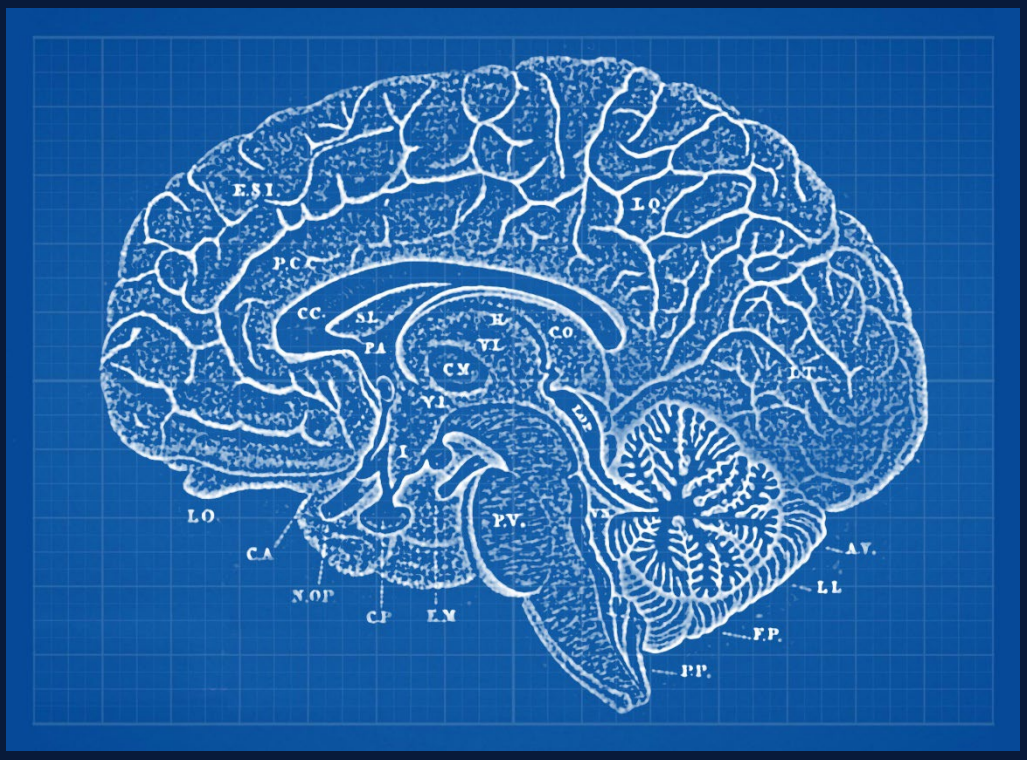
Writing Your Own Blueprint



2023 NIH Blueprint Diversity Conference

NIH Blueprint for Neuroscience Research

Monday, July 10, 2023
11:00 a.m. – 5:00 p.m. EDT

Tuesday, July 11, 2023
10:50 a.m. – 5:00 p.m. EDT

Table of Contents

Meeting Information 3
Agenda (Day 1) 4
Agenda (Day 2) 5
Speaker Biographies 6
Poster Room A (2:00 – 3:00 pm EDT) 10
Poster Room B (3:00 – 4:00 pm EDT) 18
Registrants 25
Resources 32

Meeting Information

Meeting Goals

The goals of this meeting are to provide a forum for diverse neuroscience scholars to interact with NIH staff, develop peer-to-peer networks, and participate in professional development opportunities and leadership training. It will bring together participants from several NIH Blueprint and BRAIN Initiative programs: BP-ENDURE alumni who are current graduate students or postdocs; D-SPAN fellows (graduate students and postdocs); BRAIN Initiative K99/R00 awardees (postdocs and junior faculty); and BRAIN Initiative supplementees (graduate students and postdocs); in addition to other diverse trainees from NINDS funded programs (e.g. Neuroscience Scholars Program). The program theme this year will focus on strengthening communication (online, presentation, and writing) to market the science. The training will work to improve or enhance your science communications skills and focus on how to develop and market your professional and scholarly brand. These skills will help you network within your scientific community to enhance your visibility and clearly convey your research to others.

The NIH Blueprint for Neuroscience Research

The NIH Blueprint for Neuroscience Research aims to accelerate transformative discoveries in brain function in health, aging, and disease. Blueprint is a collaborative framework that includes the NIH Office of the Director together with NIH Institutes and Centers that support research on the nervous system. By pooling resources and expertise, Blueprint identifies cross-cutting areas of research and confronts challenges too large for any single Institute or Center. Since its inception in 2004, Blueprint has supported the development of new research tools, training opportunities, and resources to assist neuroscientists.

In addition to supporting cross-cutting neuroscience activities like research training, workforce diversity, and therapeutic development, Blueprint also funds research initiatives. Topics have ranged from transforming our understanding of dynamic neuroimmune interactions to enhancing our fundamental knowledge of interoception, supporting the development of innovative tools and technologies to monitor and manipulate biomolecular condensates, and more. To learn about both current and past areas of research, visit the Blueprint Research Initiatives page.

The NIH Office of the Director and these NIH Institutes and Centers participate in the NIH Blueprint for Neuroscience Research:

- **NCCIH**
- **NEI**
- **NIA**
- **NIAAA**
- **NIBIB**
- **NICHD**
- **NIDA**
- **NIDCR**
- **NIEHS**
- **NIMH**
- **NINDS**
- **OBSSR**

For further information about the NIH Blueprint, visit https://neuroscienceblueprint.nih.gov/

Agenda (Day 1)

All times in EDT

Monday, July 10, 2023

Main Zoom Session (Sign in with your personal link)

11:00 – 11:15 am	**NIH BRAIN Initiative Welcome and NIH Neuroscience Research Vision** *John Ngai, Ph.D., Director, NIH BRAIN Initiative*
11:15 – 11:25 am	**Meeting Goals** *Michelle Jones-London, Ph.D., Chief, Office of Programs to Enhance Neuroscience Workforce Diversity (OPEN), National Institute of Neurological Disorders and Stroke*
11:25 – 12:00 pm	**Network Building Activities**
12:00 – 4:55 pm	**Science Communication Training** *Bri McWhorter, Founder and CEO, Activate to Captivate* **Strengthening Communication to Market Your Science:** *This training will cover techniques to help you improve your scientific communication skills as you elevate and market your professional scholarly brand. This interactive session will cover tips to overcome presentation nerves, present your work in various spaces, and distill complex information into engaging narratives. These new skills will help you network within numerous communities to enhance your visibility and clearly convey your work to others.* *Training will involve breakout rooms. There will be breaks at 12:45pm (15 min break), 2:25pm (30 min break), and 3:45pm (10 min break).*
4:55 – 5:00 pm	**Wrap-Up**

Agenda (Day 2)

All times in EDT

Tuesday, July 11, 2023 (Day 2)

Main Zoom Session (Sign in with your personal link)

10:50 – 11:00 am	**Network Building Activities**
11:00 – 11:15 am	**NIH Blueprint Welcome and NIH Investment in Neuroscience** *Walter Koroshetz, M.D., Director, NINDS*
11:15 – 11:20 am	**Day 1 Summary and Day 2 Goals** *Lauren Ullrich, Ph.D., Program Director, OPEN, NINDS*
11:20 – 12:20 pm	***Keynote:*** **Designing an Online Presence for Academics** *Letisha R. Wyatt, Ph.D., Assistant Professor of Neurology, Oregon Health & Science University*
12:20 – 1:20pm	***Panel Discussion:*** **Communicating Science in Creative Ways** Moderator: *Marguerite Matthews, Ph.D., Program Director, OPEN, NINDS* *AZA Allsop, M.D., Ph.D., Resident in Psychiatry, Yale School of Medicine* *Sarah Kucenas, Ph.D., Professor of Biology, University of Virginia* *Natalia Vélez, Ph.D., Postdoctoral Fellow, Harvard University*
1:20 – 1:30 pm	**Closing Remarks**
1:30 – 2:00 pm	**Lunch Break**
2:00 – 5:00pm	**Poster Session in Gather.Town** *Poster Room A (2:00 – 3:00pm)* *Poster Room B (3:00 – 4:00pm)* *Networking (4:00 – 5:00pm)*

Speaker Biographies

AZA Allsop, M.D., Ph.D.
Resident in Psychiatry
Yale School of Medicine

AZA Allsop is an artist, neuroscientist, and psychiatrist. He conducts research at the intersection of social cognition, music, mindfulness, and psychedelics. His research and clinical work is guided by the belief that decoding these tools will provide a better understanding of how social groups function and offer insights into treating mental suffering and enhancing the evolution of society at large. He studied Biology, Philosophy, and Jazz Studies at North Carolina Central University, received his MD from Harvard Medical School, PhD in Neuroscience from MIT and was an Emerson Scholar at Berklee College of Music. He served as chief resident on the Clinical Neuroscience Research Unit in the Department of Psychiatry at Yale University. He teaches meditation and yoga and co-founded Renaissance Entertainment, LLC, and Mefreely, LLC. companies that combine music, science, and community building to impact social change.

Michelle Jones-London, Ph.D.
Chief, Office of Programs to Enhance Neuroscience Diversity (OPEN)
National Institute of Neurological Disorders and Stroke (NINDS)

Dr. Michelle D. Jones-London serves as Chief, Office of Programs to Enhance Neuroscience Workforce Diversity (OPEN-WD). In this position, she plays a critical role in guiding the Institute's diversity efforts and chairs the NINDS Diversity Working Group. Dr. Jones-London joined NINDS as a Program Director in July 2006. Dr. Jones-London earned her Ph.D. in Neuroscience from the Department of Neuroscience and Anatomy at Pennsylvania State University College of Medicine. She then received postdoctoral training as a research fellow at University of Pennsylvania in the Department of Psychiatry. Dr. Jones-London came to the NIH in July 2004 as an Emerging Leader Fellow; she performed duties across the Department of Health and Human Services including the Center for Scientific Review, FDA Office of Women's Health Science Program, and the Immediate Office of the Secretary, Intergovernmental/Tribal Affairs Office. Dr. Jones-London directs the diversity training and workforce development programs at NINDS which include Diversity and Re-Entry Supplements, Predoctoral Fellowships to Promote Diversity in Health-Related Research (F31), Career Development Awards to Promote Diversity (MOSAIC K99/R00 and K01) and Diversity Research Education Grants (R25) (including the Neuroscience Scholars Program with SfN). She also provides oversight for the Institute's diversity outreach initiatives at several other national scientific conferences. Her trans-NIH efforts include oversight for the NIH Blueprint ENDURE and DSPAN (F99/K00) programs, the BRAIN Initiative Diversity K99/R00, former Project Scientist for the NIH National Research Mentoring Network (NRMN) and part of the leadership team for NIH Faculty Institutional Recruitment for Sustainable Transformation (FIRST). Her research interests have focused on understanding monoaminergic neurotransmitter regulation and mechanisms of behavioral psychopharmacology in animal models of disorders such as ADHD, Tourette Syndrome, and depression.

Walter Koroshetz, M.D.
Director
National Institute of Neurological Disorders and Stroke

Walter J. Koroshetz, M.D., was selected Director of NINDS on June 11, 2015. Dr. Koroshetz joined NINDS in 2007 as Deputy Director, and he served as Acting Director from October 2014 through June 2015. Previously, he served as Deputy Director of NINDS under Dr. Story Landis. Together, they directed program planning and budgeting, and oversaw the scientific and administrative functions of the Institute. He has held leadership roles in a number of NIH and NINDS programs including the NIH's BRAIN Initiative, the Traumatic Brain Injury Center collaborative effort between the NIH intramural program and the Uniformed Health Services University, and the multi-year work to develop and establish the NIH Office of Emergency Care Research to coordinate NIH emergency care research and research training.

Sarah Kucenas, Ph.D.
Professor of Biology
University of Virginia

Sarah Kucenas is fascinated by the developing brain. Specifically, she and her research group study how glia act as engineers of neural development. Her long-term goal is to understand the mechanisms that mediate cellular interactions between neurons and glia and use this information to better understand how the human nervous system is initially sculpted, maintained, and behaves during disease. Sarah earned a B.Sc. in Biology from Valparaiso University in 2000 and went on to earn a Ph.D. in Pharmacological & Physiological Science from Saint Louis University with Dr. Mark Voigt in 2005. After Dr. Kucenas' postdoctoral work with Dr. Bruce Appel at Vanderbilt University, she joined the faculty at the University of Virginia in 2009. Sarah has a 11-year-old daughter, Madelyn, 3 (VERY big) dogs, and is a life-long swimmer.

Marguerite Matthews, Ph.D.
Program Director, OPEN
NINDS

Marguerite Matthews, PhD, is a Program Director in the Office of Programs to Enhance Neuroscience Workforce Diversity. Dr. Matthews manages various NINDS diversity initiatives and programs that provide neuroscience research training and career development. Before working at NINDS, Dr. Matthews came to NIH as a 2016-2018 AAAS Science & Technology Policy Fellow in the Office of Extramural Research, within the Division of Biomedical Research Workforce and the Division of Loan Repayment. She earned a BS in biochemistry from Spelman College and a PhD in neuroscience from the University of Pittsburgh, studying the development of the dopamine system during adolescence. She completed her postdoctoral fellowship at the Oregon Health & Science University, where she examined the development of the human brain using functional and structural neuroimaging.

