# ANA2016 141st ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION

## BALTIMORE, MD • OCTOBER 16-18, 2016

BALTIMORE MARRIOTT WATERFRONT HOTEL

**OCTOBER 15, 2016**
**PRE-MEETING SYMPOSIUM**
*The Neuroscience of Consciousness and Coma*

ANA ANNUAL MEETING

# FINAL PROGRAM


AMERICAN NEUROLOGICAL ASSOCIATION
since 1875
www.2016.myana.org
www.myana.org

## THE 141ST ANA ANNUAL MEETING

Enjoy outstanding scientific symposia covering the latest research in the fields of neurology and neuroscience and take the opportunity to network with leaders in the world of academic neurology at the 141st ANA Annual Meeting in Baltimore, Maryland, October 16-18, 2016.

## IMPORTANT DATES

**Early Registration Deadline:**
August 29, 2016

**Hotel Reservation Deadline:**
October 1, 2016

**Onsite Registration Opens:**
October 15, 2016

**Pre-conference Symposium:**
October 15, 2016

**NINDS Career Development Meeting (invitation only):**
October 14 - 15, 2016

**Annual Meeting Dates:**
October 16 - 18, 2016

## LOCATION

Baltimore Marriott Waterfront Hotel
700 Aliceanna Street
Baltimore, MD 21202

## REGISTER ONLINE

**http://2016.myana.org**

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# FROM THE CHAIR

## DEAR COLLEAGUES,

It is my honor to recognize the enthusiasm and outreach efforts of the ANA Board of Directors, Scientific Program Advisory Committee, and colleagues throughout academic neurology, whose work resulted in a record number of abstract submissions for this, the American Neurological Association's 141st Annual Meeting.

The ANA's Annual Meeting, which will be held on October 16-18, 2016 in Baltimore, MD, will offer exceptional scientific presentations and posters, as well as an incomparable opportunity to connect with colleagues throughout academic neurology. The scientific symposia presented at the meeting will cover a broad spectrum of subspecialties. The poster sessions are packed with the latest emerging neuroscience, and interactive lunch workshops will take scientific breakout sessions to the next level. This meeting also offers a wonderful opportunity for career development at all levels of academic neurology and networking opportunities with leaders in the field.

The Scientific Program Advisory Committee is pleased with the scientific program and eagerly anticipates the Pre-Meeting Symposium on The Neuroscience of Consciousness and Coma. The brain is the most complex organ in the human body, and consciousness is its most complex function. Recent research is providing increased awareness of stages of recovery from brain injury, and the recognition of consciousness in some people previously thought to be too severely injured to regain consciousness. Neurologists are expected to be experts in guiding the families of patients with severe traumatic brain injury, yet the speed, complexity, and limitations of the research is challenging to incorporate into one's clinical practice. I hope you will take advantage of this evening session as a panel of experts discuss the neuroscience of consciousness and coma.

In addition to the outstanding programming, we are pleased to mention that our 2016 Annual Meeting will afford each of us the opportunity to welcome and celebrate our colleagues from the Italian Neurological Society. We are thrilled to continue the tradition of recognizing colleagues and collaborators from overseas as was done in the past several years with the Indian Academy of Neurology in 2015, Mexican Academy of Neurology in 2014, French Société Française de Neurologie in 2013 and Association of British Neurologists in 2012. This coincides with new efforts within the ANA to consider potential programs to extend collaborations in neurological teaching and research with academic neurologists globally.

We are confident that this year's ANA Annual Meeting will be exceptional.

Laura Ranum, Ph.D.
Chair, Scientific Program Advisory Committee
*University of Florida*

# SCHEDULE AT A GLANCE

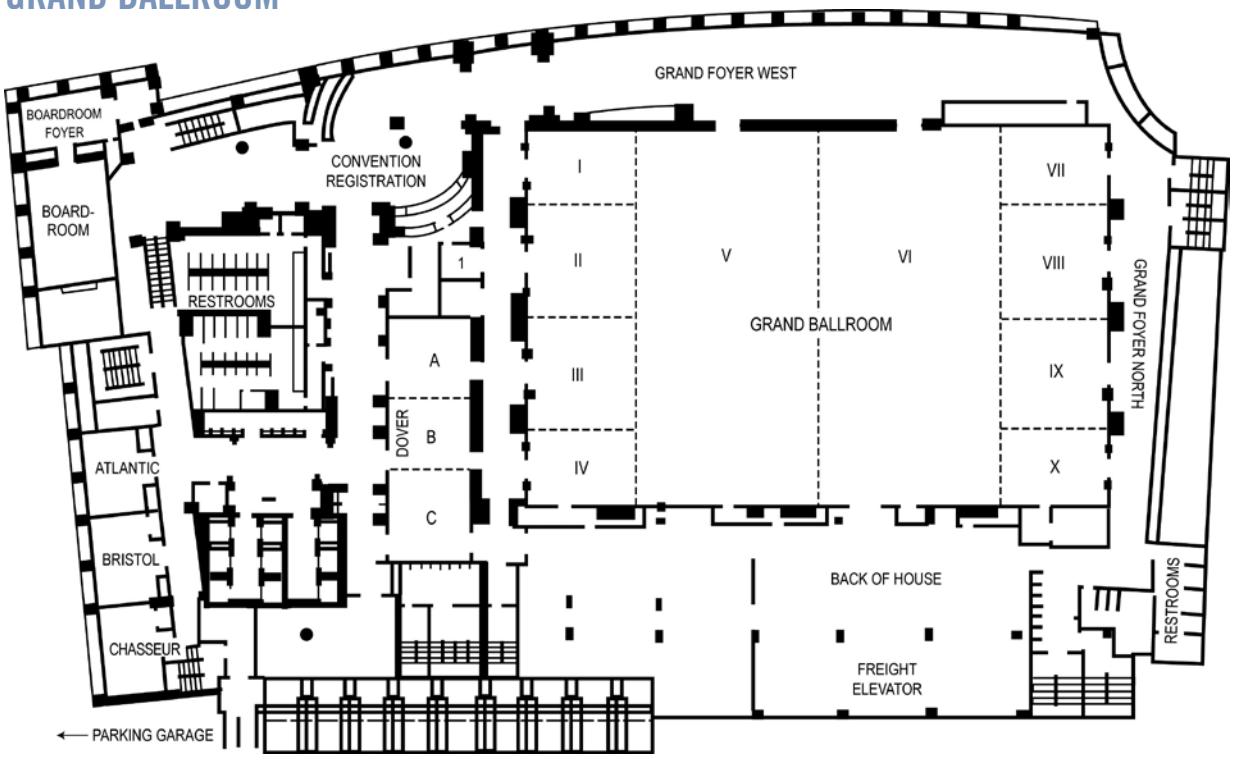
| | | | | | | |
|---|---|---|---|---|---|---|
| **FRIDAY, OCTOBER 14, 2016** | | | | | | |
| 4:00 PM – 7:30 PM | NINDS/ANA Career Development Symposium (invitation only) *Laurel A-D* | | | | | |
| **SATURDAY, OCTOBER 15, 2016** | | | | | | |
| 7:15 AM – 7:45 PM | NINDS/ANA Career Development Symposium (invitation only) *Laurel A-D* | | | | | |
| 3:00 PM – 7:00 PM | Registration Hours *Harborside Registration (A&B)* | | | | | |
| 6:00 PM – 10:00 PM | **Pre-Meeting Symposium: The Neuroscience of Consciousness and Coma** *Grand Ballroom 6* | | | | | |
| **SUNDAY, OCTOBER 16, 2016** | | | | | | |
| 6:00 AM – 5:45 PM | Registration Hours *Harborside Registration (A&B)* | | | | | |
| 7:00 AM – 9:00 AM | Breakfast *Grand Ballroom Foyer* | | | | | |
| 7:30 AM – 9:00 AM | **Career Development Workshops** | | | | | |
| | **Training Opportunities for Students, Residents and Fellows: Preparing for a Successful Career in Academic Neurology** *Grand Ballroom 1&2* | **Early and Mid/Senior Career Level Faculty Development Course I: "Navigating Academic Track: Roads Well Traveled"** *Grand Ballroom 9&10* | **AUPN Chair Career Level Faculty Development Course I: Measuring Academic Productivity: Implementation of an aRVU Model** *Grand Ballroom 7&8* | | | |
| 9:00 AM – 9:15 AM | Coffee Break *Grand Ballroom Foyer* | | | | | |
| 9:15 AM – 11:15 AM | **Plenary Session - Rethinking the Blood Brain Barrier: Brain Drains and Innate Immunity in Neurologic Disease** *Grand Ballroom 5&6* | | | | | |
| 11:15 AM – 11:45 AM | New Member Meet and Greet* *Grand Ballroom 7&8* | | | | | |
| 11:30 AM – 1:00 PM | Lunch *Grand Ballroom Foyer* | | | | | |
| 11:45 AM – 1:00 PM | **Interactive Lunch Workshops** | | | | | |
| | 1. Driving and Neurological Disease *Dover A/B* | 2. Hot Topics in Endovascular - Intra-arterial Lysis Controversies *Kent A/B/C* | 3. Optical Coherence Tomography in Multiple Sclerosis *Dover C* | 4. Are Alzheimer's and Parkinson's Diseases of Childhood? *Grand Ballroom 3&4* | 5. Emerging Impact of Digital Technologies on Diagnosis and Treatment *Essex A/B/C* | 6. Meet the Editors* *Grand Ballroom 1&2* |
| 1:15 PM – 3:15 PM | **Plenary Session: Derek Denny Brown Young Neurological Scholar Symposium** *Grand Ballroom 5 & 6* | | | | | |
| 3:30 PM – 5:30 PM | **Special Interest Group Symposia** | | | | | |
| | 1. Behavioral Neurology *Dover A/B* | 2. Cerebrovascular Disease *Kent A/B/C* | 3. Epilepsy *Essex A/B/C* | 4. Movement Disorders *Grand Ballroom 1&2* | 5. Multiple Sclerosis *Grand Ballroom 3&4* | 6. Neurocritical Care *Grand Ballroom 7&8* | 7. Neuro-Oncology *Grand Ballroom 9&10* |
| 5:30 PM – 7:00 PM | Poster Presentation & Reception I *Harborside Ballroom* | | | | | |
| **MONDAY, OCTOBER 17, 2016** | | | | | | |
| 6:30 AM – 5:45 PM | Registration Hours *Harborside Ballroom (A&B)* | | | | | |
| 7:00 AM – 9:00 AM | Breakfast *Grand Ballroom Foyer* | | | | | |
| 7:30 AM – 9:00 AM | **Career Development Workshops** | | | | | |
| | **Early and Mid/Senior Career Level Faculty Development Course II Off the Beaten Track: "Roads Less Traveled"** *Grand Ballroom 9&10* | | | **AUPN Chair Career Level Faculty Development Course II Teleneurology in Academic Practice** *Grand Ballroom 7&8* | | |
| 9:00 AM – 9:15 AM | Coffee Break *Grand Ballroom Foyer* | | | | | |
| 9:15 AM – 11:15 AM | **Plenary Session: Beyond the Genome: Toward Precision Medicine in Neurology** *Grand Ballroom 5&6* | | | | | |
| 11:15 AM – 11:45 AM | **Executive Session of Membership*** *(All Members are encouraged to attend) Grand Ballroom 5&6* | | | | | |
| 11:30 AM – 1:00 PM | Lunch *Grand Ballroom Foyer* | | | | | |
| 11:45 AM – 1:00 PM | **Interactive Lunch Workshops** | | | | | |
| | 1. Cell-based Therapies for MS *Dover A/B* | 2. Whither Neuro-rehabilitation *Kent A/B/C* | 3. Seizures and Neuropathology *Grand Ballroom 1&2* | 4. Special Topics in Residency Education *Grand Ballroom 3&4* | 5. Advances in Understanding and Treatment of Mitochondrial Disorders *Grand Ballroom 7&8* | 6. Meet the Neurology Department Chairs *Dover C* |
| 11:45 AM – 1:00 PM | **Additional Workshops** | | | | | |
| | 1. Summary of the ABPN MOC Program: Life-long Learning for Neurologists* *Laurel A&B* | | | 2. Women of the ANA Luncheon *Waterview Ballroom* | | |

