Serrano, PhD

Health Scientist Administrator
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20817
Phone: (301) 496-8931
Email: katrina.serrano@nih.gov

Ching-yi Shieh

Statistician
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 432
Bethesda, MD 20892
Phone: (301) 496-9186
Email: shiehchingyi@mail.nih.gov

Eric Shiroma

Staff Scientist/Epidemiologist
National Institute on Aging, NIH
7201 Wisconsin Avenue, Suite 2N300
Bethesda, MD 20814
Phone: (301) 496-5966
Email: eric.shiroma@nih.gov

Katherine P. Snedaker, LCSW

Executive Director
PINK Concussions
17 Harborview Avenue
Norwalk, CT 06854
Phone: (203) 984-0860
Email: katherine@pinkconcussions.com

Minkyo Song, MD, PhD

National Cancer Institute, NIH
9609 Medical Center Drive
Bethesda, MD 20892-9776
Phone: (240) 276-7985
Email: minkyo.song@nih.gov

Elizabeth Spencer

Deputy Director
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard
Bethesda, MD 20817
Phone: (301) 402-1770
Email: elizabeth.spencer@nih.gov

Bethany Stangl, PhD

National Institute on Alcohol Abuse and
Alcoholism, NIH
10 Center Drive
Building 10, Room 2-2352
Bethesda, MD 20892
Phone: (301) 451-9401
Email: stanglbi@mail.nih.gov

Al Staropoli, MS

Writer
Campbell and Company
8553 Boca Glades Boulevard W
Boca Raton, FL 33434
Phone: (202) 320-8040
Email: sciwriter@mac.com

Dori M. Steinberg, PhD, RD

Assistant Professor
Duke University
310 Trent Drive, Box 90519
Durham, NC 27708
Phone: (919) 613-5453
Email: dori.steinberg@duke.edu

Denise Stredrick

Health Scientist Administrator
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard
Rockville, MD 20852
Phone: (301) 594-9575
Email: denise.stredrick@nih.gov

Elizabeth A. Turcotte, BS, BA

Postbaccalaureate IRTA Fellow
National Institute of Child Health and Human
Development, NIH
4881 Battery Lane, #21
Bethesda, MD 20814
Phone: (319) 215-8807
Email: elizabeth.turcotte@nih.gov

L. Christine Turtzo, MD, PhD

Staff Clinician
National Institute of Neurological Disorders and
Stroke, NIH
10 Center Drive
Building 10, Room B1D733
Bethesda, MD 20850
Phone: (301) 435-7659
Email: turtzolc@mail.nih.gov

Claire-Marie Vacher

Children's National Medical Center
111 Michigan Avenue NW
Washington, DC 20010
Phone: (240) 593-7935
Email: cvacher3@cnmc.org

Dimitrios Vatakis, PhD

Scientific Review Officer
Center for Scientific Review, NIH
6701 Rockledge Drive
Bethesda, MD 20892
Phone: (301) 827-7480
Email: vatakisdn@nih.gov

Kristin Voegtline, PhD

Assistant Professor of Pediatrics
Johns Hopkins School of Medicine
200 N Wolfe Street, Room 2076
Baltimore, MD 21286
Phone: (410) 614-3866
Email: kvoegt1@jhu.edu

Neeta Vora, MD

University of North Carolina at Chapel Hill
3010 Old Clinic Building, CB 7516
Chapel Hill, NC 27514
Phone: (919) 966-2222
Email: nvora@med.unc.edu

Molly Wagster

Chief, Behavioral and Systems
Neuroscience Branch
National Institute on Aging, NIH
Gateway Building, Suite 3E400
7201 Wisconsin Avenue
Bethesda, MD 21230
Phone: (301) 594-7768
Email: wagster@nia.nih.gov

Jason Wan

National Institute of Dental and Craniofacial
Research, NIH
6701 Democracy Boulevard
Bethesda, MD 20892
Phone: (301) 594-9898
Email: jasonwan@mail.nih.gov

Annie Waterman

Clinical Operations Manager
National Institute of Allergy and Infectious
Diseases, NIH
5601 Fishers Lane
Bethesda, MD 20852
Phone: (240) 292-4831
Email: annie.waterman@nih.gov

Davene White, RN, NNP, MPH

Director, HUH CARES Public Health Programs
Howard University Hospital
Department of Pediatrics and Child Health
2041 Georgia Avenue NW
Washington, DC 20060
Phone: (202) 865-7012
Email: dmwhite@howard.edu