Bri McWhorter
Founder and CEO
Activate to Captivate

Bri McWhorter is the Founder and CEO of Activate to Captivate, where she teaches communication techniques from an actor's point of view. She specializes in public speaking, scientific communications, interview skills, and interpersonal communications. She has taught workshops at Fortune 500 companies, is an executive coach for numerous organizations, and leads certificate programs at top universities. She enjoys helping people connect with their audiences by distilling complex information into captivating narratives. She has an MFA in Acting from University of California, Irvine and a BA in Theater and Performance Studies from University of California, Berkeley.

John Ngai, Ph.D.
Director
NIH Brain Research through Advancing Innovative Neurotechnologies (BRAIN®) Initiative

John J. Ngai, Ph.D., is the Director of the NIH's Brain Research through Advancing Innovative Neurotechnologies (BRAIN®) Initiative, where he oversees the long-term strategy and day-to-day operations of this ground-breaking enterprise. Dr. Ngai earned his bachelor's degree in chemistry and biology from Pomona College, Claremont, California, and Ph.D. in biology from the California Institute of Technology (Caltech) in Pasadena. He was a postdoctoral researcher at Caltech and at the Columbia University College of Physicians and Surgeons before starting his faculty position at the University of California at Berkeley. During more than 25 years as a UC Berkeley faculty member, Dr. Ngai has trained 20 undergraduate students, 24 graduate students and 15 postdoctoral fellows in addition to teaching well over 1,000 students in the classroom. His work has led to the publication of more than 70 scientific articles in some of the field's most prestigious journals and 10 U.S. and international patents. Dr. Ngai has received many awards including from the Sloan Foundation, Pew Charitable Trusts, and McKnight Endowment Fund for Neuroscience. As a faculty member, Dr. Ngai has served as the director of Berkeley's Neuroscience Graduate Program and Helen Wills Neuroscience Institute. He has also provided extensive service on NIH study sections, councils and steering groups, including as previous co-chair of the NIH BRAIN® Initiative Cell Census Consortium Steering Group.

Lauren Ullrich, Ph.D.
Program Director, OPEN
NINDS

Lauren Ullrich received her PhD and MS in Neuroscience from Georgetown University, researching memory in early Alzheimer's disease for her thesis and published on teaching, pedagogy, and professional development in science. She received her B.A. from Swarthmore College in psychobiology. Prior to coming to NINDS as a AAAS Science & Technology Fellow, Lauren worked for the Society for Neuroscience in a range of policy and programmatic areas, including government and public affairs; scientific rigor and reproducibility; workforce and training; and animals in research. At NINDS, she helps coordinate NINDS's diversity activities, which span the pipeline from neuroscience education outreach (grades K-12) to funding opportunities and mentoring networks across critical career transition points.

Natalia Vélez, Ph.D.
Postdoctoral Fellow
Harvard University

Natalia Vélez is a NIH DSPAN postdoctoral fellow at Harvard University and an incoming assistant professor at Princeton University (August 2023). Her research examines the foundations of human collaboration using a combination of behavioral experiments, computational modeling, and neuroimaging using fMRI. Outside of the lab, Natalia loves drawing and sharing portraits of scientists alongside their research.

Letisha Wyatt, Ph.D.
Assistant Professor of Neurology
Oregon Health & Science University

Letisha R. Wyatt is faculty in the Department of Neurology. She holds a Ph.D. in Molecular Pharmacology &Toxicology from the University of Southern California (2013). Her graduate and postdoctoral research focused on purinergic signaling in the central nervous system as a molecular target for new treatments for alcohol abuse and stroke. Letisha is a former NIH predoctoral fellow and has a strong record of mentorship in the laboratory and classroom. As the Director of Diversity in Research in the Office of Research and Innovation Office, part of her work involves supporting campus-wide efforts for enhancing inclusivity in laboratories and graduate education. More recently, Letisha has taken on the role of Director of Innovative Policy at the Racial Equity and Inclusion Center in the Vollum Institute where she focuses on building operational plans and internal systems to achieve organizational effectiveness in progressive workplace policies that advance racial equity. A major goal of her effort in this area is to engage with institutional leadership so that they may be empowered to identify structural racism and actively participate in the work to dismantle it. Read more about Letisha's role on her personal website (https://www.letisharwyatt.com/) and view her work on ORCID: 0000-0003-1026-5232.

Poster Room A (2:00 – 3:00 pm EDT)

Cognition

1. Effectivity of a Creative Educational Instrument to Improve the Assessment of Spelling Skills in Hispanic/Latinx Children

Presenting Author: Almarely Berrios Negron, Ponce Health Sciences University
Additional Author(s): Mario Bermonti Perez, Ponce Health Sciences University

Baddeley's Working Memory Model (BWMM) comprises the Central Executive Network (CEN) and the Phonological Loop System (PLS), responsible for modulating sustained attention and overseeing verbal comprehension, respectively. To study the PLS and assess spelling skills, we developed a Python-based tool named 'mSpelling.' However, we aim to make it more engaging by evaluating the impact of visual arts on sustained attention, as they have shown promise in enhancing motivation and engagement. This combination of visual arts and technology remains unexplored in Puerto Rico. Therefore, there is a critical need to design a new approach to improve cognitive skill assessment for the Hispanic/Latinx community. This study aims to examine the effectiveness of implementing visual arts in a tool that measures spelling skills and assess if it can effectively increase motivation, engagement, and sustained attention. We will select 50 children from second to fourth grade using convenience sampling. A Multiple Regression Analysis will be conducted using SPSS to compare the original version of the tool with the creative version that includes visual arts. Motivation will be measured by the quantity of completed tasks per version, with children having a total of 50 tasks and the freedom to choose the number of tasks they wish to complete. Greater task completion will indicate higher motivation. Engagement will be evaluated through on-screen time (OST) breaks, counting each instance where the children's focus shifts away from the screen as a break, representing reduced sustained attention. Fewer OST breaks will indicate improved sustained attention. These measurements will be correlated to assess sustained attention. It is hypothesized that the creative version of mSpelling will enhance children's motivation and engagement, ultimately improving their sustained attention. We anticipate observing an increase in the number of tasks completed and a decrease in OST breaks in the creative version of mSpelling. This research aims to address the significant gap in evaluating the effectiveness of creative tools incorporating visual arts and technology, considering the specific needs of Hispanic/Latinx children. The knowledge gained from this study will inform the design of future versions of this tool.

Acknowledgement of funding sources: This research was supported by NIH NIMH R25 MH055929, Training in Professional Development for Neuroscience.

2. Localizing Arithmetic in the Adult Bilingual Brain

Presenting Author: Vanessa Cerda, Vanderbilt University
Additional Author(s): Macarena Suarez-Pellicioni[1]; James Booth[2]; Nicole Wicha[3]
[1]University of Alabama; [2]Vanderbilt University; [3]University of Texas at San Antonio

Verbally memorized multiplication tables are thought to be language-specific memories. Supporting this idea, bilinguals are typically faster and more accurate in the language in which they learned math (LA+) than in their other language (LA-). Previous studies suggest that the left superior and middle temporal gyri (STG/MTG) are associated with representing memorized arithmetic facts. The inferior frontal gyrus (IFG) is engaged in effortful retrieval when facts do not have a robust memory

representation in temporal cortex, such as with less practiced, large multiplication problems. Additionally, the intraparietal sulcus (IPS) is engaged when mental calculation is necessary. The current study aimed to investigate two questions in Spanish-English bilingual adults: 1) Does solving multiplication problems in LA- show greater recruitment of IFG reflecting more effortful verbal retrieval or IPS reflecting reliance on calculation processes? 2) Is there a language by problem size interaction, where language differences are more pronounced for less practiced, large multiplications (e.g., 8x9) in comparison to more familiar, small ones (e.g., 2x3)? We used functional magnetic resonance imaging (fMRI) while participants performed two localizer tasks to identify hypothesis-driven regions of interest (ROIs) in verbal areas (left STG/MTG, left IFG) and quantity areas (bilateral IPS) as well as multiplication verification tasks in English and Spanish. No cluster reached significance for the direct comparison of languages (question 1) or for the interaction between language and problem size (question 2). An exploratory analysis found a main effect of problem size, where small problems recruited left STG/MTG and left IFG to a greater extent than large problems, suggesting greater verbal involvement for these problems. Additionally, large problems recruited right IPS to a greater extent than small problems, suggesting greater reliance of calculation for these problems. Our results suggest that bilingual adults engage similar brain regions across languages, even for more difficult problems.

Acknowledgement of funding sources: This research was funded by award 5R21HD098878-02 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development. V.R.C. was funded by the NIH Blueprint and BRAIN Initiative Diversity Specialized Predoctoral to Postdoctoral Advancement in Neuroscience Award 8K00HD112280-03.

3. Learning Representations of Environmental Priors in Visual Working Memory

Presenting Author: Tahra Eissa, University of Colorado Boulder
Additional Author(s): Zachary Kilpatrick, University of Colorado Boulder

Experience helps us learn the structure of the environment. Such learning can be described by statistical inference models in which environmental priors adapt to new information across time and influence future estimates. For instance, we may learn that certain colors are overrepresented in the world (e.g., I will see more greens and browns if I am in a forest), leading to cognitive biases in favor of more common environmental features. Humans display systematic biases when retaining estimates of an object's features in working memory, which may reflect learned priors of object feature values. However, the neural mechanisms that support inferring the environmental prior and producing biases in working memory have not been identified.

We build on observations from human response data that show systematic biases in a delayed-estimation task of color reports to determine if humans modulate their biases based on their experience. Considering subject responses when task stimuli (colors) were drawn from heterogeneous distributions that did not necessarily correspond with reported population biases, we confirm that most subjects' response distributions are better described by models that learn feature distributions than those that do not. We also found that a neural circuit model that infers the environmental prior via long-term potentiation and homeostatic plasticity could replicate these behavioral findings and represent experienced stimulus history. Changes to synaptic connectivity shape the persistent and collective neural activity that encodes the stimulus estimate in working memory, producing neural activity attractors are aligned to common stimulus values and mechanistically implementing probabilistic priors. This work suggests that systematic limitations in

working memory reflect efficient representations of inferred environmental structure, providing new insights into how humans integrate environmental knowledge into their cognitive strategies.

Acknowledgement of funding sources: BRAIN Initiative 1K99NS127855-01A1 and 1R01EB029847-01

4. Orbitofrontal Ensembles Integrate Taste, Movement, and Reward Predictions During Learning

Presenting Author: Evan Hart, National Institute on Drug Abuse
Additional Author(s): Lisette Bahena[1]; Geoffrey Schoenbaum[1]
[1]National Institute on Drug Abuse

Learning the meaning of cues is necessary for survival. Humans and other animals learn where and how to acquire food, find mates, or avoid predators. Cues signal many aspects of survival behavior. Road signs tell us which way to turn to acquire food (movement), which specific food is offered (e.g., ice cream vs frozen yogurt; taste/outcome identity), as well as whether food is offered at all (reward prediction). We also discriminate cues which share the same meaning from their sensory properties (sensory cue identity - e.g., a red octagon and a red light both mean stop). The integrated representation of these features of the behavioral landscape has been called a "cognitive map", the construction and use of which confer the ability to make predictions based on direct experience and to make inferences in novel situations. But how is this information acquired, integrated, and used - what neural circuits underly cognitive map formation? The orbitofrontal cortex (OFC) is thought to be important for cognitive map formation, particularly generating reward predictions. Generally these studies were performed in highly trained subjects and not during learning, when maps are formed, leaving unanswered questions about how the OFC forms integrated representations and how their activity evolves to highlight information of biological relevance. I recorded OFC ensemble spiking activity in rats during the learning of a task that allowed me to dissociate sensory from motor, taste, and reward encoding. During learning, OFC ensemble activity evolved toward simpler representations of reward and movement predictions, suggesting that cognitive map formation in the OFC involves simplifying task demands into whichever information is most directly relevant. These data have important implications for understanding the role of the OFC in learning and using predictive information carried by cues to make correct decisions, psychological processes affected in myriad neurological and psychiatric conditions.

Acknowledgement of funding sources: K99 DA053633; Fi2 GM133534; ZIA DA000587

5. Scaling of Smaller Pyramidal Neuron Size and Lower Energy Production in Schizophrenia

Presenting Author: Kirsten E Schoonover, University of Pittsburgh
Additional Author(s): Nora E Miller[1]; Kenneth N Fish[1]; David A Lewis[1]
[1]University of Pittsburgh

Background: Dorsolateral prefrontal cortex (DLPFC) dysfunction in schizophrenia appears to reflect, in part, alterations in layer 3 pyramidal neurons (L3PNs). Fewer dendritic spines and smaller cell bodies are thought to result in lower activity, and thus less demand for energy production in L3PNs. However, somal size and markers of energy production have not been simultaneously assessed within individual L3PNs.