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for* ***AMA PRA Category 1 Credits***™.

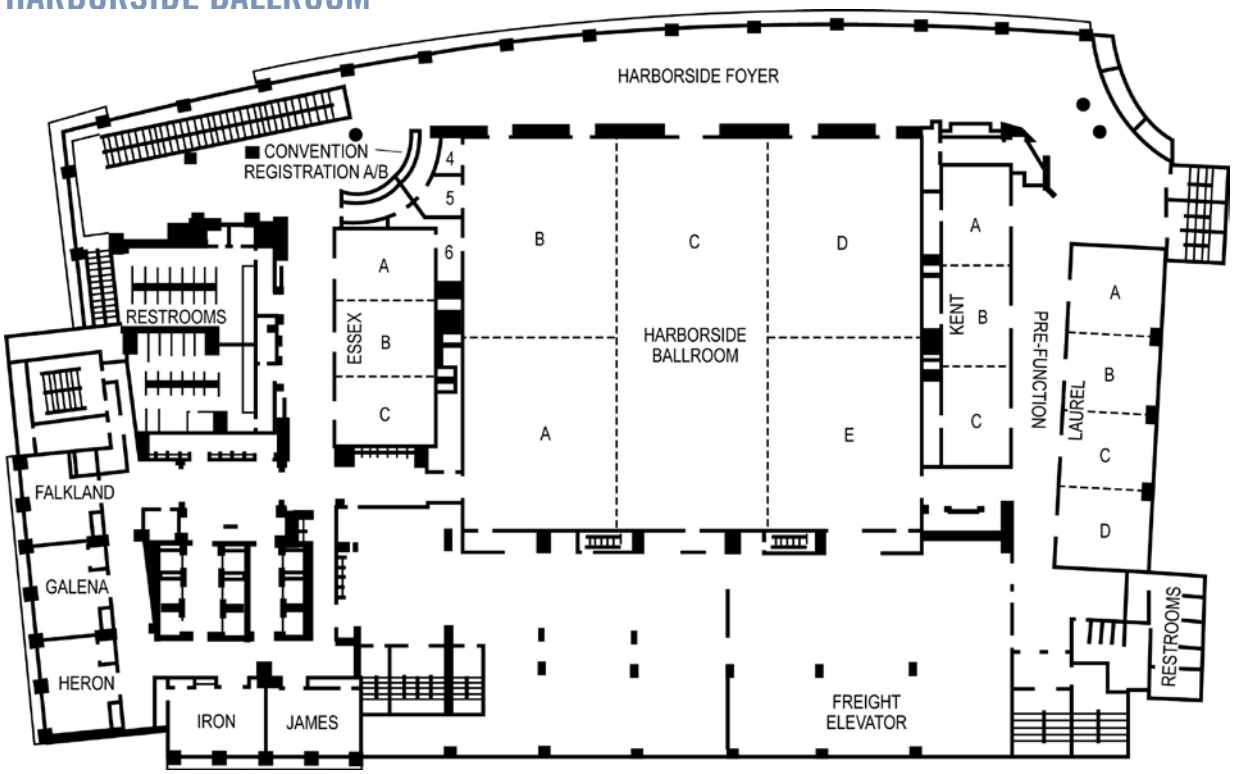
| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **1:15 PM – 3:15 PM** | **Plenary Session - Presidential Symposium: The Role of Academic Neurology in Improving Health** *Grand Ballroom 5&6* | | | | | | | |
| **3:30 PM – 5:30 PM** | **Special Interest Group Symposia** | | | | | | | |
| | 1. Autoimmune Neurology *Dover A/B* | 2. Case Studies *Dover C* | 3. Dementia and Aging *Kent A/B/C* | 4. Education *Grand Ballroom 1&2* | 5. Headache and Pain *Grand Ballroom 3&4* | 6. Health Services Research *Grand Ballroom 7&8* | 7. Interventional Neurology *Grand Ballroom 9&10* | 8. Neuromuscular Disease *Essex A/B* | 9. Sleep Disorders and Circadian Rhythm *Essex C* |
| **5:30 PM – 7:00 PM** | Poster Presentation & Reception II *Harborside Ballroom* | | | | | | | | |
| **7:30 PM – 9:00 PM** | President's Reception *Grand Ballroom 5&6* | | | | | | | | |
| **TUESDAY, OCTOBER 18, 2016** | | | | | | | | | |
| **6:30 AM – 2:00 PM** | Registration Hours *Harborside Ballroom (A&B)* | | | | | | | | |
| **7:00 AM – 9:00 AM** | Breakfast *Grand Ballroom Foyer* | | | | | | | | |
| **7:30 AM – 9:00 AM** | **Career Development Workshops** | | | | | | | | |
| | **Early and Mid/Senior Career Level Faculty Development Course III The View from the NIH and Successful Grant Writing** *Grand Ballroom 9&10* | | | | **AUPN Chair Career Level Faculty Development Course III Neuroscience Service Lines in Academic Neurology** *Grand Ballroom 7&8* | | | | |
| **9:00 AM – 9:15 AM** | Coffee Break *Grand Ballroom Foyer* | | | | | | | | |
| **9:15 AM – 11:15 AM** | **Plenary Session - The Social-Emotional Brain: From Neurobiology to Neurological Disease** *Grand Ballroom 5&6* | | | | | | | | |
| **11:30 AM – 1:00 PM** | Lunch *Grand Ballroom Foyer* | | | | | | | | |
| **11:45 AM – 1:00 PM** | **Interactive Lunch Workshops** | | | | | | | | |
| | 1. Impact of Clinical Trials on Neurologic Practice *Dover A/B* | | 2. Topics and Cases in Neuroinfectious Disease *Kent A/B/C* | | 3. Refractory Status Epilepticus: Mechanisms and Management *Grand Ballroom 1&2* | | 4. Neurosarcoid: The Great Imitator *Dover C* | | 5. Meet the NINDS* *Grand Ballroom 3&4* |
| **11:45 AM – 1:00 PM** | **Additional Workshops** | | | | | | | | |
| | 1.e-Mentoring Program Lunch* *Laurel B* | | | | 2. AUPN'S Networking Lunch for Small Academic Departments of Neurology* *Laurel A* | | | | |
| **1:15 PM – 3:15 PM** | **Plenary Session: Selective Neuronal Dysfunction and Degeneration** *Grand Ballroom 5&6* | | | | | | | | |
| **3:15 PM** | Meeting Adjournment | | | | | | | | |