Jamie White

Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard
Bethesda, MD 20952
Phone: (301) 496-9200
Email: jamie.white@nih.gov

Elizabeth Wilder, PhD

Director, Office of Strategic Coordination
Children's National Medical Center
6001 Executive Boulevard
Rockville, MD 20852
Phone: (301) 496-0149
Email: wildere@od.nih.gov

LaKeisha Williams, PharmD, MPH
Clinical Assistant Professor of Pharmacy
Tulane University
248 Masters Point Court
Slidell, LA 70458
Phone: (504) 988-6282
Email: llgeorge@xula.edu

Lamont Williams
Acting Communications Director
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20817
Phone: (301) 435-6828
Email: lamont.williams@nih.gov

ClarLynda Williams-DeVane, PhD
Assistant Professor/Director Bioinformatics
North Carolina Central University/Duke
(BIRCWH)
9001 Mustard Seed Lane
Garner, NC 27529
Phone: (919) 368-0010
Email: clarlynda.williams@ncsu.edu

Barton L. Wise, MD
Associate Professor
Center for Musculoskeletal Health
University of California, Davis
School of Medicine
4625 2nd Avenue, Suite 2000
Sacramento, CA 95817
Phone: (916) 734-4534
Email: blwise@ucdavis.edu

Anchi Wu
Intramural Post-baccalaureate Research Fellow
National Institutes of Health
445 Elmcroft Boulevard
Rockville, MD 20850-5670
Phone: (408) 981-5532
Email: anchi.wu.1@gmail.com

Ashley Xia
Program Director
National Institutes of Health
6707 Democracy Boulevard, Room 6074
Bethesda, MD 20817
Phone: (301) 480-9505
Email: ashley.xia@nih.gov

Yanqin Yang
National Institutes of Health
10 Center Drive, Room 7S261
Bethesda, MD 20892
Phone: (301) 827-0858
Email: yanqin.yang@nih.gov

Wei Yao
University of California, Davis
4625 2nd Avenue
Sacramento, CA 95817
Phone: (916) 734-0763
Email: yao@ucdavis.edu

Melanie Young, MS, MPH
Biologist
U.S. Environmental Protection Agency
1301 Constitution Avenue NW
Washington, DC 20004
Phone: (202) 250-8817
Email: young.melanie@epa.gov

Tanzira Zaman, MD
Fellow, Division of Pulmonary Sciences
and Critical Care Medicine
University of Colorado
755 E 19th Avenue, #521
Denver, CO 80203-5533
Phone: (434) 242-3917
Email: tanzira.zaman@ucdenver.edu

Wei-Qin Zhao
Scientific Review Officer
Center for Scientific Review, NIH
6701 Rockledge Drive
Bethesda, MD 20892
Phone: (301) 827-7238
Email: wei-qin.zhao@nih.gov

Steven J. Zullo, PhD
Program Officer
National Institute of Biomedical Imaging and
Bioengineering, NIH
6707 Democracy Boulevard
Bethesda, MD 20892
Phone: (240) 271-9007
Email: steven.zullo@nih.gov

INVITED SPEAKERS

Nicole E. Basta, PhD

Assistant Professor
University of Minnesota
1300 Second Avenue South
West Bank Office Building, Suite 300
Minneapolis, MN 55454
Phone: (612) 625-6616
Email: nebasta@umn.edu

Kyle J. Burghardt, PharmD

Assistant Professor of Pharmacy Practice
Wayne State University Eugene Applebaum
College of Pharmacy and Health Sciences
259 Mack Avenue, Suite 2190
Detroit, MI 48201
Phone: (313) 577-3132
Email: kburg@wayne.edu

Jayne Danska, PhD

Senior Scientist
The Hospital for Sick Children
555 University Avenue
Toronto, ON M5G 1X8
Phone: (416) 813-8810
Email: jayne.danska@sickkids.ca

Scott Hultgren, PhD

Washington University School of Medicine
666 South Euclid Avenue
St. Louis, MO 63110
Phone: (314) 362-6772
Email: hultgren@wustl.edu

David A. Hunstad, MD

Associate Professor of Pediatrics
(Infectious Diseases)
Washington University School of Medicine
Campus Box 8208
660 South Euclid Avenue
Saint Louis, MO 63010
Phone: (314) 780-0301
Email: dhunstad@wustl.edu