Study Design: Fluorescent in situ hybridization (FISH) of vesicular glutamate transporter 1 (VGLUT1) mRNA was combined with immunohistochemical-labeling of NeuN to validate the cytoplasmic distribution of VGLUT1 mRNA as a means for unbiased identification of L3PNs and quantification of

their somal size. Dual-label FISH for VGLUT1 mRNA and a marker of energy production, cytochrome C oxidase subunit 4I1 (COX4I1) mRNA, was used to determine the somal size and COX4I1 mRNA levels, respectively, in individual DLPFC L3PNs from schizophrenia and unaffected comparison (UC) subjects.

Study Results: Measures of L3PN somal size with NeuN immunohistochemistry or VGLUT1 mRNA provided nearly identical results (ICC=0.96). Mean somal size of VGLUT1 mRNA-positive L3PNs was 8.7% smaller and mean COX4I1 mRNA levels per L3PN were 16.7% lower in schizophrenia. These measures were strongly positively correlated across individual L3PNs in both subject groups (rrm=0.81-0.86).

Conclusions: Using an unbiased methodology to identify L3PNs, we replicated findings of smaller somal size and lower COX4I1 mRNA levels in schizophrenia. The normal scaling of COX4I1 mRNA levels with L3PN somal size in schizophrenia supports the hypothesis that lower energy production reflects lower excitatory drive to L3PNs in the illness.

Acknowledgement of funding sources: This work was supported by the National Institute of Mental Health (grant numbers NIMH043784 to DAL, NIMH122943 to KES).

Motivation and Emotion

6. Footshock-Induced Changes in Social Behavior Are Mediated by Tac2- and CRH-Expressing Neurons in the dBNST

Presenting Author: Michael Conoscenti, University of Utah
Additional Author(s): Nicholas Poll[1]; Lorrin Malady[1]; Moriel Zelikowsky[1]
[1]University of Utah

The dorsal bed nucleus of the stria terminalis (dBNST) is an anatomically and functionally diverse brain region that mediates the behavioral and biological changes induced by a variety of stressors, including exposure to unpredictable footshock. Exposure to an acute stressor leads to widespread upregulation of corticotropin-releasing hormone (CRH), which in turn mediates stress-induced anxiety and aggression. In addition, we have found that social stress increases expression of tachykinin 2 (Tac2) in the dBNST, which is required for stress-induced persistent fear. Using in situ hybridization, we found that approximately 50% of Tac2-containing dBNST cells co-express CRH. However, the functional role of this unique overlapping population remains entirely unexplored. Here, we test the hypothesis that footshock-induced changes in social behavior are mediated by Tac2 and CRH co-expressing neurons in the dBNST (dBNSTTac2∩CRH). Tac2-Cre, CRH-Cre, or Tac2-Cre;CRH-Flp transgenic C57Bl6/N mice were exposed to an acute footshock stressor (FS; 10, 1mA shocks randomly distributed across a 60-minute session), or the context without shock, and later tested for an array of social behaviors, including aggression, social interaction, anti-social behavior, and non-social behaviors using the resident intruder assay. We define the contributions of Tac2+, CRH+, and Tac2∩CRH+ neurons in the dBNST toward stress-induced social changes using a variety of approaches including chemogenetic and optogenetic manipulations, in situ hybridization using RNAScope, and in vivo calcium imaging using microendoscopes. Specifically, we found that FS-induced aggression and/or social avoidance are reduced when Tac2 and CRH+ dBNST neurons are silenced. Conversely, unstressed mice exhibit aggression and/or social avoidance when these cells

are optogenetically stimulated. Finally, dBNSTTac2∩CRH seem preferentially active during a social interaction following exposure for footshock. Taken together, our findings illustrate a critical role for dBNSTTac2∩CRH neurons in social avoidance and aggression induced by stress.

Acknowledgement of funding sources: NIH Grant 1R01MH132822-01

7. The Role of L-type Calcium Channels on Drug and Mood-Related Behavioral Responses in Female Rats During Cocaine Abstinence

Presenting Author: Violet M Kimble, Yale University
Additional Author(s): Eric Nunes[1]; Nii Addy[1]
[1]Yale University

Voltage-gated L-type calcium channels (LTCCs) are crucial in mediating drug and mood-related behavioral responses in rodents. Prior work from our lab has shown that blocking LTCCs attenuates cue-induced cocaine seeking during abstinence by increasing dopamine (DA) release in the nucleus accumbens in male rats. Yet, the role of LTCC blockade on cue-induced cocaine seeking during abstinence in female rats is unknown. Female rats were trained to self-administer I.V. cocaine (~0.5mg/kg/inf), where they had access to active and inactive levers, with the active lever providing a cocaine infusion and audio-visual cue. In the cue-test phase, the active lever no longer yields reward but still provides the cue. Systemic administration of the LTCC blocker isradipine (0.1mg/kg, 0.4mg/kg, 1.2mg/kg, I.P.) attenuated cue-induced cocaine seeking behavior in females, but not for sucrose taking or seeking. We next sought to determine if cocaine-abstinence induces pro-depressive effects on social interaction. Isradipine was administered 15 minutes before a 5-minute social interaction test, during which we measured time investigating, time immobile, and the number of investigations within the 50 mm radius of the stranger rat. The test involved a stranger rat in a three-chamber box with wire cup-like containers, allowing visual interaction. Our preliminary data thus far is not indicating a behavioral effect of cocaine abstinence on social interaction, yet data collection is still ongoing. Our hypothesis is that female rats will show a reduction in social interaction during cocaine abstinence, which will be attenuate by LTCC blockade. This study contributes to our understanding of LTCCs' involvement in natural and drug reward processing and the potential gender-specific mechanisms associated with these processes. Further exploration of LTCCs in substance abuse populations may shed light on the etiology and symptomatology of mood disorders, thus fostering the development of more effective interventions and treatment strategies.

Acknowledgement of funding sources: This work was supported by National Institutes of Health (NIH) grants R01MH108663-03 (EJN, and NAA) and T32NS041228-22 (VMK).

8. Encoding of External Threat and Risk Taking in the Acute Stress Response

Presenting Author: Victoria Sayo Turner, University of California, San Francisco
Additional Author(s): Rachel O O'Sullivan[1]; Shazreh T Hassan[1]; Mazen A Kheirbek[1]
[1]University of California, San Francisco

Stressful experiences can lead to classic anxiety behaviors like avoidance. However, it is not well understood how stressful experiences and anxiety-related states are encoded across the brain. Here, we investigate how neural representations of internal state and the surroundings differ during approach and avoidance of risk. The ventral hippocampus (vHPC) has long been thought to be an inhibitory regulator of the corticosteroid stress response, part of a negative feedback loop that controls the size of stress responses. Downstream, the hypothalamic CRH-expressing neurons are

known to be the gatekeeper of the cortisol stress response, but recent studies also implicate a non-hormonal role for these cells in anxiety-related behavior. What is still unknown is how these two regions respond in the same animals to specific stressful cues, and how the ventral hippocampus may be involved in regulating CRH-related avoidance behaviors. To test how both regions respond to situations of uncontrollable stress (footshock) and controllable stress (elevated plus maze), we recorded simultaneously in vHPC and hypothalamic CRH neurons using calcium imaging. Our results show that vHPC and hypothalamic CRH neuron responses to stress-related stimuli are specific to the stimuli or behavioral motif, and do not follow simple one-to-one correlations. This suggests that the historical model of vHPC modulation of the stress response is incomplete without accounting for other inputs to the hypothalamic CRH neurons. To investigate the difference in these two regions during risk taking, we analyzed neural responses during exploration of exposed and sheltered areas of the elevated plus maze. Recording in this setting allowed us to observe neural correlates of self-regulated stressful experiences. Using advanced behavioral and trajectory analysis, we observed that vHPC tends to track the anxiety-related information of an animal's surroundings, while the hypothalamic CRH cells show specific signatures when animals retract from exposed areas versus when they continue further exploration. Together, these findings suggest a model of the vHPC as an internal gauge of risk in the surroundings, while PVN may serve to gate specific motivational behaviors like approach or avoidance. Understanding the contributions of multiple brain areas to anxiety- and fear-related states will help us better predict the mechanisms that become dysregulated in psychiatric disease.

Acknowledgement of funding sources: M.A.K. was supported by the National Institute of Mental Health (R01 MH108623, R01 MH111754 and R01 MH117961); the National Institute on Deafness and Other Communication Disorders (R01 DC019813); a One Mind Rising Star Award; a research grant from the Human Frontier Science Program (RGY0072/2019); the Esther A. and Joseph Klingenstein Fund; the Pew Charitable Trusts; the McKnight Memory and Cognitive Disorders Award; and the Ray and Dagmar Dolby Family Fund.

Neural Excitability, Synapses, and Glia

9. Possible Interactions Between O-GlcNAcylation and Phosphorylation in Modulating Hippocampal GABAergic Transmission

Presenting Author: Shekinah Phillips, University of Alabama at Birmingham
Additional Author(s): Lori McMahon, Medical University of South Carolina

Crosstalk between O-GlcNAcylation and phosphorylation affects the regulation of various proteins involved in cellular function such as transcription, translation, and transportation. Separately, serine phosphorylation and O-GlcNAcylation modulate GABA-gated currents, yet no studies have examined how the interplay between both serine modifications affect $GABA_AR$ function and the strength of GABAergic transmission. Phosphorylation of Ser 409 on the β 1 subunit by protein kinase A (PKA) decreases $GABA_AR$ currents, while PKA dependent phosphorylation of Ser 408 and Ser 409 on the β3 subunit increases $GABA_AR$ currents in HEK293 cells. Studies from our lab have recently shown that an acute increase in O-GlcNAcylation significantly decreases the amplitude of synaptic inhibitory postsynaptic currents in CA1 pyramidal cells and dentate granule cells. While it is unknown which serines undergo O-GlcNAcylation on $GABA_AR$ β subunits, it is possible that phosphorylation and O-GlcNAcylation occur on the same serines thereby competing with one another, or may occur on

separate serines, which may or may not interact in a functional manner. Because phosphorylation and O-GlcNAcylation have potent effects on $GABA_AR$ function, it is important to determine how O-GlcNAcylation effects $GABA_AR$ function simultaneously with serine phosphorylation, and if prior phosphorylation prevents or augments the effect of O-GlcNAcylation. To test this, we used whole-cell recordings of evoked IPSCs from CA1 pyramidal cells and dentate granule cells in acute slices from 3-5 week old male and female rats and bath applied the PKA activator, forskolin (50µM), either before (CA1, n= 11; dentate n=6) or after (CA1, n= 11; dentate n=10) bath application of glucosamine and the OGA inhibitor, thiamet-G to increase O-GlcNAcylation. The data show a possible interaction such that a prior increase in O-GlcNAcylation, which depresses evoked IPSC amplitude, elicits a forskolin-dependent increase in IPSC amplitude. On the other hand, forskolin elicits a depression of the IPSC amplitude in the absence of a prior increase in O-GlcNAcylation. These data suggest the polarity of a PKA-dependent modulation of $GABA_AR$ function is dictated by the presence or absence of a co-occurring O-GlcNAc modification. Whether serine phosphorylation and O-GlcNAcylation are competing for the same site or different sites, and how these post-translational modifications are interacting at the level of the $GABA_AR$ requires additional investigation.

Acknowledgement of funding sources: The research was funded by NIH NINDS 5 R21 NS111945-02.

10. Single Cell Transcriptome Profiling of Human and Mouse Brain Reveals Enrichment of Alcohol-Related Immune Genes in Astrocytes and Microglia

Presenting Author: Nihal Salem, The University of Texas at Austin

Additional Author(s): Anna Warden[1]; Eric Brenner[1]; Alison Goate[2]; Amanda Roberts[3]; Marisa Roberto[3]; Dayne Mayfield[1]

[1]The University of Texas at Austin; [2]Icahn School of Medicine at Mt Sinai; [3]The Scripps Research Institute

Transcriptomic studies have identified dysregulated signatures in post-mortem brain samples from alcohol-dependent individuals as well as animal models of alcohol consumption and dependence. Identification of cell-type specific transcriptomic changes in response to chronic alcohol use will improve our understanding of mechanisms mediating escalation of alcohol use and consequently refine targetable mechanisms to which therapeutics can be developed. Our goal was to identify cross-species and cell-type specific transcriptome changes associated with alcohol use. We utilized single nuclei RNA sequencing (snRNA-seq; 10x Genomics) to examine the dorsolateral prefrontal cortex from AUD compared to matched control cases (New South Wales Brain Tissue Resource Centre), and from C57Bl6/J mice exposed to a chronic intermittent ethanol (CIE) paradigm. For the CIE mouse study, animals were exposed to 16 hours of ethanol vapor/day (or air as control) for 4 days followed by 72 hours of forced abstinence then with 2 bottle choice for 2 hours; this paradigm was repeated for four cycles followed by animal sacrificing and brain harvesting. snRNA-seq was performed on micro-punches obtained from medial prefrontal cortex. We identified differentially expressed genes and their enriched pathways in each cell type. In both human and mouse, glial cells including oligodendrocytes, astrocytes, and microglia were the most susceptible to alcohol. We identified overlapping expression changes in microglia and astrocytes between human and mouse brains. Cell-type enrichment studies in alcohol-treated mice showed overlapping expression changes in microglia and astrocytes that were associated with neuroimmune effector cell types like that found in human brain. These cross-species expression studies indicated that glial cells that are critical for the escalated voluntary alcohol in mouse models may also be important regulators of excessive alcohol drinking associated with alcohol dependence in humans. Cross species multiomics approaches used to delineate cell type-specific differences in brain function could greatly advance

knowledge molecular mechanisms associated with AUD and identify translationally relevant drug targets.