# FLOOR PLANS

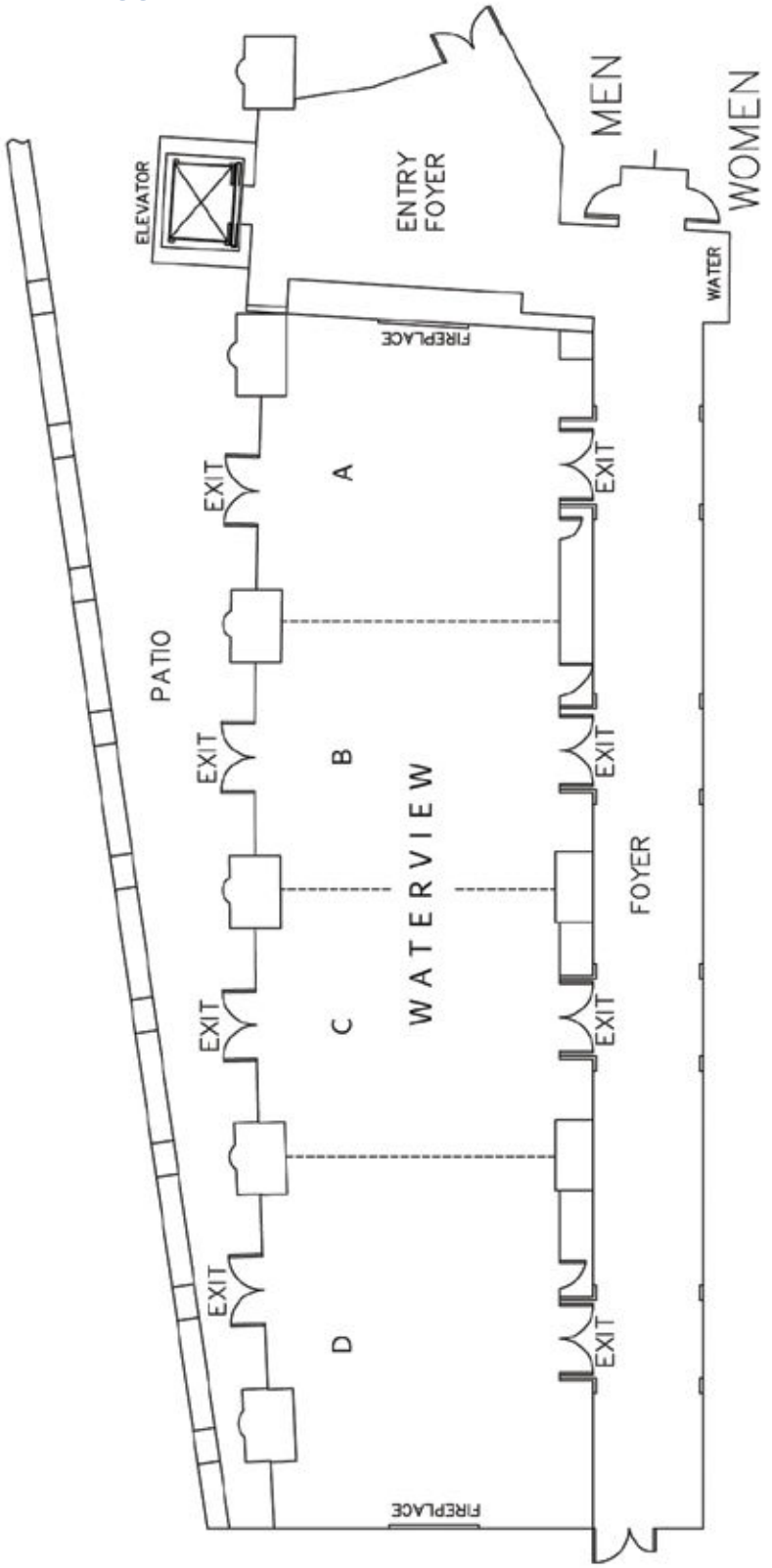
## GRAND BALLROOM




## HARBORSIDE BALLROOM



## WATERVIEW BALLROOM



# GENERAL INFORMATION

## HOTEL INFORMATION

**Baltimore Marriott Waterfront Hotel**
700 Aliceanna Street
Baltimore, MD21202
Main Phone: (410) 385-3000
Check-In Time: 4:00 PM
Check-Out Time: 12:00 PM

**BALTIMORE MARRIOTT WATERFRONT HOTEL**

## ON-SITE REGISTRATION HOURS

*Harborside A&B*

| | |
|---|---|
| Saturday, October 15 | 3:00 PM–7:00 PM |
| Sunday, October 16 | 6:00 AM–5:45 PM |
| Monday, October 17 | 6:30 AM–5:45 PM |
| Tuesday, October 18 | 6:30 AM–2:00 PM |

## POSTER HOURS

*Harborside Ballroom, 4th Floor*

Sunday, October 16 11:00 AM–7:00 PM
*Poster presenters will be in attendance from 5:30 PM to 7:00 PM*

Monday, October 17 11:00 AM–7:00 PM
*Poster presenters will be in attendance from 5:30 PM to 7:00 PM*

## SPEAKER READY ROOM

*Galena, 4th Floor*

| | |
|---|---|
| Saturday, October 15 | 3:00 PM–7:00 PM |
| Sunday, October 16 | 6:00 AM–5:45 PM |
| Monday, October 17 | 6:30 AM–5:45 PM |
| Tuesday, October 18 | 6:30 AM–2:00 PM |

## LUNCH

*Grand Ballroom Foyer, 3rd Floor*

| | |
|---|---|
| Sunday, October 16 | 11:30 AM–1:00 PM |
| Monday, October 17 | 11:30 AM–1:00 PM |
| Tuesday, October 18 | 11:30 AM–1:00 PM |

Boxed lunches are available to be taken into Interactive Lunch Workshops.

## PRESS ROOM

*Heron, 4th Floor*

| | |
|---|---|
| Saturday, October 15 | 3:00 PM–7:00 PM |
| Sunday, October 16 | 6:00 AM–5:45 PM |
| Monday, October 17 | 6:30 AM–5:45 PM |
| Tuesday, October 18 | 6:30 AM–2:00 PM |

## WIRELESS CONNECTION

Wi-Fi: All guest rooms booked under the ANA block will be equipped with complimentary high-speed wireless Internet access during the official conference dates. To connect, turn on Wi-Fi in the device. While in the meeting rooms, look for the network SSID: Marriott_CONF. A splash page will pop up for Guest-Tek. Hit the button "I Accept" and enter the Passcode: 2016ANNUAL (Please Note: This is not case sensitive and there are no spaces). Enter First and Last Name. Hit the button "I Accept". Proceed to internet as normal.

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While on your smartphone, point your mobile browser to m.core-apps.com/ana_annual16 to be directed to the appropriate download version for your phone.


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**#ANAmtg2016**

CONTINUED

## CONTINUING MEDICAL EDUCATION:
### ACCREDITATION & DESIGNATION STATEMENT(S)

**American Neurological Association 141st Annual Meeting**

The American Neurological Association is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The Annual Meeting and Pre-Conference Symposium: The Neuroscience of Consciousness and Comma offer CME to eligible participants. Detailed information pertaining to CME can be found at the following website: https://2016.myana.org/continuing-medical-education

## EVALUATIONS ONLINE

Within a week following the event, you will receive an email containing a link to the evaluation. Please complete the online evaluation within a week of receipt in order to obtain any CME. You will be provided with a certificate following completion of the evaluation. If you have any questions please contact ANA Meeting Manager, Heidi Jetter, at hjetter@myana.org or **1.856.380.6916**.

## CONSENT TO USE OF PHOTOGRAPHIC IMAGES

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## LANGUAGE

The official language of the Annual Meeting is English. No simultaneous translation is available.

## ADA

ANA fully complies with the legal requirements of the Americans with Disabilities Act rules and regulations. If any participant is in need of special accommodations they should notify the hotel and indicate the type of assistance needed. ANA cannot ensure the availability of appropriate assistance without advance notice.

**Disclaimer:** The ANA does not endorse or affiliate with third-party companies, products or services including those that may have elected to support the 2016 Annual Meeting Program.

# PROGRAM

## SATURDAY, OCTOBER 15

**3:00 – 7:00 PM REGISTRATION HOURS**
*Harborside A&B*

**6:00 – 10:00 PM PRE-MEETING SYMPOSIUM: THE NEUROSCIENCE OF CONSCIOUSNESS AND COMA**

**Co-Chairs:** Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins School of Medicine*
Leigh Hochberg, M.D., Ph.D., *Brown University, Harvard Medical School, Massachusetts General Hospital, Brigham and Women's Hospital, Spaulding Rehabilitation Hospital, Providence VA Medical Center*

**Faculty:** Nicholas Schiff, M.D., *Weill Cornell Medical College*
Emory Brown, M.D., Ph.D., *Harvard Medical School and Massachusetts Institute of Technology*
Christopher Koch, M.D., Ph.D., *Allen Institute for Brain Science*
Melanie Boly, M.D., Ph.D., *University of Wisconsin School of Medicine and Public Health*

**6:00 – 7:00 PM DINNER**
*Grand Ballroom 6*

**7:00 – 10:00 PM SYMPOSIUM**
*Grand Ballroom 6*

**7:00 – 7:05 PM**
**Welcome**
**Speaker:** Leigh Hochberg, M. D., Ph.D., *Massachusetts General Hospital*

**7:05 – 7:35 PM**
**Neural Correlates of Consciousness: Progress and Problems**
**Speaker:** Christof Koch, M.D, Ph.D., *Allen Institute for Brain Science.*

**7:40 – 8:10 PM**
**Deciphering the Dynamics of the Unconscious Brain Under General Anesthesia**
**Speaker:** Emery Brown, M.D., Ph.D., *Massachusetts General Hospital*

**8:10 – 8:25 PM**
**Coffee and Dessert Break**

**8:25 – 8:55 PM**
**Mechanisms Underlying Cognitive Motor Dissociation Following Severe Brain Injuries**
**Speaker:** Nicholas Schiff, M.D., *Weill Cornell Medical College*

**9:00 – 9:25 PM**
**Consciousness: From Theory to Practice**
**Speaker:** Melanie Boly, M.D., Ph.D., *University of Wisconsin School of Medicine and Public Health*

**9:30 – 10:00 PM**
**Panel Discussion**

This Pre-Meeting Symposium is on the Neuroscience of Consciousness and Coma. The brain is the most complex organ in the human body, and consciousness is its most complex function. Recent research provides increased awareness of stages of recovery from brain injury, and the recognition of consciousness in some people previously thought to be too severely injured to regain consciousness. Neurologists are now expected to be experts in guiding the families of patients with severe traumatic brain injury, yet the speed, complexity and limitations of the research can prove challenging when incorporating the results into one's clinical practice.