Emeran A. Mayer, MD

University of California, Los Angeles
21448 Entrada Road
Topanga, CA 90290
Phone: (310) 614-5287
Email: emayer@ucla.edu

Margaret M. McCarthy, PhD

Professor and Chair
University of Maryland School of Medicine
655 W Baltimore Street, BRB 4-009
Baltimore, MD 21201
Phone: (410) 706-2655
Email: mmccarthy@som.umaryland.edu

Virginia M. Miller, PhD

Mayo Clinic
200 First Street SW
Rochester, MN 55902
Phone: (507) 284-2290
Email: miller.virginia@mayo.edu

Anne Z. Murphy, PhD

Associate Professor
Georgia State University
Neuroscience Institute
100 Piedmont Avenue SE
Atlanta, GA 30303
Phone: (571) 243-3908
Email: amurphy@gsu.edu

Brian Nieman, PhD

Senior Scientist
The Hospital for Sick Children
25 Orde Street
Toronto, ON M5T 3H7
Phone: (416) 813-7654
Email: brian.nieman@sickkids.ca

Anna Penn, MD, PhD

Associate Professor and Attending Neonatologist
Children's National Medical Center
111 Michigan Avenue NW, Room M7635
Washington, DC 20010
Phone: (202) 476-4324
Email: apenn@childrensnational.org

Michèle Ramsay, PhD

Director of the SBIMB, Professor of Human
Genetics
University of the Witwatersrand
SBIMB, 9 Jubilee Road
Johannesburg, South Africa
Phone: (014) 119-7777
Email: michele.ramsay@wits.ac.za

Barbara Stranger, PhD

University of Chicago
900 E 57th Street, KCBD 10134
Chicago, IL 60637
Phone: (617) 669-1200
Email: bstranger@medicine.bsd.uchicago.edu

Eugenia Trushina, PhD

Associate Professor, Department of Neurology
Mayo Clinic
200 First Street SW
Rochester, MN 55905
Phone: (507) 284-8197
Email: trushina.eugenia@mayo.edu

Lauren A. Weiss, PhD

Associate Professor
University of California, San Francisco
401 Parnassus Avenue, Room A101
Nina Ireland Lab, Box F-0984
San Francisco, CA 94143
Phone: (415) 476-7650
Email: lauren.weiss@ucsf.edu

MODERATORS**Rajeev K. Agarwal, PhD**

Senior Research Program Officer
Office of Research on Women's Health,
OD, NIH
6707 Democracy Boulevard, Suite 400
Bethesda, MD 20878
Phone: (301) 402-1770
Email: rajeev.agarwal@nih.gov

Paul Barrett, PhD

Health Specialist
Office of Strategic Coordination, OD, NIH
6001 Executive Boulevard
Rockville, MD 20852
Phone: (301) 827-6911
Email: paul.barrett@nih.gov

Colin Fletcher, PhD

Program Director
National Human Genome Research
Institute, NIH
5635 Fishers Lane, Suite 4076
Bethesda, MD 20892-9305
Phone: (301) 480-2270
Email: fletcherc2@mail.nih.gov

Judy Hewitt, PhD

National Institute of Allergy and Infectious
Diseases, NIH
5601 Fishers Lane
Bethesda, MD 20892
Phone: (240) 627-3274
Email: jhewitt@nih.gov

Jennifer L. Troyer, PhD

Program Director
National Human Genome Research
Institute, NIH
5635 Fishers Lane, Suite 4076
Rockville, MD 20852
Phone: (301) 312-3276
Email: troyerj@mail.nih.gov

CONTRACTOR STAFF**Michele Dillon**

Director of Conference Services
Rose Li and Associates, Inc.
9501 Wadsworth Drive
Bethesda, MD 20817
Phone: (240) 630-0774
Email: michele@roseliassociates.com

Valery Leng

Meetings Assistant
Rose Li and Associates, Inc.
9501 Wadsworth Drive
Bethesda, MD 20817
Phone: (301) 793-8588
Email: valery.leng@roseliassociates.com

Carrine V.M. Todman-Lewis, DBA, MTA, CMP

Events Strategist
Rose Li and Associates, Inc.
9501 Wadsworth Drive
Bethesda, MD 20817
Phone: (240) 630-0773
Email: carrine@roseliassociates.com