Acknowledgement of funding sources: This work was supported by NIH/NIAAA grants R01AA012404 (rdm), U01AA020926 (rdm), and K00AA029955 (nas).

Techniques

11. Dual Imaging of Cytosolic and Endoplasmic Reticular Ca2+ Dynamics in Dendrites In Vivo

Presenting Author: Justin O'Hare, Columbia University
Additional Author(s): Jamie Wang[1]; Margele Shala[2]; Franck Polleux[3]; Attila Losonczy[3]
[1]Duke University; [2]Cambridge University; [3]Columbia University

Synaptic plasticity is thought to support behavioral adaptation by updating how information propagates though neuronal circuits, but this process remains enigmatic due to its many levels of complexity. For instance, multiple circuits transmitting distinct streams of information often target anatomically- and functionally-specialized regions of a neuron's dendritic arbor. This combinatorial complexity is likely key to a neuron's ability to form complex receptive fields that faithfully represent the dynamic environments in which we live. Intracellular Ca2+ release (ICR) from endoplasmic reticulum (ER) was recently shown to operate at this circuit-subcellular intersection, acting preferentially in apical dendrites of hippocampal CA1 pyramidal neurons (CA1PNs) to shape the experience-dependent formation of spatial receptive fields commonly referred to a 'place fields'. However, it remains an open question as to when ICR is engaged: ER-resident Ca2+ has never been directly monitored in mammalian neurons in vivo. To address this foundational gap in knowledge, we simultaneously imaged ER Ca2+ dynamics and ongoing neuronal activity in radial oblique dendrites of individual CA1PNs as mice learned to navigate a series of novel virtual environments. We are now assessing ER Ca2+ dynamics to determine when ICR occurs relative to ongoing somatic and dendritic activity, place field formation, and animal behavior.

Acknowledgement of funding sources: This work was supported by the National Institutes of Health (grants R01MH100631, R01NS094668, and U19NS104590 to A.L.; grants R01NS067557 and R01NS094668 to F.P.; grants F32MH118716 and K99NS127815 to J.O.; the Zegar Family Foundation (A.L.); and the Foundation Roger De Spoelberch (F.P.).

Poster Room B (3:00 – 4:00 pm EDT)

Development

12. Genetic Regulation of Dopaminergic Neuron Regeneration in the Planarian Nervous System
Presenting Author: Kendall Clay, University of Georgia
Additional Author(s): Taylor Medlock-Lanier[1]; Rachel Roberts-Galbraith[1]
[1]University of Georgia

Although the ability to regrow tissue after injury exists in many species across the animal kingdom, the ability to regenerate a brain is rare. One organism that can regenerate a brain de novo is the planarian. Planarians are flatworms with a robust regenerative capability and a complex nervous system consisting of a brain, ventral nerve cords, and a peripheral nervous system. Our lab works toward uncovering different facets of brain regeneration in the asexual planarian Schmidtea mediterranea to elucidate how robust regeneration in nature is accomplished. Brains are highly complex tissues, so to parse out the mechanisms underlying neural regeneration, we have begun with individual neuronal cell types. This project focuses on elucidating the genetic mechanisms behind the regeneration of dopaminergic neurons.

From available transcriptomic data (Fincher, et al., 2018), we compiled a list of seventy-three candidate genes with expression enriched in cells expressing high levels of tyrosine hydroxylase, which encodes an enzyme critical in dopamine synthesis. Through in situ hybridization, we determined that at least 85% of the genes of interest are expressed in the nervous system. Next, we used RNA interference to knock down candidate genes. Using in situ hybridization with a TH probe, we assessed the impact of each gene knockdown on dopaminergic neuron regeneration.

Knocking down two genes, amyloid-beta protein precursor or lim domain only 3, significantly decreased the number of TH+ cells in regenerating brains (52.89 and 50.62% of control average, respectively, $p<0.05$). We are currently assessing whether these genes play roles in the regeneration of the brain as a whole and whether they impact other cell types, including GABAergic neurons and glial cells. Our findings will help us understand the innerworkings of planarian brain regeneration, which may lead to future applicable interventions for human health.

Acknowledgement of funding sources: UGA Neuroscience Program, UGA Office of Research, Alfred P. Sloan Foundation, McKnight Foundation, ARCS Foundation, NIH Genetics T32 (1T32GM142623), NINDS (5R25NS107179-02 and 5R01NS128096-02).

13. A Choroid Plexus Apocrine Secretion Mechanism Alters Fetal Cerebrospinal Fluid Proteome and Instructs Cortical Development
Presenting Author: Ya'el Courtney, Harvard Medical School
Additional Author(s): Elizabeth Yimer[1]; Neil Dani[2]; Frederick Shipley[2]; Maria Lehtinen[2]
[1]Harvard Medical School; [2]Boston Children's Hospital

During embryonic brain development, cerebral cortical neurons form from neural progenitor cells that divide along the brain's ventricles and in contact with cerebrospinal fluid (CSF). CSF is produced largely by the choroid plexus (ChP), a ciliated epithelial tissue in each ventricle whose cells secrete CSF and its contents, including instructive factors like IGF-2. The ChP also serves as a blood-CSF

barrier, protecting the brain from infection and inflammation. Its cultivation of proper CSF composition is crucial to healthy development. Indeed, CSF aberrations are increasingly implicated in neurodevelopmental disorders including autism spectrum disorder (ASD), hydrocephalus, and schizophrenia.

Despite its lifelong role in titrating CSF contents, ChP secretory machinery is poorly understood. Advances in imaging approaches enabled our discovery that, in addition to vesicular exocytosis, the ChP displays a high-capacity regulated exocytosis mechanism called apocrine secretion. We developed a toolbox using highly expressed G-protein coupled serotonin receptor 5HT2C to evoke and interrogate apocrine secretion using standard biochemical approaches, multi-photon imaging, expansion microscopy, and electron microscopy. We demonstrate this mechanism's functionality during embryonic development, confirm that these events alter CSF composition in vivo, and report findings on their contents. We further provide evidence that this altering this secretion process negatively affects cortical brain development. This mechanism may be sensitive to stressors like maternal inflammation and environmental teratogens, with resulting perturbations leading to impaired brain development.

Acknowledgement of funding sources: Y. Courtney is a Howard Hughes Medical Institute Gilliam Fellow. This work is supported in part by the New York Stem Cell Foundation and NIH grant R01 NS088566.

14. Investigating Functional Effects of scn1lab Mutation in a Zebrafish Genetic Model of ASD

Presenting Author: April Pruitt, Yale University
Additional Author(s): Hellen Weinschutz Mendes[1]; David Jin[1]; Tianying Chen[1]; Uma Neelakantan[1]; Ellen Hoffman[1]
[1]Yale University

Background: Previous exome-sequencing studies have identified over one hundred high confidence risk genes contributing to autism spectrum disorder (ASD). Variants in the genes SCN1A and SCN2A, which encode the voltage gated sodium channels Nav1.1 and Nav1.2, respectively, have been associated with Dravet Syndrome, infantile-onset epilepsy, intellectual disability, and ASD. Here we investigate functional effects of SCN1A/2A mutations in larval zebrafish mutants of scn1lab, an ortholog of the ASD risk genes SCN1A and SCN2A.

Methods: We evaluated scn1lab mutation effects on three levels: behavior, circuit activity, and transcriptional profiles. First, we characterized behavior of scn1lab-mutant larval zebrafish using two paradigms: rest-wake assay and visual-startle response assay. To assess pan-neuronal activity, we then conducted whole-brain activity mapping and measured differences in brain volume. To understand how scn1lab mutations affect global brain transcriptional profiles and biological pathways, we performed RNA-seq and pathway analysis. To understand how early exposure to estrogenic compounds impacts behavior and neuronal development, we then repeated these assays in the presence of beta-estradiol 17-cypionate.

Results: scn1lab-mutant larvae show reduced daytime activity, nighttime hyperactivity, and hyperactive startle response to light. scn1lab-mutants also show decreased brain activity, decreased brain volume, and a deficit in GABAergic neuronal populations. We found dysregulated synaptic, GABAergic, and glutamatergic pathways in scn1lab-mutants. Estrogenic compounds ameliorated

hypersensitivity to light-stimulus, reduced PTZ-induced seizures, and led to alterations in global transcriptional profiles.

Conclusion and Discussion: These data show that scn1lab mutants exhibit disruptions in visuomotor, rest-wake, and excitatory-inhibitory circuitry, leading to alterations in visual-startle responses and rest-wake activity. In future studies, we aim to assess associations between zebrafish and human SCN2A RNA-seq datasets by directly mapping across species and examining correlations between global t-statistic and FDR of differentially expressed genes.

Acknowledgement of funding sources: EH was supported by NIH R01MH116002, Binational Science Foundation, Kavli Foundation, National Genetics Foundation, Simons Foundation, Spector Fund, and the Swebilius Foundation. AP was supported by training grant T32GM007499, Kavli Institute for Neuroscience Scholar Fellowship, and Autism Science Foundation Predoctoral Fellowship.

Neurodegenerative Disorders and Injury

15. Basal Ganglia Disruption in a Mouse Model of Progressive Supranuclear Palsy

Presenting Author: Rose B Creed, University of California, San Francisco
Additional Author(s): Alexandra B Nelson, University of California, San Francisco

Progressive Supranuclear Palsy (PSP) is a neurodegenerative disease that affects movement, behavior, and cognition. Due to an overlap in symptoms with Parkinson's Disease, PSP is considered an atypical parkinsonian disorder, but PSP patients have distinct clinical and pathological features. Clinically, PSP patients have early gait abnormalities, frequent falls, gaze palsy (slowed saccadic eye movements), and tend not to respond to dopamine replacement therapy. Pathologically, aggregated Tau protein (rather than alpha synuclein) accumulates in the brain of PSP patients. As in other neurodegenerative disorders, a combination of cellular dysfunction and cell loss is believed to drive disease symptoms. However, a lack of animal models for PSP has hindered investigation of the causal links between neuropathology, cellular and circuit dysfunction, and symptoms. Here we have utilized the Thy1-hTau.P301S mouse model of tauopathy, as well as an AAV-Tau overexpressing model to determine whether Tau pathology (1) is sufficient to recapitulate key PSP phenotypes in mice and (2) results in aberrant neural activity in motor control nuclei. We find Tau transgenic mice have impaired motor performance in both the open field and accelerating rotarod test. In addition, we detected gait abnormalities in Tau transgenic mice. Similar to the human condition, we find that Tau transgenic mice have tau pathology in several basal ganglia nuclei, a group of subcortical nuclei involved in motor control, which were recently identified as an initial site of Tau pathology in PSP patients. We have also been using head-fixed and freely moving eye movement measurements to determine whether Tau pathology in basal ganglia is sufficient to cause impairments in eye movement kinematics. Overall these findings highlight the utility of Tau overexpression to modelling PSP and provides a platform to investigate the changes in neural structure and function that drive the movement abnormalities seen in disease.

Acknowledgement of funding sources: NIH/NINDS K00; Burroughs Wellcome Fund Postdoctoral Diversity Enrichment Award

16. VEGF- A Produced by Neurons Contributes to Cerebral Malaria Pathology

Presenting Author: Cori Fain, Mayo Clinic
Additional Author(s): Sabhya Rana[1]; Fang Jin[1]; Meredith Lilley[1]; Mark Maynes[1]; Gary Sieck[1]; Aaron Johnson[1]
[1]Mayo Clinic

Cerebral malaria (CM) is a severe complication of Plasmodium falciparum infection. Biomarkers of disease severity include blood-brain-barrier (BBB) permeability, severe edema, and vascular endothelial growth factor (VEGF) upregulation in post-mortem brain tissue. In previous work using T1 and T2-weighted MRI we observed the recapitulation of vascular permeability in the Plasmodium berghei ANKA murine model of experimental cerebral malaria (ECM). Vascular permeability was noted to be regional, occurring in olfactory bulb, hippocampus and brainstem regions on day 6-8 of PbA infection. In this work we determined through RNAscope in situ hybridization assay that at 6 dpi, areas of known permeability in the brain coincide with upregulation of VEGF mRNA transcripts. We next sought to identify the main cell-types responsible for this upregulation, with focus on brainstem pathology. Here we report that mature NeuN+ neurons in brainstem display significantly increased production of VEGF mRNA transcripts during ECM. This upregulation is observed in neurons near regions of tight junction protein disruption. Neurons positive for VEGF protein were often seen being engulfed by microglia and/or astrocytes in late ECM; indicating a possible role for VEGF in neuronal pathology. In perforin deficient mice, VEGF mRNA did not increase in neurons day 6 of PbA infection, indicating a possible role for CD8 T cells in upregulation of VEGF. Using a novel inducible mouse model, we induced neuron-specific ablation of VEGF during PbA infection, which resulted in decreased proportion of CD8 T cell brain infiltration. This in turn, prevented disruption of tight junction proteins and preserved ambulatory behaviors day 6 post infection. This resulted in overall extended survival. In conclusion, these findings highlight a potential role for CD8 T cells in promoting BBB disruption through neuronal-VEGF upregulation, identifying this cytokine as one that could be targeted therapeutically to treat human CM.