## SUNDAY, OCTOBER 16

**6:00 AM – 5:45 PM ANNUAL MEETING REGISTRATION**
*Harborside A&B, 4th Floor*

**7:00 – 9:00 AM CONTINENTAL BREAKFAST**
*Grand Ballroom Foyer, 3rd Floor*

**7:00 AM – 7:30 AM TRAINEE BREAKFAST**

**7:30 – 9:00 AM TRAINING OPPORTUNITIES FOR STUDENTS, RESIDENTS AND FELLOWS: PREPARING FOR A SUCCESSFUL CAREER IN ACADEMIC NEUROLOGY**

*Grand Ballroom 1 & 2, 3rd Floor*

Drs. Allison Willis and David Fink of the American Neurological Association's Board of Directors have initiated a fellow and resident career development course, which will launch at the ANA 2016 Annual Meeting.

This new session, designed with Fellows and Residents in mind, will feature the following agenda:

**7:00 - 7:30 AM BREAKFAST WITH THE ANA BOARD OF DIRECTORS**

The ANA Board of Directors is composed of academic neurologists at every level, representing all subspecialties from every region of the country. Join the Board for breakfast and an informal discussion on preparing for, entering, and succeeding in a career in academic neurology.

This is a wonderful opportunity to interact with leading academics and discuss the selection of an academic path, areas of research focus, or how to navigate the faculty position seeking process.

**7:30 - 9:00 AM FELLOW AND RESIDENT CAREER DEVELOPMENT SESSION**

**SESSION AGENDA**

**What it is like to be a junior faculty member in an academic department**
Presented by Lesli E. Skolarus, M.D., M.S., *University of Michigan*

**International Opportunities in Academic Neurology**
Presented by Farrah J. Mateen, M.D., Ph.D., *Harvard Medical School*

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for **AMA PRA Category 1 Credits**™.*

**Panel discussion**
Featuring Lesli E. Skolarus, M.D., M.S.; Farrah J. Mateen, M.D., Ph.D.; Allison Willis, M.D., M.S.

## 7:30 – 9:00 AM FACULTY DEVELOPMENT COURSES

### EARLY AND MID CAREER LEVEL FACULTY DEVELOPMENT COURSE I:

**Navigating the Academic Track - "Roads Well Traveled"**
*Grand Ballroom 9 &10*

**Chair:** David Greer, M.D., M.A., FCCM, FAHA, FNCS, FAAN, *Yale School of Medicine*

**Co-Chair:** Amy Pruitt, M.D., *University of Pennsylvania, Perelman School of Medicine*

**Faculty:** Lauren Sansing, M.D., M.S., FAHA, *Yale School of Medicine*
Ellen Mowry, M.D., MCR, *Johns Hopkins School of Medicine*
Steven Feske, M.D., *Brigham and Women's Hospital, Harvard Medical School*
S. Andrew Josephson, M.D., *University of California, San Francisco*

This course is designed to benefit those in the early to mid-stages of their career.

### AUPN CHAIR CAREER LEVEL FACULTY DEVELOPMENT COURSE I:

**Measuring Academic Productivity: Implementation of an aRVU Model**
*Grand Ballroom 7&8*

**Chair:** L. John Greenfield, Jr., M.D., Ph.D., *University of Arkansas for Medical Sciences*

**Faculty:** Arthur Grant, M.D., Ph.D., *SUNY Downstate Medical Center*
Augusto Miravalle, M.D., *University of Colorado Denver School of Medicine*

This course is designed to benefit those in Chair positions or on a trajectory to advance into Chair positions.

## 9:00 – 9:15 AM COFFEE BREAK
*Grand Ballroom Foyer, 3rd Floor*

## 9:15 – 11:15 AM SYMPOSIUM: RETHINKING THE BLOOD BRAIN BARRIER: BRAIN-DRAINS AND INNATE IMMUNITY IN NEUROLOGIC DISEASE

*Grand Ballrooom 5&6, 3rd Floor*

**Chair:** David Holtzman, M.D., *Washington University School of Medicine in St. Louis*

**Co-Chair:** Laura Ranum, Ph.D., *University of Florida*

Our understanding of the role of innate immunity and the separation between the blood and the brain continues to evolve. Healthcare providers are not aware of these evolving concepts or their potential impact on neurodegenerative disease. This symposium will provide an update on the roles of innate immunity in neurologic disease and new insights into understanding that the glymphatic and newly discovered lymphatic systems provide a drainage system for the brain, and connect the central nervous system with the immune system and the periphery. These and other discoveries change our understanding of the biology of the brain and potential opportunities for drug delivery to the brain.

**Learning Objectives**
1. Understand that the neurodegenerative diseases often involve innate immune responses that play a role in disease modulation.
2. Understand the likely roles that microglia and specific microglia molecules play in the pathogenesis of neurodegenerative diseases.
3. Understand the function of glia in the brain and the role of the glymphatic system in clearing extracellular proteins from the brain.
4. Understand that the newly discovered lymphatic system for the brain allows the entrance and exit of lymphatic fluid, immune cells and waste from the brain.

**9:20 – 9:45 AM**
**Implications of the Meningeal Lymphatic Pathways**
Kari Alitalo, M.D., M.Sc.D., *Wihuri Research Institute, University of Helsinki*

**9:45 – 10:10 AM**
**The brain's glymphatic system: Potential Role in Sleep and Neurodegenerative Diseases**
Helene D. Benveniste, M.D., Ph.D., *Stony Brook School of Medicine*

**10:10 – 10:35 AM**
**TREM2 and the Brain's Innate Immune System: Relevance to Alzheimer's Disease, Multiple Sclerosis, and Neurodegeneration**
Marco Colonna, M.D., *Washington University School of Medicine in St. Louis*

**10:35 – 11:00 AM**
**Modulating the Blood – Brain Barrier for the Enhanced Delivery of Therapeutics**
Lorraine Iacovitti, Ph.D., *Thomas Jefferson University*

### DATA BLITZ PRESENTATIONS:

**11:00 – 11:02 AM**
**Effect of Sleep Deprivation and Sodium Oxybate on CSF A40 and A42 Kinetics**
Brenden Lucey, M.D., *Washington University School of Medicine*

**11:03 – 11:05 AM**
**Alpha-M Integrin (CD11b) Facilitates Pathogenic Leukocyte Trafficking in the Acute Inflammatory Demyelinating Polyradiculoneuropathy Variant of Guillain-Barre Acute Syndrome**
Eroboghene Ubogu, M.D., *University of Alabama at Birmingham*

**11:06 – 11:08 AM**
**Coordinated B Cell Compartmentalization with Antigen Presentation in a Murine Autoimmune Model of Multiple Sclerosis**
Gregory Wu, M.D., Ph.D., *Washington University in Saint Louis*

**11:09 – 11:11 AM**
**New Clinical and Immunological Features of Anti-GABAA Receptor Encephalitis**
Marianna Spatola, M.D., *Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS)*

**11:12 – 11:15 AM**
**Antiglycolipid Antibodies in Neurological Diseases Subsequent to Mycoplasma Pneumoniae Infection**
Susumu Kusonoki, M.D., Ph.D., *Kindai University Faculty of Medicine*

# PROGRAM

### 11:00 AM – 7:00 PM POSTER VIEWING
*Harborside A&B, 4th Floor*

*Poster presenters will be in attendance from 5:30 – 7:00 pm*

### 11:15 – 11:45 AM NEW MEMBER MEET AND GREET*
*Grand Ballroom 7&8, 3rd Floor*

### 11:30 AM – 1:00 PM LUNCH
*Grand Ballroom Foyer, 3rd Floor*

Boxed lunches available to be taken into Interactive Lunch Workshops

### 11:45 AM – 1:00 PM INTERACTIVE LUNCH WORKSHOPS

**These workshops are "lunch and learns".**

#### 1: DRIVING AND NEUROLOGICAL DISEASE
*Dover A/B, 3rd Floor*

**Moderator:** Matthew Rizzo, M.D., *University of Nebraska Medical Center*

Current status of research and clinical guidelines to determine when patients with prevalent neurological diseases that impair driving should stop doing this activity.

#### 2. HOT TOPICS IN ENDOVASCULAR - INTRA-ARTERIAL LYSIS CONTROVERSIES
*Kent A/B/C, 4th Floor*

**Moderator:** Elisabeth Marsh, M.D., *Johns Hopkins University School of Medicine*

**Co-Moderator:** Rafael Llinas, M.D., *Johns Hopkins University School of Medicine*

**Faculty:** Geoffrey Colby, M.D., *Johns Hopkins University*
Carolyn Cronin, M.D., Ph.D., *University of Maryland School of Medicine*
Erin Lawrence, MSN, *Johns Hopkins Medicine*
Hardin Pantle, M.D., *Johns Hopkins Medicine*
Carolyn Philips, M.D., *Johns Hopkins Medicine*

As we do more endovascular cases, some hot topics are arising. We shall highlight/debate some of these such as:
- Re-routing patients – forgo PSC for CSC
- Ambulance Triage – score, telemed (ipad), mobile stroke unit
- Intervening on Tandem lesions

#### 3. OPTICAL COHERENCE TOMOGRAPHY IN MULTIPLE SCLEROSIS
*Dover C*

**Moderator:** Rachel C. Nolan, B.A., *New York University*

**Faculty**: Laura Balcer, M.D., MSCE, *New York University*

Speakers are 2015 recipients of the Barancik Prize for innovation in MS research by the National Multiple Sclerosis Society. Talks will demonstrate innovative uses of optical coherence tomography in multiple sclerosis research.