Acknowledgement of funding sources: This work was supported by Ruth L. Kirschstein National Research Service Award Individual Predoctoral Fellowship F31 1F31NS116924-01, NIH R01 NS103212, and the Mayo Clinic Ph.D. Training Grant in Basic Immunology (T32 AI07425-24).

17. Neuroprotective BCLw Potentiates ATP Evoked Calcium Transients in Murine Fibroblasts

Presenting Author: Joy Franco, Harvard Medical School; Dana-Farber Cancer Institute
Additional Author(s): Taylor Copeland[1]; Sophia Tang[2]; Maria F Pazyra-Murphy [1,2] ; Gregory H Bird[2]; Loren D. Walensky[2]; Lisa V. Goodrich[1,2]; Rosalind A. Segal[1,2]
[1]Harvard Medical School; [2]Dana-Farber Cancer Institute

Sensory neurons, such as the spiral ganglion neurons (SGNs) that transmit sound information from the cochlea to the brainstem, are vulnerable to degeneration with increasing age. This degeneration manifests as a progressive dying back of the afferent terminals from their sensory organs and decreased sensory perception. The mechanisms underlying this age-related neuropathy are not yet defined, but the Bclw-IP3R1 signaling axis has been identified as a potential contributor. Mutation of the pro-survival Bcl2 family member Bclw causes an age-dependent decline in thermosensation, a reduction in intra-epidermal nerve fibers, and increased vulnerability to paclitaxel-induced

neuropathy. In vitro studies demonstrated that treatment of cultured dorsal root ganglion neurons with a Bclw-BH4 mimetic prevents paclitaxel induced axonal degeneration, suggesting the BH4 domain of Bclw may also confer protection against age-related damage. To determine whether BCLw contributes to calcium regulation and axonal survival via interactions with IP3R, we employed an in vitro system for measuring intracellular calcium release initiated by IP3R. Using the ratiometric calcium indicator, FuraRed, to assess calcium release in immortalized murine embryonic fibroblasts (MEFs) from Bclw-/- and wild type (WT) mice. Timelapse images of MEFs from both genotypes in response to stimulation with ATP—a purinergic receptor agonist that induces formation of the diffusible ligand IP3 and IP3R stimulation. We found that only ~40% of Bclw-/- MEF show detectable calcium release to ATP, whereas ~90% of WT MEFs respond to the same stimuli. We then asked if pre-treatment with a BH4 mimetic would rescue the sensitivity of the KO MEFs. We found that Bclw-/- MEFs treated with the BH4 mimetic were ~30% more responsive than vehicle treated cells. These results are in contrast to what has been observed for other Bcl2 pro-survival family members. We are actively investigating whether this mechanism may have therapeutic potential for neurodegeneration including age-related sensorineural hearing loss.

Acknowledgement of funding sources: This work is supported by award number 8K00AG078230-03 from the National Insitute on Aging through the NIH NINDS DSPAN program to JF and from a Blavatnik Sensory Disorders Grant to LVG.

18. Non-essential Role for Cx3cr1-Expressing EPH Receptor A4 in a Murine Model of TBI

Presenting Author: Jatia Mills, Virginia Tech
Additional Author(s): Eman Solima[1]; Jing Ju[1]; Alexandra Kaloss[1]; Erwin Kristobal Gudenschwager[1]; Nathalie Groot[1]; Colin Kelly[1]; Elizabeth Kowalski[1]; Mohamed Elhassanny[1]; Michael Chen[1]; Xia Wang[1]; Michelle Theus
[1]Virginia Tech

Adult Erythropoietin-producing human hepatocellular (Eph) receptors contribute significantly to central nervous system injury. Our findings demonstrated that Cx3cr1-expressing cells within the perilesional cortex showed increased levels of EphA4 after induction of controlled cortical impact (CCI) injury in mice. Cx3cr1 is a fractalkine receptor, commonly expressed on resident microglial and peripheral-derived macrophage (PDM) cells. The aim of this study is to identify the role of microglial-specific EphA4 in CCI-induced damage. Cx3cr1CreERT2/EYFP knock-in/knock-out mice expressing EYFP in Cx3cr1+ cells were used to evaluate microglia in EphA4-deficient mice following 1-month tamoxifen injections. CCI-Injured wild-type (WT) Cx3cr1CreERT2/EYFP/EphA4+/+ mice displayed increased EphA4 expression on the EYFP-positive cx3cr1 cells within the peri-lesion. Immunohistochemical applications were further used to differentiate between the peripheral-derived macrophage and resident microglia using anti-Ccr2, which selectively labeled PDMs and not microglia. We then exploited GFP bone marrow chimeric mice to discriminate EphA4 expression on microglia (TMEM119+/GFP-) versus PDMs (GFP+) following CCI. Finally, the use of Cx3cr1-CreERT2/EYFP/EphA4f/f (KO) mice, which show no detectable transcript for EphA4 in microglia only, demonstrated no discernible difference in lesion volume or blood brain barrier (BBB) disruption when compared to the WT mice. These findings illustrate that although EphA4 is upregulated on cortical microglia after TBI, it plays a nonessential role in acute response following TBI.

Acknowledgement of funding sources: We recognize The Center for Engineered Health for grant support, and Mellissa Markus for flow cytometry support. This work was supported by the National

Institute of Neurological Disorders and Stroke of the National Institutes of Health, NS096281, NS119540, and NS121103 (MHT).

19. Sexually Dimorphic Amygdala Dysfunction in a Mouse Model of Global Cerebral Ischemia

Presenting Author: Jose Vigil, University of Colorado Anschutz Medical Campus
Additional Author(s): Erika Tiemeier[1]; Nicholas E Chalmers[1]; Paco S Herson[2]; Nidia Quillinan[1]
[1]University of Colorado Anschutz Medical Campus; [2]Ohio State University

Modern medical advances have increased the odds of surviving an ischemic event such as cardiac arrest. With more people surviving and recovering from these ischemic insults, it is increasingly apparent that survivors experience long-term effects on brain function. We have previously identified cognitive dysfunction in a mouse model of global cerebral ischemia (GCI) which is attributed to hippocampal neurodegeneration and impaired hippocampal plasticity. However, no study has attempted to identify amygdala dysfunction after GCI, despite clinical evidence of emotional dysfunction. Therefore, it is important to identify the effect that GCI has on the amygdala, the emotional center of the brain. I hypothesize GCI induces dysfunction of L-type calcium channels (LTCCs) within the basolateral amygdala (BLA) thereby contributing to deficits in amygdala-dependent behavior and LTP in male mice. Experimental GCI was induced in adult (8-12 week) mice via cardiac arrest and subsequent cardiopulmonary resuscitation (CA/CPR). CA was induced for 8-minutes and subsequent resuscitation by epinephrine injection, ventilation, and mild chest compressions. 7-days post GCI, the amygdala-dependent delay fear conditioning paradigm was used to assess amygdala-dependent learning and memory. Synaptic plasticity was evaluated by performing LTP recordings in the BLA, and LTCC function was assessed using whole-cell voltage clamp recordings. Behavioral testing revealed that only male mice are diminished in their ability to form associative memories. Similarly, plasticity of the cortical inputs to the BLA are impaired only in males. Interestingly intra-amygdala recordings revealed no disruption of LTP in this circuit and we observed no cell death within the BLA of either sex. Whole-cell LTCC mediated currents were minimally affected by GCI, however, additional 2-photon calcium imaging experiments will evaluate LTCC function at more distal synapses after GCI. These results support the role of the amygdala in cognitive-affective impairments after CA despite a lack of neuronal cell death in this brain region.

Acknowledgement of funding sources: Diversity Supplement NS046072; AHA Predoctoral Fellowship

---

Sensory Systems

19. Redundancy and OFF Responses Enable Contrast Enhancement in the Olfactory Bulb of Awake Mice

Presenting Author: Elizabeth Hanson Moss, Oregon Health and Science University
Additional Author(s): Delaram Pirhayatifard[1]; Doris Ling[2]; Jacob Reimer[3]; Baranidharan Raman[2]; Ankit Patel[1]; Benjamin Arenkiel[3]
[1]Rice University; [2]Washington University in St. Louis; [3]Baylor College of Medicine

Spatially stereotyped olfactory bulb (OB) activity has been extensively demonstrated when odors are presented under controlled conditions. When odors are presented with controlled respiration, under anesthesia, or as simple, monomolecular, low concentration odors, odor response maps are

reliable, sparse, and easily separable. However, OB responses to more complex or naturalistic odors, presented in the awake state, are denser, more variable from trial-to-trial, and more temporally complex. Thus, an important, outstanding question is how dense, largely overlapping odor representations in the OB are separated into distinct odor percepts. Towards understanding how odors are identified and discriminated from highly mixed OB activity patterns, we examine large-scale population activity in the OB in response to odors using two-photon mesoscopic imaging of bilateral OBs through cranial windows in awake mice. In Thy1-GCaMP6F mice, which express a fluorescent calcium indicator in mitral cells and their glomerular apical dendrites, we directly monitor odor responses from OB glomeruli. We then use machine learning to define linear classifiers that identify odor presentations from glomerular response patterns. A linear classifier trained on subsets of odor responses is reproducibly able to decode odor identity from withheld responses, indicating that odor responses are largely reliable and stable over days, in agreement with much previous work. However, an analysis of the value added to the classifier by individual glomeruli shows that a larger portion of glomeruli contribute to the odor representation than would be suggested by rigidly stereotyped OSN inputs alone. These results suggest that odor codes in the glomerular layer are more distributed than previously appreciated. Intriguingly, a more distributed odor code in the OB may help enable robust odor encoding in the face of trial-to-trial variability, behavioral state variability, awake-state feedback, and neuromodulation.

Acknowledgement of funding sources: NIH NIDCD K99DC019505, NIH NINDS UF1NS111692

21. Feature-Based Attention Modulates Spatial Frequency Processing in Early Human Visual Cortex

Presenting Author: Luis D Ramirez, Boston University
Additional Author(s): Feiyi Wang[1]; Sam Ling[2]
[1]Tufts University; [2]Boston University

With limited biological resources, the brain utilizes selective mechanisms like attention to prioritize relevant sensory information for processing. A signature of selective attention is an increase in gain for neurons encoding the attended feature. In the taxonomy of attention, feature-based attention distinguishes itself from spatial attention in that it increases gain for neurons in early visual cortex that are tuned to the attended feature, even when their spatial receptive fields exist outside the locus of attention. Spatial frequency is a fundamental feature encoded by the human visual system. How the global effects of feature-based attention alter spatial frequency tuning for neural populations in early visual cortex remains unclear. Here, we leverage a model-based fMRI approach to estimate voxel-wise population spatial frequency tuning (pSFT) in V1–V3 while subjects were cued to perform a letter detection task at disparate spatial frequencies (0.5 or 2 cpd). We find that pSFT shift with attention in the ipsilateral hemisphere, towards the attended SF, with more distal populations from the attended SF experiencing stronger changes in pSFT.