#### 4: ARE ALZHEIMER'S AND PARKINSON'S DISEASES OF CHILDHOOD?
*Grand Ballroom 3&4, 3rd Floor*

**Moderator:** Dimitri Krainc, M.D., Ph.D., *Northwestern University Feinberg School of Medicine*

**Co-Moderator:** Russell Swerdlow, M.D., *University of Kansas*

**Faculty:** Carlo Colosimo, M.D., *Santa Maria University Hospital*
Gennarina Arabia, M.D., M.Sc., *University "Magna Graecia" of Catanzaro*

Evidence from studies of Parkinson's and Alzheimer's Disease suggest that structural injury and metabolic dysfunction may precede clinical evidence of the disease by decades, and may be present in childhood. Examples of this evidence includes the role of the apolipoprotein E (APOE) Îµ4 allele in the disruption of functional connectivity in the developing brain, and the role of lysosomal dysfunction both in synucleinopathies and lysosomal storage diseases.

#### 5: EMERGING IMPACT OF DIGITAL TECHNOLOGIES ON DIAGNOSIS AND TREATMENT
*Essex A/B/C, 3rd Floor*

**Moderator:** John Krakauer, M.D., *Johns Hopkins University*

**Faculty:** Rosalind Pickard, S.M., Sc.D., FIEEE, *Massachusetts Institute of Technology and Empatica, Inc*
*Omar Ahmad, Ph.D., Johns Hopkins University*

Billions of people now use wireless digital technologies transforming how we communicate with one another. The potential of these technologies to diagnose, monitor, and treat neurological diseases is just beginning to emerge. The proliferation of smart phones and apps integrating accelerometers and GPS have resulted in an explosion of new wearables that have the potential to provide clinicians with powerful new ways to diagnose and monitor responses 24/7, for example in the treatment of epilepsy or movement disorders. In addition, such technologies allow massive data collection, which may allow deeper understanding of inter-individual differences in disease expression and ultimately lead to personalized treatment approaches. The future also promises to harness the power of digital technologies to not just collect information, but also as treatment in themselves.

#### 6: MEET THE EDITORS*
*Grand Ballroom 1&2, 3rd Floor*

**Faculty:** Andrew Josephson, M.D., *JAMA Neurology*
Jack Kessler, M.D., *Annals of Clinical and Translational Neurology*
Cliff Saper, M.D., Ph.D., *Annals of Neurology*

Editors from the named journals will be available to discuss the submission process, publishing, tips, and other key topics of interest.

### 1:15 – 3:15 PM SYMPOSIUM: DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR SYMPOSIUM

*Grand Ballroom 5&6, 3rd Floor*

**Chair:** Thomas Sutula, M.D., Ph.D., *University of Wisconsin School of Medicine and Public Health*

**Co-Chair:** Allison Brashear, M.D., M.B.A., *Wake Forest University School of Medicine*

The Derek Denny-Brown Young Neurological Scholar Symposium is an opportunity for young researchers to share groundbreaking research

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for **AMA PRA Category 1 Credits**™.*

in the field of Neurology. This symposium will feature sessions from the three 2016 Derek Denny-Brown awardees, the Wolfe Research Prize awardee and the Grass Award recipient.

**The Derek Denny-Brown Young Neurological Scholar Award** recognizes early- to mid-career neurologists and neuroscientists. This award honors those neurologists and neuroscientists in the first 10 years of their career at the assistant/associate faculty (equivalent) level who have made outstanding basic and clinical scientific advances toward the prevention, diagnosis, treatment and cure of neurological diseases. As of 2016, the Membership, Honorary and Awards Committee can designate up to a total of three (3) awards in any of the following three areas: Physician Scientist – Basic, Physician Scientist – Clinical, Neuroscientist – relevant to disease. This year the committee has awarded one (1) Physician Scientist – Basic and two (2) Physician Scientist – Clinical.

**The 2016 Grass Foundation – ANA Award in Neuroscience** was established in 2007 to recognize outstanding young physician scientists conducting research in basic or clinical neuroscience. The Grass Foundation was established in 1955 by Albert and Ellen Grass to advance research and education in neuroscience, with a special focus on investigators early in their careers.

**Wolfe Research Prize for Investigators Identifying New Causes or Novel Treatments of Axonal Peripheral Neuropathy** was established to honor outstanding investigators who identify a new cause or treatment of axonal peripheral neuropathy. Eligible candidates include faculty members at any stage in their career (M.D., M.D./Ph.D., or Ph.D.) who identify either a new cause or treatment of axonal peripheral neuropathy. The same researcher can qualify for this award annually if he/she can demonstrate evidence of repeated success in identifying new causes or treatments for axonal peripheral neuropathy.

**1:20 - 1:45 PM**
*Presentation of Derek Denny-Brown Young Neurological Scholar Award in Clinical Science*
**Emerging roles of RNA in Stroke**
Glen Jickling, M.D., M.S.c, FRCPC, *University of California, Davis*

**1:48 – 2:13 PM**
*Presentation of Derek Denny-Brown Young Neurological Scholar Award in Clinical Science*
**Genomic Studies of Developmental Epilepsies Identify Importance of Synaptic Activation Response Pathways**
Alexander Robert Paciorkowski, M.D., *University of Rochester*

**2:16 – 2:31 PM**
*Presentation from the Grass Awardee*
**The Tale of Neural Integrator – From Nystagmus to Dystonia**
Aasef Shaikh, M.D., Ph.D., *Case Western Reserve University*

**2:34 – 2:59 PM**
*Presentation of Derek Denny-Brown Young Neurological Scholar Award in Basic Science*
**Understanding the Role of C9orf72 in Neurodegeneration and Neuroinflammation**
Robert Baloh, M.D., Ph.D., *Cedars-Sinai*

**3:02 – 3:15 PM**
*Presentation from the Wolfe Research Prize Awardee*
**Peripheral Neuropathic Changes in Pachyonychia Congenita**
Michael Polydefkis, M.D., M.H.S., *Johns Hopkins University School of Medicine*

## 3:15 – 3:30 PM COFFEE BREAK

*Grand Ballroom Foyer, 3rd Floor*

## 3:30 PM – 5:30 PM SPECIAL INTEREST GROUP SYMPOSIA

*Dover A/B/C, 3rd Floor*

### BEHAVIORAL NEUROLOGY

**Chair**: Bradford Dickerson, M.D., *Massachusetts General Hospital, Harvard Medical School*

**Co-Chair**: David Wolk, M.D., *University of Pennsylvania, Perelman School of Medicine*

In this session, the speakers will review several major human brain networks subserving "frontal systems" cognitive-affective function and recent advances in our understanding of their functional neuroanatomy and normal behavioral function. Abnormalities of these networks in patients with neurological disorders will be discussed.

**DATA BLITZ PRESENTATION:**
**3:30 PM – 3:40 PM**
**Baseline Aphasia Severity Determines the Effect of Right Pars Triangularis Theta Burst Stimulation on Naming**
Joely Mass, BA Candidate, *University of Pennsylvania*

**3:40 – 3:45 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**3:45 – 4:05 PM**
**The Hierarchical Organization of Frontal Cortex**
Andrew Kayser, M.D., Ph.D., *University of California at San Francisco*

**4:05 – 4:10 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**4:10 – 4:20 PM**
**Cerebeller and DCS to Augment Chronic Aphasia Treatment**
Rajani Sebastian, Ph.D., *Johns Hopkins University School of Medicine*

**4:20 – 4:25 PM**
**Q&A**

**LEADER IN THE FIELD PRESENTATION:**
**4:25 – 4:45 PM**
**The Role of the Frontal Lobes in Value-Based Decision-Making**
Lesley Fellows, M.D,. Ph.D., *Montreal Neurological Institute and Hospital*

**4:45 - 4:50 PM**
Q&A

**DATA BLITZ PRESENTATION:**
**4:50 – 5:00 PM**
**Clinicopathological Features of NCL Occur in Humans with Progranulin Haploinsufficiency**
Michael Ward, M.D., Ph.D., *National Institutes of Health*

**5:00 – 5:05 PM**
**Q&A**

# PROGRAM

LEADER IN THE FIELD PRESENTATION:
**5:05 – 5:25 PM**
**Neural Underpinnings of Novelty Processing**
Kirk Daffner, M.D., *Harvard Medical School/Brigham and Women's Hospital*

**5:25 – 5:30 PM**
**Q&A**

## CEREBROVASCULAR DISEASE

*Kent A/B/C, 4th Floor*

**Chair:** Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins University School of Medicine*

**Co-Chair:** Seemant Chaturvedi, M.D., *University of Miami Miller School of Medicine*

Three areas will be discussed by experts in the field: (1) treatment trials for cerebral amyloid angiopathy; (2) patient-centered, community-based post-stroke care; and (3) neuroimaging and stroke risk in sickle cell disease in children and adults. Many practicing stroke clinicians manage patients in whom all of these issues will be relevant, without knowledge of the latest research in this area. This session will include talks by experts in these areas, and will include review of recent studies and evidence-based medicine. In addition, new research findings in the field of stroke will be presented by trainees and junior investigators. There will be opportunities for questions and answers for each talk, to increase interaction between attendees and speakers.

LEADER IN THE FIELD PRESENTATION:
**3:30 PM – 3:50 PM**
**Vascular Physiology and Treatment Trials for Cerebral Amyloid Angiopathy**
Steven M. Greenberg, M.D., Ph.D., *Massachusetts General Hospital*

**3:50 – 4:00 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:00 PM – 4:08 PM**
**Normalization of BUN/ Creatinine Ratio in Acute Ischemic Stroke Patients is Associated with Less Infarct Expansion**
Mona Bahouth, M.D., *Johns Hopkins Medicine*

**4:08 – 4:10 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:10 – 4:30 PM**
**Comprehensive Post-Acute Stroke Services (COMPASS) Model of Care and Navigation Through the Stroke Continuum**
Cheryl Bushnell, M.D., M.H.S., *Wake Forest Baptist Health*

**4:30 – 4:40 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:40 – 4:48 PM**
**Burden and Outcomes of Acute Ischemic Stroke in Patients with Major Depression**
Zeeshan Mansury, M.D., M.P.H., *Drexel University College of Medicine*

**4:48 – 4:50 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:50 PM – 5:10 PM**
**New MRI-Based Methods for Assessing Stroke Risks in Children and Adults with Sickle Cell Disease**
Lori Jordan, M.D., Ph.D., *Vanderbilt University*

**5:10 – 5:20 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**5:20 – 5:28 PM**
**TGF-1 Modulates Microglial Phenotype and Promotes Recovery After Intracerebral Hemorrhage**
Lauren Sansing, M.D., M.S., F.A.H.A., *Yale University*

**5:28 – 5:30 PM**
**Q&A**

## EPILEPSY: EPILEPTOGENESIS - FROM MECHANISMS TO CLINICAL TRIALS

*Essex A/B/C, 4th Floor*

**Chair:** Gregory Worrell, M.D., *Mayo Clinic*

This session will cover various facets related to the topic of epileptogenesis. A series of experts will highlight the most current and relevant knowledge of animal models of epileptogenesis, its potential electrical biomarkers, and possible clinical trial design targeting the study of candidate antiepileptogenic interventions. The implications of this information on routine clinical care will be discussed.

LEADER IN THE FIELD PRESENTATION:
**3:30 – 3:55 PM**
**Human Cellular and Animal Models of Genetic Epilepsies**
Jack Parent, M.D., *University of Michigan Department of Neurology*

**3:55 - 4:00 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:00 – 4:20PM**
**Progress in Epileptogenesis: Mechanisms, Biomarkers, Therapies**
Kevin Staley, M.D., *Massachusetts General Hospital*

**4:20 - 4:25 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:25 – 4:40 PM**
**Genome-Wide Long Non-Coding RNA Analysis in Mouse Pilocarpine Models of Temporal Lobe Epilepsy**
Yoonhyuk Jang, M.D., *Department of Neurology, Laboratory of Neurotherapeutics, Co*

**4:40 – 4:45 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:45 – 5:05 PM**
**EEG Biomarkers of Epileptogenesis**
Gregory Worrell M.D., *Mayo Clinic*

**5:05 – 5:10 PM**
**Q&A**

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LEADER IN THE FIELD PRESENTATION:
**5:10 - 5:25 PM**
**Epileptogenesis Clinical Trial Design**
Daniel Friedman, M.D., *NYU, Langone Medical Center*

**5:25 -5:30 PM**
**Q&A**

## MOVEMENT DISORDERS
*Grand Ballroom 1&2, 3rd Floor*

Our limited understanding of the pathogenesis of movement disorders is a key barrier preventing the discovery and development of novel therapeutic strategies. In the 2016 Movement Disorders SIG, leading scientists will present the latest findings on the molecular pathogenesis of Parkinson's disease and other movement disorders. All speakers will emphasize how these novel findings may translate into new therapeutic approaches for movement disorders.

**Chair:** Peter Todd, M.D., Ph.D., *University of Michigan*

**Co-Chair:** Pravin Khemani, M.D., *University of Texas Southwestern*

LEADER IN THE FIELD PRESENTATION:
**3:30 PM – 3:50 PM**
**Development of Targeted Therapies for Parkinson's Disease**
Dimitri Krainc, M.D., Ph.D., *Northwestern University Feinberg School of Medicine*

**3:50 – 3:55 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**3:55 PM – 4:05 PM**
**Distinct Synuclein Seeds in Parkinson's Disease and Multiple System Atrophy**
Tritia Yamasaki, M.D., Ph.D., *University of Kentucky*

LEADER IN THE FIELD PRESENTATION:
**4:05 – 4:25 PM**
**Cerebellar Ataxia in the Era of Neurogenomics and Precision Medicine**
Brent Fogel, M.D., Ph.D., *University of California, Los Angeles*

**4:25 – 4:30 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:30 – 4:40 PM**
**Repurposing of Prostate Cancer Therapeutics as a Brain Bioavailable Nurr1 Transactivator for Treatment of Synucleinopathies in Parkinson's Disease**
Giulio Pasinetti, *M.D., Ph.D., Icahn School of Medicine at Mount Sinai*

LEADER IN THE FIELD PRESENTATION:
**4:40 PM – 5:00 PM**
**Functional Neuroimaging to Understand Cognitive Dysfunction in Parkinson's Disease**
Kathleen Poston, M.D., M.S., *Stanford University*

**5:00 – 5:05 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**5:05 – 5:25 PM**
**Biomarker Discovery in Parkinson's Disease**
Alice Chen-Plotkin, M.D., *University of Pennsylvania*

**5:25 – 5:30 PM**
**Q&A**

## MULTIPLE SCLEROSIS
*Grand Ballroom 3&4, 3rd Floor*

**Chair:** Robert Naismith, M.D., *Washington University in St. Louis*

**Co-Chair:** Gregory Wu, M.D., Ph.D., *Washington University in St. Louis*

This SIG will focus on multiple sclerosis (MS) as a neurodegenerative disease. Invited speakers will discuss emerging understanding of the cellular and molecular biology of MS disease progression, clinical methods for assessing risk of progression using biomarkers, state-of-the-art techniques for monitoring disease progression using imaging, and cutting-edge therapeutic efforts aimed at protecting and restoring function in patients with MS. Data blitz speakers have been selected from submitted abstracts that address understanding, monitoring or treating progression in MS.

LEADER IN THE FIELD PRESENTATION:
**3:30 PM – 3:50 PM**
**Update in Pediatric MS**
Jennifer Graves, M.D., Ph.D., *UCSF Medical Center*

**3:50 – 4:00 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:00 – 4:20 PM**
**Cognition in Pediatric MS**
Lauren Krupp, M.D., *NYU Langone Medical Center*

**4:20 – 4:30 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:30 – 4:40 PM**
**DTI-Based Whole Brain Sulcal CSF Volume Is Associated with Cognitive Impairment in Multiple Sclerosis**
Flavia Nelson, M.D., *UT Health*

DATA BLITZ PRESENTATION:
**4:40 – 4:50 PM**
**Claudin-4 is Required for Astrocytic Tight Junction Formation and Protects Against Inflammatory Lesion Size and Severity in the Central Nervous System**
Sam Horng, M.D., Ph.D., *Icahn School of Medicine at Mount Sinai*

DATA BLITZ PRESENTATION:
**4:50 – 5:00 PM**
**Cognitive Impairment in Primary-Progressive Multiple Sclerosis: A Role for Functional Connectivity and Neuronal Variability Changes of Resting-State Activity?**
Maria Petracca, M.D., Ph.D. *Icahn School of Medicine at Mount Sinai*

**5:00 – 5:10 PM**
**Q&A**

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DATA BLITZ PRESENTATION:
5:10 – 5:20 PM
**In Vivo Imaging of Cortical Demyelination and Remyelination in a Mouse Model of Multiple Sclerosis**
Jennifer Orthmann-Murphy, M.D., Ph.D., *University of Pennsylvania*

DATA BLITZ PRESENTATION:
5:20 – 5:30 PM
**Effects of Intermittent Fasting in an Animal Model of Multiple Sclerosis**
Francesca Cignarella, Ph.D., *Washington University in St. Louis*

## NEUROCRITICAL CARE

*Grand Ballroom, 7&8, 3rd Floor*

**Chair:** Paul Nyquist, M.D., M.P.H., *Johns Hopkins University*

**Co-Chair:** Christiana Hall, M.D., *UT Southwestern*

**Co-Chair:** Kevin Sheth, M.D., *Yale University*

This special interest group will give an update on Neurocritical care advances in the last year. We will focus on the monitoring of cerebral blood flow in sepsis, the control of cerebral edema, and the monitoring and control of blood glucose in brain injury.

LEADER IN THE FIELD PRESENTATION:
3:30 – 3:50 PM
**Cerebral Blood Flow: Management and Monitoring in Sepsis Associated Encephalopathy**
Paul Nyquist, M.D., M.P.H., *Johns Hopkins University*

3:50 – 4:00 PM
**Q&A**

DATA BLITZ PRESENTATION:
4:00 – 4:10 PM
**Reemergence of Japanese Encephalitis in South Korea, 2010-2015**
Jun-Sang Sunwoo, *Seoul National University Hospital*

LEADER IN THE FIELD PRESENTATION:
4:10 – 4:30 PM
**Cerebral Edema after Acute Neurovascular Injury**
Kevin Sheth, M.D., *Yale University*

**Q&A**
4:30 – 4:40 PM

DATA BLITZ PRESENTATION:
4:40 – 4:50 PM
**Early Detection of Edema in Malignant Anterior Circulation Stroke: A Risk Prediction Tool**
Charlene Ong, M.D., *Massachusetts General Hospital, Brigham and Women's Hospital, Harvard Medical School*

LEADER IN THE FIELD PRESENTATION
4:50 – 5:10 PM
**Clinical Management of Serum Glucose in Acute Ischemic Stroke**
Christiana Hall, M.D., *UT Southwestern*

5:10 – 5:20 PM
**Q&A**

DATA BLITZ PRESENTATION:
5:20 – 5:30 PM
**Sarm1 Knockout Attenuates Traumatic Axonal Injury and Improves Functional Outcome After Traumatic Brain Injury in Mice**
Nils Henninger, M.D., *UMass Memorial Healthcare*

## NEURO-ONCOLOGY

*Grand Ballroom, 9&10, 3rd Floor*

**Chair:** Howard Fine, M.D., *Weill Cornell Medicine*

**Co-chair:** Scott Pomeroy, M.D., Ph.D., *Harvard Medical School, Children's Hospital, Boston*

This SIG will focus on recent discoveries in the genetic, epigenetic, and tumor micro-environmental oncogenic mechanisms relevant to the diagnosis and therapy (current and emerging) of adult and pediatric primary brain malignancies.