Acknowledgement of funding sources: NIH R01EY02163 to S. Ling; NIH F99NS124144 to L. D. Ramirez

---

Registrants

Anusha Allawala, PhD
University of California, San Francisco
D-SPAN F99/K00
anusha.baker@gmail.com
Twitter: @neuronush

Hector Arciniega, PhD
NYU Grossman School of Medicine
D-SPAN F99/K00
hector.arciniega@nyulangone.org
Twitter: @arciniegaphd

Sikoya Ashburn, PhD
University of North Carolina at Chapel Hill
s.ashburn.phd@unc.edu
D-SPAN F99/K00, NSP Alum
Twitter: @sikotree
LinkedIn:
https://www.linkedin.com/in/drsashburn/

Antonio Aubry, PhD
Icahn School of Medicine, Mt Sinai
BRAIN K99/R00
antonio.aubry@mssm.edu
LinkedIn: https://www.linkedin.com/in/tony-aubry-591b125a

Ariel Batallan Burrowes, PhD
Center for Addiction and Mental Health
NSP Scholar
ariel.batallan@camh.ca
LinkedIn: https://www.linkedin.com/in/ariel-batall%C3%A1n-burrowes-348532233/

Almarely Berrios-Negron, MS
Ponce Health Sciences University
NSP Scholar, SPINES
aberrios20@stu.psm.edu
Twitter: @heyitsalma
LinkedIN:
https://www.linkedin.com/in/almarelyberrios

Julianna Brutman, PhD
University of Washington
D-SPAN F99/K00
jbrutman@uw.edu
Twitter: @jnbrutman
LinkedIn: https://www.linkedin.com/in/julianna-brutman/

Jillybeth Burgado, BS
University of California, San Francisco; Salk Institute for Biological Studies
ENDURE Alum, NSP Scholar
jburgado@salk.edu

Monica Carbajal, BA
University of Memphis
NSP Scholar
mscrbjal@memphis.edu

Gabriela Carrillo, PhD
Boston Children's Hospital; Harvard Medical School
D-SPAN F99/K00
gabriela.carrillo@childrens.harvard.edu
Twitter: @gabbylcarrillo

Jaime Castrellon, PhD
University of Pennsylvania
D-SPAN F99/K00
jaimejfc@sas.upenn.edu
Twitter: @jjfcastrellon
LinkedIn:
https://www.linkedin.com/in/jcastrellon/

Victoria Castro, PhD
The University of Texas at El Paso
D-SPAN F99/K00
vlcastro2@miners.utep.edu
Twitter: @vlcsci

Christian Cazares, PhD
Salk Institute for Biological Studies
D-SPAN F99/K00, NSP Scholar
ccazares@salk.edu
Twitter: @fleabrained

Vanessa Cerda, PhD
Vanderbilt University
D-SPAN F99/K00
vanessa.cerda.1@vanderbilt.edu

Noële Certain, PhD
Yale University
D-SPAN F99/K00
noele.certain@yale.edu
Twitter: @noelecertain
LinkedIn:
https://www.linkedin.com/in/noelecertain/

KendallClay, BS
University of Georgia
NSP Scholar
kendall.clay@uga.edu
Twitter: @kendallbclay
LinkedIn:
https://www.linkedin.com/in/kendallbclay/

Lorianna Colon, PhD
Children's Hospital of Philadelphia; University of Pennsylvania
NSP Scholar
colonlm@chop.edu

Michael Conoscenti, PhD
University of Utah
NSP Scholar
michael.conoscenti@neuro.utah.edu

Laura Cortes, PhD
University of California, Los Angeles
D-SPAN F99/K00
lauracortes@ucla.edu
Twitter: @frontalcortes

Ya'el Courtney, BS
Boston Children's Hospital, Harvard University
ENDURE Alum
yaelcourtey@g.harvard.edu
Twitter: @scienceyael
LinkedIn:
https://www.linkedin.com/in/yaelcourtney/

Rose Creed, PhD
University of California, San Francisco
D-SPAN F99/K00
rose.creed@ucsf.edu

Adelis Cruz, BS
Texas A&M University
D-SPAN F99/K00
acruz20@tamu.edu
Twittter: @AdelisCruz

John Del Rosario, PhD
Washington University in St. Louis
D-SPAN F99/K00
johnd@wustl.edu
Twitter: @johndr01

Tahra Eissa, PhD
University of Colorado, Boulder
BRAIN K99/R00
tahra.eissa@colorado.edu

Aziz Elbasheir, BA
Emory University
ENDURE Alum, Diversity Supplement
aelbas2@emory.edu
Twitter: @elbasheiraziz

Cori Fain, PhD
Mayo Clinic
NSP Scholar
fain.cori@mayo.edu
Twitter: @cori_fain
LinkedIn: https://www.linkedin.com/in/cori-fain-52679ab7/

Mitchell Farrell, PhD
Albert Einstein College of Medicine
D-SPAN F99/K00
mitchell.farrell@einsteinmed.edu
Twitter: @mitch__farrell

Brianna Fitzgerald, BS
University of Alabama at Birmingham
NSP Scholar
bfitzger@uab.edu
Twitter: @bfitz_sci
LinkedIn: https://www.linkedin.com/in/brianna-fitzgerald-049838179/

Joy Franco, PhD
Harvard Medical School
D-SPAN F99/K00
joya_franco@dfci.harvard.edu
Twitter: @engineering_joy
LinkedIn: https://www.linkedin.com/in/joy-franco-93b165140/

Olivia Gautier, BS
Stanford University
ENDURE Alum
ogautier@stanford.edu

Mark Gergues, MS
University of Carlifornia, San Francisco
D-SPAN F99/K00
mark.gergues@ucsf.edu
Twitter: @mmgergues

Magaly Guzman Sosa, BS
Purdue University
NSP Scholar
mguzmans@purdue.edu

Hala Haddad, BA
Brown University
ENDURE Alum
hala_haddad1@brown.edu
Twitter: @halahaddad15
LinkedIn: https://www.linkedin.com/in/hala-haddad1

Evan Hart, PhD
National Institute on Drug Abuse
BRAIN K99/R00
evan.hart@nih.gov

Shiva Hassanzadeh-Behbahani, PhD
Harvard Medical School
D-SPAN F99/K00
sh1376@georgetown.edu
LinkedIn:
https://www.linkedin.com/in/shivahassanzadeh

Martina Hernandez, BS
Virginia Commonwealth University
D-SPAN F99/K00
hernandezml@vcu.edu
Twitter: @neuraldrama

Margo Heston, BS
University of Wisconsin-Madison
D-SPAN F99/K00
mheston@wisc.edu
Twitter: @margoheston
LinkedIn: https://www.linkedin.com/in/margo-heston-2ab1545a

Ashley Holloway, BS
Northwestern University
D-SPAN F99/K00
ashleyholloway2023@u.northwestern.edu

Victoria Honnell, MS
St. Jude Children's Research Hospital
D-SPAN F99/K00
victoria.honnell@stjude.org
Twitter: @victoriahonnell

Naznin Jahan, BA
University of California, San Francisco
D-SPAN F99/K00
naznin.jahan@ucsf.edu

Maanasa Jayachandran, PhD
National Institute of Neurological Disorders and Stroke
D-SPAN F99/K00
maanasa.jayachandran@nih.gov
Twitter: @maanasa_jay
LinkedIn: https://www.linkedin.com/in/maanasa-n-jayachandran-phd-421579257/

Julia Jones, BS
The Scripps Research Institute
D-SPAN F99/K00
jjones@scripps.edu
LinkedIN: https://www.linkedin.com/in/julia-jones-16810a84/

Megan Kelley, MS
Yale School of Medicine
D-SPAN F99/K00
megan.kelley@yale.edu

Violet Kimble, BS
Yale University
NSP Scholar
violet.kimble@yale.edu
Twitter: @violet_kimble
LinkedIn: https://www.linkedin.com/in/violet-k/

Dennisha King, MS
University of Rochester
NSP Scholar
dennisha_king@urmc.rochester.edu

Omar Koita, BA
Vollum Institute
D-SPAN F99/K00
koita1@ohsu.edu

Tony Larkin, PhD
University of Michigan
BRAIN K99/R00
telark@umich.edu
Twitter: @telark

Brandon Logeman, PhD
Harvard University
BRAIN K99/R00
blogeman@fas.harvard.edu
Twitter: @brandon_logeman

Esteban Lucero, BS
University of Colorado Anschutz Medical Campus
ENDURE Alum, D-SPAN F99/K00
esteban.lucero@cuanschutz.edu
Twitter: @estebanmlucero

Gabriela Manzano, PhD
Weill Cornell Medicine
D-SPAN F99/K00
gmn4001@med.cornell.edu
Twitter: @gabamanzano

Angelica M. Minier-Toribio, BA
Icahn School of Medicine at Mount Sinai
D-SPAN F99/K00
angelica.minier-toribio@icahn.mssm.edu
Twitter: @minierangelica
LinkedIN: http://linkedin.com/in/angelica-minier

Courtney McDermott, BA
Rutgers University
D-SPAN F99/K00
courtney.mcdermott@rutgers.edu
Twitter: @crmcdermott
LinkedIN: https://www.linkedin.com/in/courtney-mcdermott-ab8a9b100/

Elizabeth Medina, BA
Washington State University
NSP Scholar
elizabeth.medina3@wsu.edu

Jatia Mills, BS
Virginia Tech
NSP Scholar
jatiam@vt.edu
Twitter: @drmills24
LinkedIn: http://linkedin.com/in/jatiamills

Shannin Moody, PhD
Louisiana State University Health Sciences Center
D-SPAN F99/K00
smood1@lsuhsc.edu

Elizabeth Moss, PhD
Baylor College of Medicine
BRAIN K99/R00
elhanson@bcm.edu
Twitter: @indeeditsliz

Maria Muhammad, BS
University of Mississippi Medical Center
D-SPAN F99/K00
mamuhammad101@gmail.com

David Narvaiz, BS
Baylor University
D-SPAN F99/K00
david_narvaiz1@baylor.edu

Alesa Netzley, BS
Michigan State University
NSP Scholar, D-SPAN F99/K00
ahnetzley@gmail.com

Stephanie Noble, PhD
Yale University
BRAIN K99/R00
stephanie.noble@yale.edu
Twitter: @sneuroble
LinkedIn:
http://linkedin.com/in/sneuroble

Kavin Nunez, PhD
NYU Langone Neuroscience Institute
D-SPAN F99/K00
kavin.nunez@nyulangone.org

Justin O'Hare, PhD
Columbia University
BRAIN K99/R00
jo2546@columbia.edu
Twitter: @justinkohare
LinkedIn: https://www.linkedin.com/in/justin-ohare/

Nathaniel Parsons, BS
Medical University of South Carolina
D-SPAN F99/K00
parsonna@musc.edu
LinkedIn: http://linkedin.com/in/nathaniel-parsons-0a02a818

Shekinah Phillips, BS
University of Alabama at Birmingham
ENDURE Alum, NSP Scholar, D-SPAN F99/K00
sphill@uab.edu
Twitter: @shekinahphilli3
LinkedIn: https://www.linkedin.com/in/shekinah-phillips-49801b80/

April Pruitt, BS
Yale University
NSP Scholar
april.pruitt@yale.edu
LinkedIn: www.linkedin.com/in/april-pruitt

Lilyana Quigley, PhD
The University of Texas at Southwestern Medical Center
D-SPAN F99/K00
lilyana.quigley@utsouthwestern.edu
Twitter: @lilyanadream
LinkedIn: http://linkedin.com/in/lilyanaquigley

Mónica Quiñones-Frías, PhD
Brandeis University
ENDURE Alum, Diversity supplement
mcqf@brandeis.edu

Luis Ramirez, BS
Boston University
ENDURE Alum, D-SPAN F99/K00
luisdr@bu.edu
LinkedIn:
https://www.linkedin.com/in/luisdramirez95/

Meghan Ramirez, BS
University of Nebraska Medical Center
Diversity Supplement, NSP Scholar, SPINES
meghan.ramirez@unmc.edu

Gabriella Robertson, BA
Vanderbilt University
D-SPAN F99/K00
gabriella.l.robertson@vanderbilt.edu

Genesis Rodriguez, MS
University of Michigan
NSP Scholar
genesiro@umich.edu
Twitter: @a_neurogenesis
LinkedIn:
http://linkedin.com/in/genesisrodriguez24

Ciorana Roman Ortiz, PhD
University of Southern California
D-SPAN F99/K00
cromanor@usc.edu
Twitter: @itsalsohalffull

Michael Rosario, PhD
Boston University
D-SPAN F99/K00
mar340@bu.edu

Lester J Rosario-Rodríguez, PhD
University of Puerto Rico-Medical Sciences Campus
D-SPAN F99/K00
lester.rosario@upr.edu
LinkedIn: https://www.linkedin.com/in/lester-j-rosario-rodr%C3%ADguez-ph-d-3498a1114/

Deanna Ross, BS
The University of Texas at Austin
ENDURE Alum
deanna.ross@utexas.edu

Nihal Salem, PhD
The University of Texas at Austin
D-SPAN F99/K00
nihal.salem@austin.utexas.edu
Twitter: @nihal_salem
LinkedIn: https://www.linkedin.com/in/nihal-ashraf-salem-a569833b/

Stephanie Sandoval-Pistorius, PhD
University of California, San Francisco
D-SPAN F99/K00
stephanie.andovalpistorius@ucsf.edu
Twitter: @steph_spisto
LinkedIn:
https://www.linkedin.com/in/stephanie-sandoval-pistorius-0a3ab413/

Kirsten Schoonover, PhD
University of Pittsburgh
D-SPAN F99/K00
schoonoverke@upmc.edu
Twitter: @keschoonover1

Alexander Silvagnoli, BS
Central Michigan University
Diversity Supplement
silva7a@cmich.edu

Yanilka Soto, BS
University of California, San Francisco
ENDURE Alum
yanilka.soto-muniz@ucsf.edu

Whitney Stevens-Sostre, PhD
University of Wisconsin-Madison
D-SPAN F99/K00
stevenssostr@wisc.edu
Twitter: @stevenssostre
LinkedIn:
https://www.linkedin.com/in/whitneystevenssostre/