LEADER IN THE FIELD PRESENTATION:
3:30 – 3:50 PM
**GABA – A receptor in Medulloblastoma: Function, Intratumoral Drug Screening and Therapeutic Strategies**
Soma Sengupta, M.D., Ph.D., *Winship Cancer Institute, Emory University Hospital*

3:50 – 4:00 PM
**Q&A**

DATA BLITZ PRESENTATION:
4:00 – 4:08 PM
**MR Imaging Features Predict Survival and Molecular Profile in Diffuse Lower Grade Gliomas**
Hao Zhou, M.D., *Washington University*

4:08 – 4:10 PM
**Q&A**

LEADER IN THE FIELD PRESENTATION:
4:10 – 4:30 PM
**Epigenetic Drivers of Stemness and Tumor Propagating Capacity in Glioblastoma**
John Laterra, Ph.D., *Johns Hopkins Sidney Kimmel Comprehensive Cancer Center*

4:30 – 4:40 PM
**Q&A**

DATA BLITZ PRESENTATION:
4:40 – 4:48 PM
**Clinical Features and Prognostic Factors of 476 Patients with Spinal Astrocytoma: An Integrated Analysis from Multi-Institutional Data and the Literature**
Harrison Bai, M.D., *University of Pennsylvania*

4:48 – 4:50 PM
**Q&A**

LEADER IN THE FIELD PRESENTATION:
4:50 PM – 5:10 PM
**Evolving Clinical Paradigms in Metastatic Brain Tumors: Genomics as a Tool**
Priscilla Brastianos, M.D., *Harvard Medical School, Massachusetts General Hospital*

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**5:10 – 5:20 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**5:20 – 5:30 PM**
**Investigating Histone H3 Post-Translational Modifications Using Paraffin-Embedded Pediatric Glioma Tissue Samples**
Amanda Saratsis, M.D., *Northwestern University Feinberg School of Medicine*

**5:30 – 7:00 PM POSTER PRESENTATIONS & RECEPTION #1**
*Harborside Ballroom, 4th Floor*

**POSTER CATEGORIES**
**Cerebrovascular Disease**
**Epilepsy**
**Behavioral Neurology**
**Multiple Sclerosis**
**Movement Disorder**
**Neuro-Oncology**
**Neurocritical Care**

Full abstracts for all posters are available in the abstract supplement and mobile application.

# MONDAY, OCTOBER 17

**6:30 AM – 5:45 PM ANNUAL MEETING REGISTRATION**
*Harborside Foyer*

**7:00 – 9:00 AM BREAKFAST**
*Grand Ballroom Foyer, 3rd Floor*

## 7:30 – 9:00 AM FACULTY DEVELOPMENT COURSES

**OFF THE BEATEN TRACK - "ROADS LESS TRAVELED"**
**Early/Mid Career Level Faculty Development Course II**
*Grand Ballroom 9&10, 3rd Floor*

**Chair:** Amy Pruitt, M.D., *University of Pennsylvania, Perelman School of Medicine*

**Co-Chair:** David Greer, M.D., M.A., F.C.C.M., F.A.H.A., F.N.C.S., F.A.A.N., *Yale School of Medicine*

**Faculty:** Farrah Matteen, M.D., Ph.D., *Massachusetts General Hospital and Harvard Medical School*
Edward Manno, M.D., M.S., *Cleveland Clinic*
Babar Khokhar, M.D., M.B.A., *Yale School of Medicine*

This course is designed to benefit those in the early to mid-stages of their career.

**TELENEUROLOGY IN ACADEMIC PRACTICE**
**AUPN Chair Level Faculty Development Course II**
*Grand Ballroom 7&8, 3rd Floor*

**Chair:** L. John Greenfield, Jr., M.D., Ph.D., *University of Arkansas for Medical Sciences*

**Faculty:** E. Ray Dorsey, M.D., M.B.A., *University of Rochester Medical Center*
Lawrence Wechsler, M.D., *University of Pittsburgh School of Medicine*

This course is designed to benefit those in Chair positions or on a trajectory to advance into Chair positions.

**9:00 – 9:15 AM COFFEE BREAK**
*Grand Ballroom Foyer, 3rd Floor*

## 9:15 – 11:15 AM SYMPOSIUM: BEYOND THE GENOME: TOWARD PRECISION MEDICINE IN NEUROLOGY

*Grand Ballroom 5&6, 3rd Floor*

**Chair:** Craig M. Powell, M.D., Ph.D., *University of Texas, Southwestern*

**Co-Chair:** Henry Paulson, M.D., Ph.D., *University of Michigan Health System*

This symposium will address advances in human genetics, genomics, and gene therapy for neurological disease. Currently, therapeutics for many neurological disorders are supportive or symptomatic rather than curative or restorative. In the "post-genome" era, neurologists have an

# PROGRAM

unrivaled opportunity to mine the new genetic information to better understand and identify novel ways to treat neurological disorders such as autism spectrum disorder, Alzheimer's disease and related dementias, ALS and other neuromuscular diseases. In this session, four leading experts in precision medicine and genome science will describe new insights into disease mechanisms, advances in diagnosis, and routes to disease-modifying therapy for a range of neurological diseases.

The Raymond D. Adams Lectureship will be presented in this symposium. This lectureship was established in 2000 to honor Dr. Raymond D. Adams, emeritus Bullard Professor of Neurology at Harvard Medical School and emeritus Chief of the Neurology Service at the Massachusetts General Hospital. An ANA member at the annual meeting presents the lecture.

**Learning Objectives:**

1. To define and understand Precision Medicine as it related to Neurology.
2. To understand the role of genetics, genomics, and related approaches to neurologic disorders.
3. To understand gene therapy approaches to treating neurologic disorders.
4. To provide several examples of neurologic disorders to which precision medicine, genomics, and gene therapies may be applied

**9:20 – 9:45 AM**
**Raymond D. Adams Lecture**
**Genetics and Integrative Genomics in Human Neuropsychiatric Disease**
Daniel Geschwind, M.D., Ph.D., *University of California, Los Angeles and David Geffen School of Medicine*

**9:45 – 10:10 AM**
**The Expanding RNA World Reveals New Perspectives on Neuromuscular Diseases**
Maurice Swanson, M.S., Ph.D., *University of Florida*

**10:10 - 10:35 AM**
**Beyond Reasonable Doubt: Proving Pathogenicity for ALS Gene Mutations**
Christopher Shaw, MBCh.B., M.D., F.R.A.C.P., F.R.C.P., F.Med.Sci., *Kings College, London*

**10:35 - 11:00 AM**
**Beyond the genome: Insights into the causes of neurodevelopmental disorders**
Evan Eichler, Ph.D., *University of Washington and Howard Hughes Medical Institute*

**DATA BLITZ PRESENTATIONS:**

**11:00 – 11:02 AM**
**Knockdown of a SMN-Associated Long Non-Coding RNA as a Novel Therapeutic Strategy for SMA**
Charlotte Sumner, M.D., *Johns Hopkins School of Medicine*

**11:03 – 11:05 AM**
**Vitamin D Genetic Risk Score Is Strongly Associated with Vitamin D Levels and Relapse Rate in Pediatric MS Patients**
Jennifer Graves, M.D., *University of California, San Francisco*

**11:06 – 11:08 AM**
**Rare Recurrent NRXN1 Deletions and CNTN6 Duplications Increase Risk for Tourette Syndrome**
Jeremiah Scharf, M.D., Ph.D., *Massachusetts General Hospital*

**11:09 – 11:11 AM**
**Molecular-Based Diagnosis of Multiple Sclerosis and Its Progressive Stage**
Peter Kosa, Ph.D., *National Institutes of Health*

**11:12 – 11:15 AM**
**Mechanism of Cytoplasmic Protein Aggregation and Neurodegeneration in Drosophila Models of C9-ALS/FTD**
Thomas Lloyd, M.D., Ph.D., *Johns Hopkins University School of Medicine*

## 11:15 – 11:45 AM EXECUTIVE SESSION OF MEMBERSHIP*
*Grand Ballroom 5&6, 3rd Floor*

## 11:00 AM – 7:00 PM POSTER VIEWING
*Harborside Ballroom, 4th Floor*

*Poster presenters will be in attendance from 5:30 – 7:00 pm*

## 11:30 AM – 1:00 PM LUNCH
*Grand Ballroom Foyer, 3rd Floor*

## 11:45 AM – 1:00 PM INTERACTIVE LUNCH WORKSHOPS

*These workshops are "lunch and learns"*

### 1: CELL- BASED THERAPIES FOR MULTIPLE SCLEROSIS
*Dover A/B, 3rd Floor*

**Moderator:** Peter Calabresi, M.D., *Johns Hopkins School of Medicine*

**Faculty:** Michael Racke, M.D., *Ohio State University,* Andrew Goodman, M.D., *University of Rochester*

An overview of various cellular therapies being developed for multiple sclerosis. Mesenchymal stems, hematopoetic stem cells, and oligodendrocyte precursor cells are being developed as potential treatments for MS. Presenters will provide overviews of each of these treatment strategies and where the field currently stands.

### 2. WHITHER NEUROREHABILITATION?
*Kent A/B/C, 4th Floor*

**Moderator:** George Wittenberg, M.D., Ph.D., F.A.S.N.R., *University of Maryland Medical Center*

**Co-Moderator:** John Krakauer, M.D., *Johns Hopkins University*

The field of neurorehabilitation is undergoing change, particularly for neurologists who practice clinically within it, and who carry out research programs. There has been reorganization within funding agencies, a number of large clinical trials that failed to show superiority for technologically advanced methods, and suspension of the UCNS certification. Yet there is a growth in technologies and knowledge about the brain mechanisms of recovery. The session will be devoted to discussing the threats and opportunities to the field as it impacts patients with neurorehabilitation needs, neurologists with clinical focus in this area, and research in the field.