Noelle Stiles, PhD
University of Southern California
BRAIN K99/R00
nstiles@usc.edu

Karen Tonsfeldt, PhD
University of California, San Diego
BRAIN K99/R00
ktonsfeldt@health.ucsd.edu

Victoria Sayo Turner, MS
University of California, San Francisco
NSP Scholar
victoria.turner@ucsf.edu

Kaysaw Tuy, BS
University of Alabama at Birmingham
D-SPAN F99/K00
ktuy@uab.edu
Twitter: @kaysawt
LinkedIn: https://www.linkedin.com/in/kaysaw-tuy-61568b94/

Lee O. Vaasjo, BS
Tulane University
D-SPAN F99/K00
lvaasjomunoz@tulane.edu

Anna Vannucci, MA
Columbia University
D-SPAN F99/K00
anna.vannucci@gmail.com

Natalia Vélez, PhD
Harvard University
nvelez@princeton.edu
D-SPAN F99/K00
Twitter: @natvelali

Jose Vigil, MS
University of Colorado Anschutz Medical Campus
Diversity Supplement, NSP Scholar
jose.vigil@cuanschutz.edu

Samara Vilca, BA
University of Miami
D-SPAN F99/K00
svilca@miami.edu
Twitter: @samaravilca
LinkedIn: https://www.linkedin.com/in/samara-vilca-a23964124/

Erika Vitale, PhD
University of Kansas
BRAIN Diversity Supplement
evitale@ku.edu

Cory White, PhD
Johns Hopkins University School of Medicine
NSP Scholar, D-SPAN F99/K00
cwhite84@jhmi.edu
Twitter: @coryjwhite

Antonio White, BS
Michigan State University
NSP Scholar
whitea79@msu.edu
Twitter: @tonedscientist
LinkedIn: https://www.linkedin.com/in/antonio-white-437652a0/

Kellie Williford, PhD
Duke University
D-SPAN F99/K00
kellie.williford@duke.edu
Twitter: @willifordkellie
LinkedIn: https://www.linkedin.com/in/kellie-williford-5981506b/

Joyce Woo, BA
Georgetown University
ENDURE Alum
jw1936@georgetown.edu
Twitter: @joycewoo22

Jie Zheng, PhD
Boston Children's Hospital, Harvard Medical School
BRAIN K99/R00
jie.zheng@childrens.harvard.edu

Resources

General resources and reminders:

- Resource on Scientific Paper Writing: https://www.nature.com/scitable/topicpage/scientific-papers-13815490/
- Individual Development Plan: https://myidp.sciencecareers.org/
- The Center for the Improvement of Mentored Experiences in Research (CIMER): https://cimerproject.org/
- NINDS Landis Award: https://www.ninds.nih.gov/funding/about-funding/types-research-support/achievement-awards/ninds-landis-award-outstanding-mentorship-ninds-investigator
- **Save the date!** 10th Annual BRAIN Initiative Meeting will be held on June 17-18, 2024 in the Bethesda area. There may be satellite events of interest on the preceding day/evening on Sunday, June 16.

From our panelists:

- Dr. AZA Allsop:
  - For more of AZA's work, go to: https://linktr.ee/mefreely
  - For playlist: https://songwhip.com/aza2/mefreely
  - Check out some of AZA's science here: https://youtu.be/p0ZBJT7KMOs
- Dr. Sarah Kucenas:
  - Story Collider: https://www.storycollider.org/stories/2023/5/19/resurfacing-stories-about-coming-back-to-oneself
  - Building Up the Nerve S3E4 Adaptive mentoring https://ninds.buzzsprout.com/558574/10507544-s3e4-adaptive-mentoring
- Dr. Natalia Vélez:
  - Website: https://nataliavelez.org/sketches
- Other:
  - BRAIN® Initiative "Show us your BRAINs! Photo Video Contest": https://braininitiative.nih.gov/news-events/show-us-your-brains-photo-video-contest
  - NIH Sound Health: https://www.nih.gov/sound-health/music-mind-2018
  - Dance your PhD: https://www.science.org/content/page/announcing-annual-dance-your-ph-d-contest

From our Keynote:

- Dr. Letisha Wyatt's slides can be found at the end of this document.

Things they don't tell you -

Steps for Curating an Effective Professional Presence

Writing Your Blueprint

2023 NINDS Blueprint Diversity Conference

Presenter: Letisha R. Wyatt, PhD | Assoc. Professor of Neurology, OHSU

What We'll Cover

What is a Professional Presence?

When Should You Develop a Professional Identity?

How to Get Started

"Professional Presence"

Refers to you overall image, demeanor, and conduct portrayed in your professional life. It encompasses the way you present yourself and interact with others in a professional setting.

- Communication skills
- Professional etiquette (e.g., punctuality, confidentiality, etc)
- Emotional intelligence
- Professional competencies
- Professional ethics
- Digital footprint

"curation"

The deliberate selection, organization, and management of content and information (e.g., research, expertise, achievements, and interests) in a way that highlights your professional identity and aligns with your goals.

When Should You Start?

Career Time point	Grad Student	Postdoc	Jr. Faculty
Early Stage	• Mentorship • Advisor/Sponsor • Leadership • Sci Training		
Middle Stage	• Funding Applications • Science Comm (talks, papers, outreach)		
Late Stage	• Networking • Self Promotion • Job Search • Career Advance.		

Benefits of an Effective Professional Presence

Knowledge Sharing & Learning

Exposure to different ideas and perspectives enhances your understanding of different research areas, methods. Also facilitates public understanding of science.

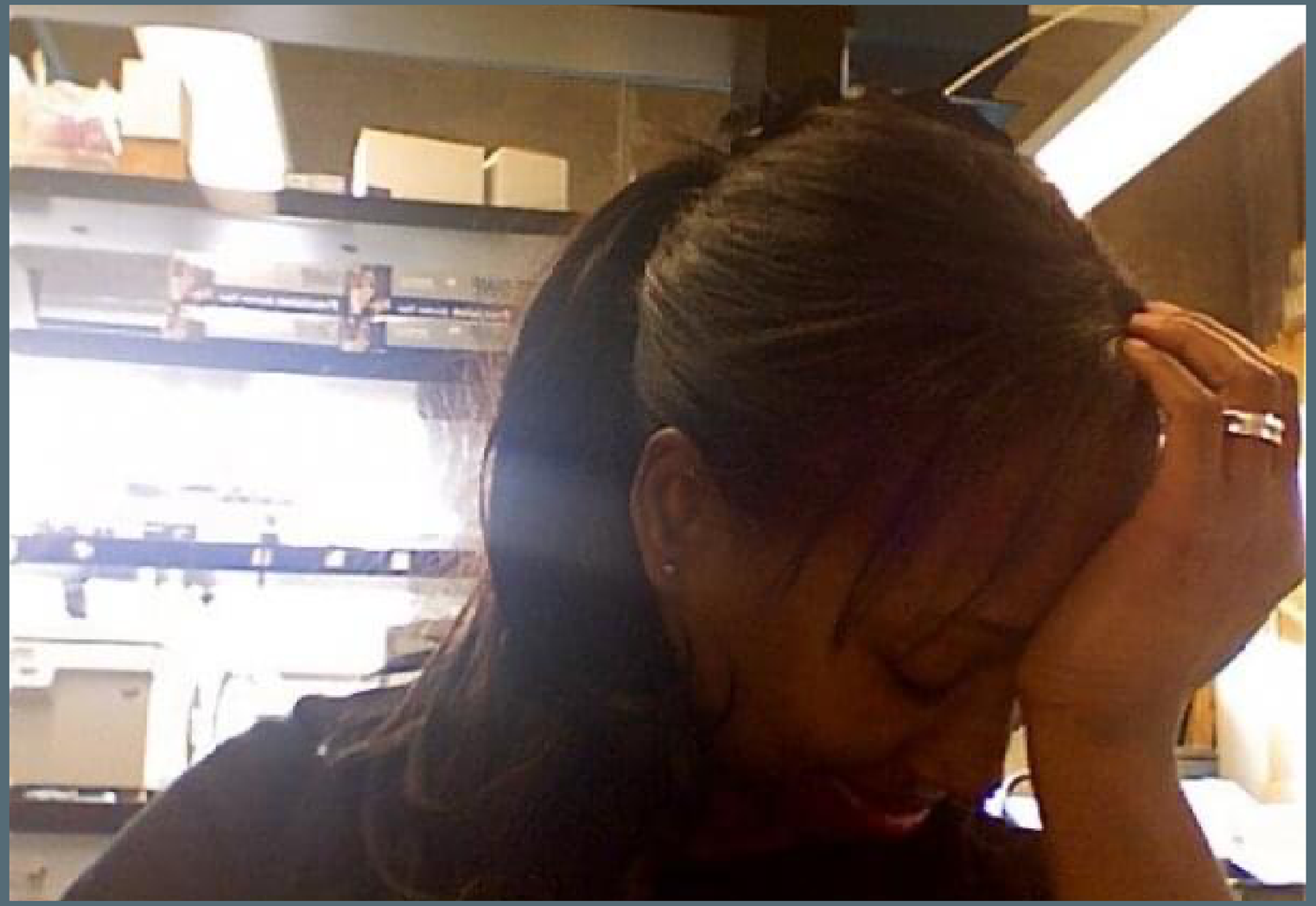
Networking Opportunities

Expand your network to establish new mentors, collaborators, and to stay up to date on emerging trends for your professional development needs.

Visibility & Recognition

Leading to invitations for speaking engagemetns, collaborations, and job or funding opportunities.

What Matters To YOU?



Why didn't this protocol work?

- Did the antibody go bad?
- Was the correct version of protocol used?
- Did I effectively teach my undergrad mentee how to do this?
- How can I measure their learning?

Roadmap

Expertise

Strengths

Values

I Define Self

II Define Goals

Personal Mission

Short

Long

Your Brand

Relevance

Discovery

Timing

III Define Audience

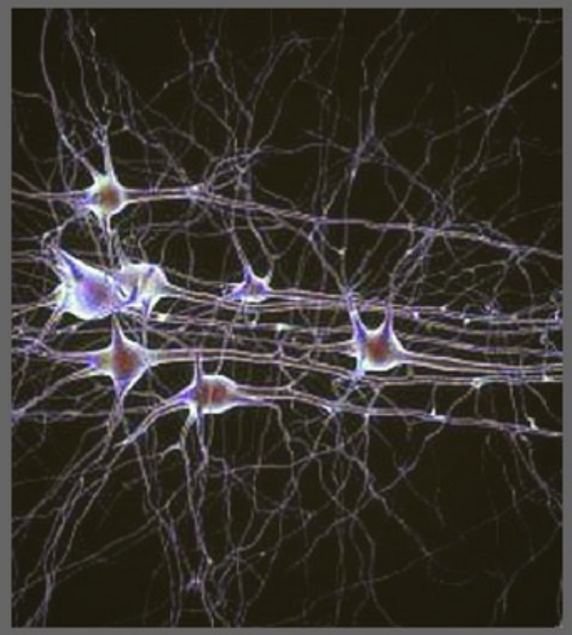
IV Craft Message

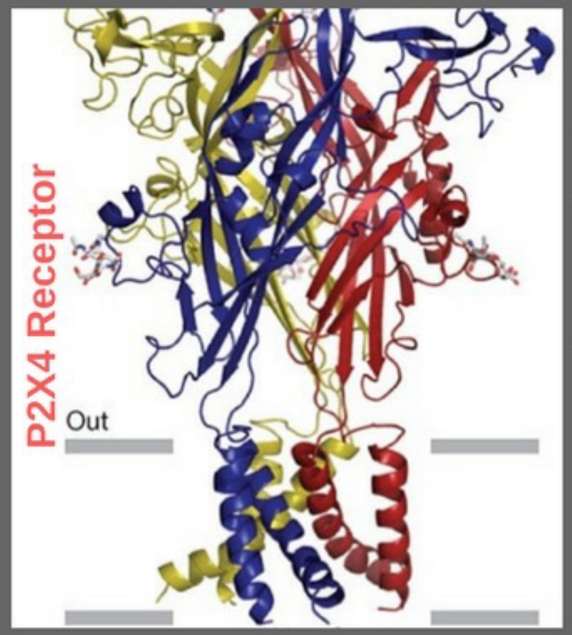
Interests

Achievements

Call to Action

Defining Self





Characterize → Drug Design → Drug Testing

Don't Overlook What you Know

RESEARCH AND ANALYTICAL SKILLS

- Locate & synthesize lots of complex info
- Think on your feet
- Intellectual Independence
- Problem-solve
- Domain Knowledge

INTERPERSONAL AND COMMUNICATION SKILLS

- Lead
- Cope with and manage complicated personalities
- Thrive in competitive environments
- Intellecutual Independence
- Convey complex information
- Build and support community
- Assemble a team

WHAT IT TAKES TO ADVANCE & FINISH

- Perform under pressure
- Focus
- Learn and adapt
- Meet high expectations
- Task Completion

ENTREPRENEURIAL SKILLS

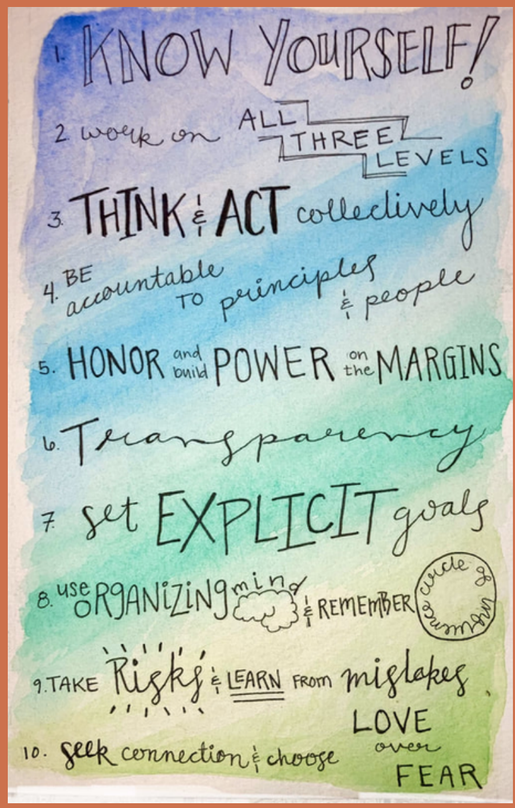
- Work independently
- Acquire Funding
- Prototype new ideas
- Understand competition
- Visionary

"values"

The beliefs that influence people's behavior and decision making.

What I value:

1. Concern for Others
2. Belonging
3. Responsibility
4. Spirituality
5. Independence
6. Concern for the Environment
7. Achievement
8. Creativity
9. Financial Prosperity
10. Privacy
11. Objective Analysis
12. Humility
13. Health and Activity
14. Interdependence



Roadmap

- Expertise
- Strengths
- Values

I Define Self

II Define Goals

- Personal Mission
- Short
- Long

Your Brand

- Relevance
- Discovery
- Timing

III Define Audience

IV Craft Message

- Interests
- Achievements
- Call to Action

Defining Goals

Your Personal Mission Statement

Is a guiding principle that can provide clarity and motivation as you navigate through your personal and professional endeavors.

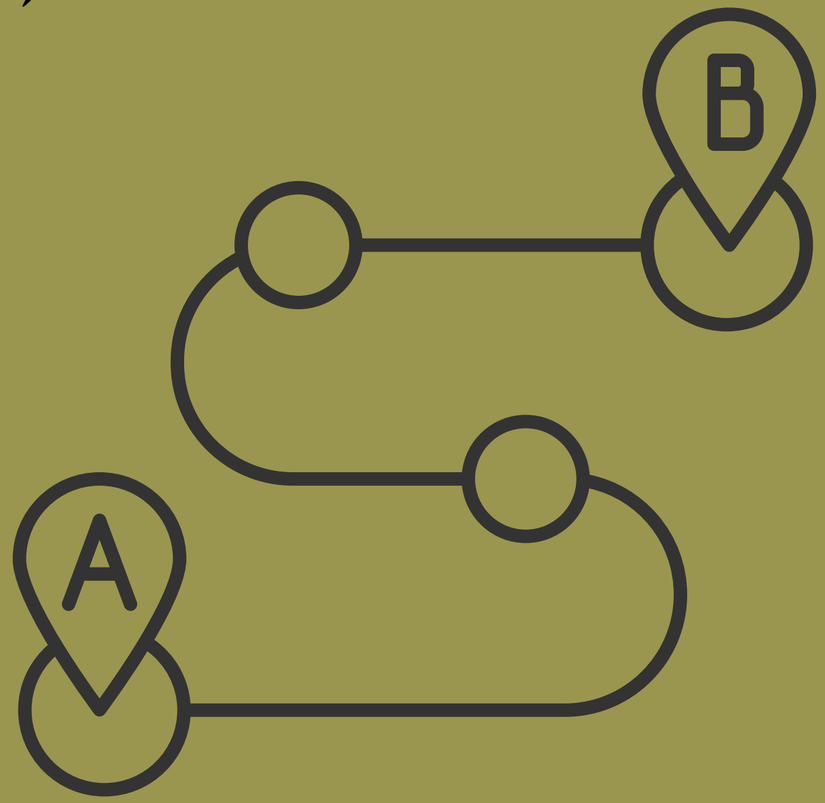
Helpful Reframe

From: I'm a PhD in X, specializing in Y

To: I'm currently a biomedical researcher and what I find most meaningful is Y [doing/learning/impact]

-Jennifer Polk (From PhD to Life)

Goal Setting with your Individual Development Plan (IDP)



1. Knowledge & Intellectual Abilities
    - The knowledge and technical skill needed to do research
2. Personal Effectiveness (inward)
    - Personal qualities needed to be an effective researcher
3. Research Management (outward)
    - People management
    - Project/Program management
    - Resource management
4. Engagement, Influence & Impact

https://ec.europa.eu/research/participants/documents/downloadPublic?documentIds=080166e5bd4ed377&appId=PPGMS

Letisha R. Wyatt, PhD

Working to build a diverse and equitable biomedical research ecosystem

UC DAVIS (2005)

BS in Neurobiology, Physiology, and Behavior

USC GRAD (2013)

PhD in Molecular Pharmacology & Toxicology

(2 gap years in clinical research)

INDUSTRY POSTDOC (2013-2016)

(existential dread)

OHSU FACULTY (2016-)

OHSU Library -> Neurology/NGP

OHSU LIBRARY

better stronger FASTER

DATA LITERACY

project & data mgmt principles
experimental design guidance
analysis & visualization tools
storage & sharing options

RESEARCH SUPPORT

efficient PubMed searches
citation management tips
presentation prep
federal funding requirements
scholarly writing (grants/manuscripts)

Lenisha Wyatt, Ph.D. | wyattle@ohsu.edu | 503.494.8627

OHSU LIBRARY

RESEARCH PROJECT WORKSHOPS

SESSION TITLES

data management fundamentals 1 & 2 | data organization best practices | efficient searches 1 & 2 (Ovid/PubMed) | citation management | intro to data analysis & visualization | scholarly communication 1 & 2

PROJECT BOOT CAMP

Complete all workshops & receive a **$250 travel award!**

space is limited, RSVP early!

SESSION INFO

Who: OHSU students, faculty, staff
What: 90 minute workshops
When: every 1st and 3rd Wednesday, begins Mar. 01, 2017 @ 3:00-4:30 pm
Where: BICC 429

DETAILS AND SIGN UP: TINYURL.COM/GOS2BFN

Roadmap

Expertise
Strengths
Values

I Define Self

II Define Goals

Personal Mission
Short
Long

Your Brand

Relevance
Timing
Discovery

III Define Audience

IV Craft Message

Interests
Achievements
Call to Action

Craft Message

Your Brand Starts with a Great Bio
(...and headshot)

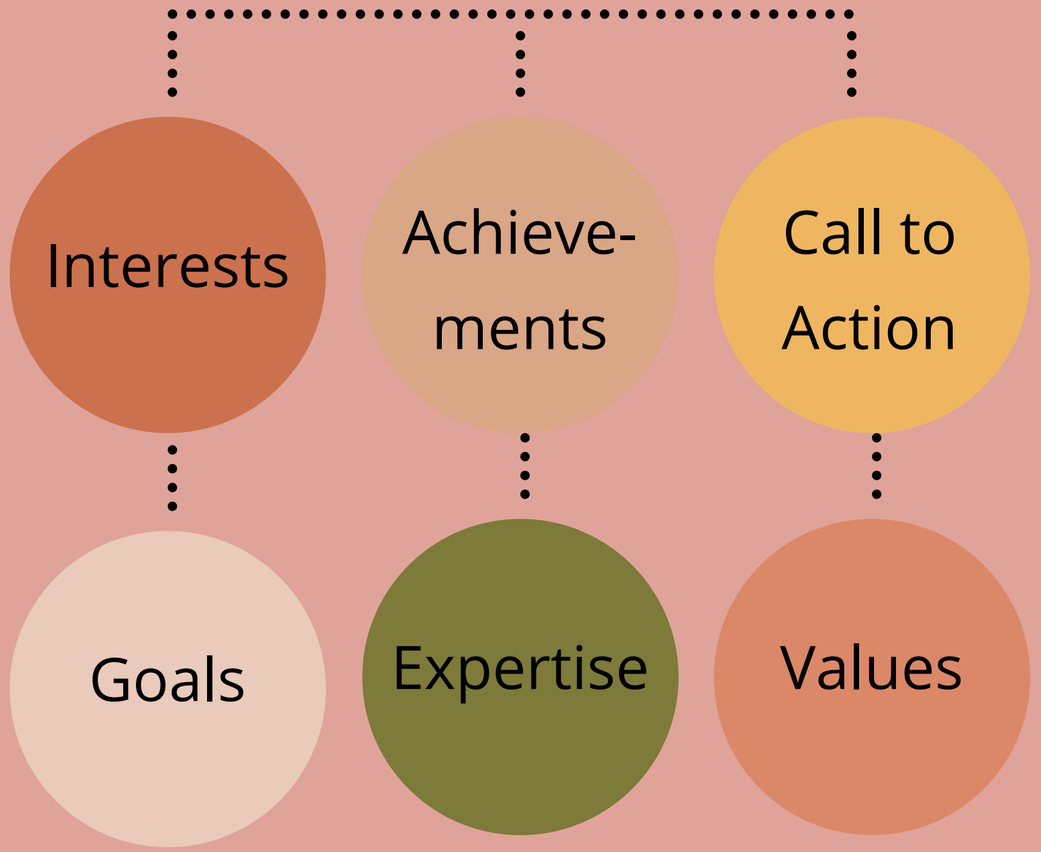
1. Your bio is a communication tool.

2. Your bio is not a list of everything you've done.

3. Your bio tells a story of your credibility, goals, activities and accomplishments.

4. Your bio should be concise, serving as an abstract of yourself or project.

Key Ingredients



WHY ARE YOU AN EXPERT (WHAT DO YOU BRING TO THE TABLE)

GOALS & INTERESTS (WHAT DO YOU WANT TO ACHIEVE)

ACHIEVEMENTS (WHAT WOULD YOU BRAG ABOUT)

A CALL TO ACTION (HOW CAN YOUR AUDIENCE ENGAGE)

Using a Recipe Makes Writing a Bio Easier

Explain what you do + **Establish credibility** +

Describe your professional or research interests +

Brag a little + **Call to action**

Explain what you do + Establish credibility + Describe your professional or research interests + Brag a little + Call to action

Letisha R. Wyatt is an Associate Professor of Neurology and Director of Diversity in Research at Oregon Health & Science University. She holds a Ph.D. in Molecular Pharmacology & Toxicology from the University of Southern California (2013). Her graduate and postdoctoral research focused on purinergic signaling in the central nervous system as a molecular target for new treatments for alcohol abuse and stroke. Letisha is a former NIH predoctoral fellow and has strong record of mentorship in the laboratory and classroom. Her work involves developing campus-wide efforts for enhancing equity in STEM, inclusivity in laboratories and graduate education, and supporting researchers in establishing strong open science and data stewardship practices. Read more about Letisha's role on her departmental webpage and connect with her on twitter: @dr_lrwyatt.

A HANDLE THAT CLEARLY IDENTIFIES YOU OR YOUR RESEARCH INTERESTS

A "BRAND" CONSISTENT PHOTO

SHORT BIO (160 CHARACTERS).

INCLUDE:

- RESEARCH INTERESTS
- AFFILIATIONS
- #HASHTAGS

LINK TO ADDITIONAL INFO

Adapted from: http://blog.impactstory.org/research-impact-challenge-ebook/

Don't Forget to *Curate*

- Follow up on your brand regularly - put it on your calendar (quarterly or 2x/year)
- Get an accountability buddy
- Document, document, document

Inspiring Resources

Deep Dives

- Impactstory 30 Day Impact Challenge ebook
- $0 Professional Headshot (diy instructions)
- IDP for pre- and postdocs
- IDP for junior faculty
- myIDP - Science Careers

Acknowledgements

Team members (past and present): Robin Champieux, Antoinette Foster, Jackie Wirz, André Walcott

Strengths and Values Assessments

- Strengths Finder Book ($20)
- Life Values Inventory ($0)
- Barrett Values Center ($20)
- Racial Equity Guiding Principles

A plug for AI

No one likes to start from a blank screen. Take the key ingredients and feed it to ChatGPT (or similar) to help you construct your first draft!

Contact Info

Letisha R. Wyatt, PhD
e: wyattL@ohsu.edu
twitter: @dr_lrwyatt
web: www.letisharwyatt.com

"*Professional Presence*"

- Your university, department/program, and research settings
- Conferences
- Google Scholar
- Social Media (Facebook, Twitter, Instagram, TikTok, Threads?)
- Professional websites (e.g., LinkedIn, university profile, lab website)
- Personal website or blog
- Professional online communities (e.g., BlackinNeuoro, SACNAS, etc)