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for **AMA PRA Category 1 Credits**™.*

### 3. SEIZURES AND NEUROPATHOLOGY: NIH- SUPPORTED OPPORTUNITIES FOR TESTING CANDIDATE THERAPEUTICS

*Grand Ballroom 1&2, 3rd Floor*

**Moderator:** David Jett, Ph.D., *National Institutes of Health, NINDS*

**Faculty:** Brian Klein, Ph.D., *National Institutes of Health, NINDS*
John Kehne, Ph.D., *National Institutes of Health, NINDS*
Shardell Spriggs, Ph.D., *National Institutes of Health, NINDS*

Seizures may have many causes, including brain trauma, epilepsy, and chemical toxins. One common feature of prolonged seizures, such as those seen in status epilepticus (SE), is damage to nerve cells within the brain. This neuropathology is clearly evident from animal studies of SE and toxic chemical exposures. The National Institutes of Health supports two program that allow investigators to screen candidate therapeutics for anticonvulsant activity, and compounds that reduce the neuropathology associated with prolonged seizures. The first program is the Countermeasures Against Chemical Threats Neurotherapeutics Screening (CNS) program, which uses animal models of organophosphorus pesticide and chemical warfare agent exposure to test compounds submitted to NIH for their anticonvulsant and neuroprotectant properties. The second program is the NINDS Anticonvulsant Screening Program that is well-known within the epilepsy community and has over 30 years of experience screening for these types of candidate therapeutics. This interactive session will describe the programs and opportunities for collaboration with the NIH.

### 4: SPECIAL TOPICS IN RESIDENCY EDUCATION

*Grand Ballroom 3&4, 3rd Floor*

**Moderator:** Teresa Jacobs, M.D. *University of Michigan*

**Co-Moderator:** Devin Brown, M.D., *University of Michigan*

**Faculty:** James Burke, M.D., Ph.D., *University of Michigan*
Lori Schuh, M.D., *Spectrum Health*

This session examines aspects of neurology education as they impact the work of the attending neurologist and the resident as a learner.

### 5: ADVANCES IN UNDERSTANDING AND TREATMENT OF MITOCHONDRIAL DISORDERS AFFECTING THE NERVOUS SYSTEM

*Grand Ballroom 7&8, 3rd Floor*

**Moderator:** Ricardo Roda, M.D., Ph.D., *Johns Hopkins University*

**Faculty:** Andrea Gropman, M.D., *Children's National Medical Center/ George Washington University*
Michio Hirano, M.D., *Columbia University Medical Center*
William Copeland, Ph.D., *National Institutes of Health, NIEHS*
Hiromi Sesaki, Ph.D., *Johns Hopkins University*
Antonio Toscano, M.D., *University of Messina*

### 6: MEET THE NEUROLOGY DEPARTMENT CHAIRS*

*Dover C, 3rd Floor*

**Faculty:** Joseph Biller, M.D., *Loyola University Chicago, Stritch School of Medicine*
Allison Brashear, M.D., *Wake Forest School of Medicine*
David Holtzman, M.D., *Washington University School of Medicine in St. Louis*
Frances Jensen, M.D., *Penn Medicine*

Prominent chairs of neurology will discuss how they handle their position, including what's involved with being a chair; what the process is for attaining their position, and how to interact with chairs.

## 11:45 AM – 1:00 PM ABPN MOC PROGRAM: LIFELONG LEARNING FOR NEUROLOGISTS*

*Laurel A&B, 4th Floor*

**Faculty:** Larry Faulkner, M.D., *President and CEO at ABPN*

Dr. Faulkner will lead the session by providing background on the ABMS MOC program, recent changes to the MOC program, and perspective on the future of MOC. Dr. Faulkner will detail the four-part ABPN MOC Program, giving specific requirements related to self-assessment, CME, and performance in practice components.

**Educational Objectives for this session:**
- To become familiar with the rationale and background of MOC
- To learn the 4-part ABPN MOC program components.

## 11:45 AM – 1:00 PM 16TH ANNUAL WOMEN OF THE ANA LUNCH PROGRAM

### UNCOVERING AND MANAGING UNCONSCIOUS BIAS IN ACADEMIC MEDICINE—WOMEN IN LEADERSHIP

*Waterview Ballroom, Lobby Level*

**Co-Chairs:** Kathleen Digre, M.D., *University of Utah*
Karen C. Johnston, M.D., MSc, *University of Virginia*
Chiquita Collins, Ph.D., *Johns Hopkins School of Medicine*

Examine your own biases, and Dr. Chiquita Collins will guide our discussion about recognizing and guarding against unconscious bias. Everyone is welcome.

## 1:15 PM–3:15 PM PRESIDENTIAL SYMPOSIUM: THE ROLE OF ACADEMIC NEUROLOGY IN IMPROVING HEALTH

*Grand Ballroom 5&6, 3rd Floor*

**Chair:** Barbara G. Vickrey, M.D., M.P.H., *Icahn School of Medicine at Mount Sinai*

**Co-Chair:** Kevin A. Kerber, M.D., M.S., *University of Michigan*

The speakers comprising this program are all leaders in scientific research that focuses on improving health at the population level. Dr. Morgenstern conducts research on causes and mechanisms to eliminate racial/ethnic disparities in stroke. Dr. Vickrey's research aims to generate evidence on ways to improve population health by designing and testing the impact of coordinated, team care models for dementia, Parkinson's disease, and stroke prevention. Dr. Dorsey studies using new technology in Parkinson's disease research and care. Dr. Johnston is Dean of a new medical school, whose stated mission is "improving health in our community as a model for the nation."

The Soriano Lectureship will be presented in this symposium. The first Soriano lecture was given in 1987 which marked the 40th year of consecutive attendance at ANA meetings by Victor Soriano and his wife. The couple chose to sponsor a lectureship to be given at the ANA, so that in future years the Sorianos "would always be linked to all of you, through a brilliant lecture delivered by an outstanding scientist…"

**Learning Objectives:**
1. Learners will have a better understanding of the role of health services research in realizing meaningful health outcomes in neurological care.
2. Learners will be better prepared to conceptualize, develop, and design research studies that aim to improve health for neurological conditions in communities.

# PROGRAM

3. Learners will have the knowledge to incorporate or partner in incorporating technological advances in care and research in academic neurology practices.

**1:15 – 1:20 PM**
**Distinguished Teacher Award**
David Gordon, M.D., F.A.A.N., F.A.N.A., F.A.H.A., *University of Oklahoma College of Medicine*

**1:20 – 1:45 PM**
**Research to Identify Neurologic Disease in Populations**
Lewis B. Morgenstern, M.D., *University of Michigan*

**1:50 -2:15 PM**
**Designing and Testing Interventions to Improve Neurologic Health Outcomes at the Population Level**
Barbara G. Vickrey, M.D., M.P.H., *Icahn School of Medicine at Mount Sinai*

**2:20 – 2:50 PM**
**Soriano Lecture**
**Placing Neurology at the Lead of Better Health**
S. Claiborne Johnston, M.D., Ph.D., M.P.H., *Dell Medical School at the University of Texas*

**2:50 – 3:15 PM**
**The Future of Teleneurology**
E. Ray Dorsey, M.D., M.B.A., *University of Rochester*

## 3:15 – 3:30 PM COFFEE BREAK
*Grand Ballroom Foyer, 3rd Floor*

## 3:30 – 5:30 PM SPECIAL INTEREST GROUP SYMPOSIA

### AUTOIMMUNE NEUROLOGY
*Dover A/B, 3rd Floor*

**Chair:** Jeffrey Gelfand, M.D., M.A.S., *University of California, San Francisco* and Stacey Clardy, M.D., Ph.D., *University of Utah*

The topics that will be presented in this session form part of a recently developed field of neurology. This session will discuss the diagnosis and treatment of autoimmune CNS and PNS disorders, improving the diagnosis and treatment of myelitis, and explain emerging insights about pathophysiology of CNS autoimmune disorders.

**LEADER IN THE FIELD PRESENTATION:**
**3:30 - 3:50 PM**
**Update on Autoimmune Encephalitis and Paraneoplastic Neurological Disorders of the CNS and PNS**
Maarten Titulaer, M.D.,Ph.D., *Erasmus Medical Center*

**3:50 – 4:00 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**4:00 – 4:10 PM**
**Pathological Mechanisms of Glycine Receptor Antibodies**
Sarah Crisp, M.B., B.C.H.I.R., Ph.D., *Institute of Neurology, University College London*

**LEADER IN THE FIELD PRESENTATION:**
**4:10 – 4:30 PM**
**Primary Immunodeficiencies and Disorders of Immune Dysregulation: Implications for Understanding CNS Autoimmunity**
Raphaela Goldbach-Mansky, M.D., M.H.S., *National Institutes of Health, NIAMS*

**4:30 – 4:40 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**4:40 – 4:50 PM**
**Treatment of Progressive Multifocal Leukoencephalopathy with Pembrolizumab, a PD-1 Inhibitor**
Irene Cortese, M.D., *NINDS*

**LEADER IN THE FIELD PRESENTATION:**
**4:50 – 5:10 PM**
**Updates in Myelitis**
Eoin P. Flanagan, M.B., B.Ch., *Mayo Clinic*

**5:10 – 5:20 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**5:20 – 5:30 PM**
**Tocilizumab Is Effective in Autoimmune Limbic Encephalitis Refractory to Weekly Rituximab**
Jung-Ah Lim, M.D., *Seoul Natural University Hospital*

### CASE-BASED LESSONS IN MEDICAL NEUROLOGY: THE INTERFACE BETWEEN NEUROLOGY AND INTERNAL MEDICINE
*Dover C, 3rd Floor*

**Chair:** Martin Samuels, M.D., DSc(hon), F.A.A.N., M.A.C.P., F.R.C.P., F.A.N.A., *Brigham and Women's Hospital, Harvard Medical School*

**Co-Chair:** S. Andrew Josephson, M.D., *University of California, San Francisco*

This session will be entirely case-based. The real case summaries are taken from selected patients seen on a busy consultation service. They will be presented as unknowns to the audience, including the relevant history, examination, imaging and laboratory studies. Attendees will be encouraged to participate in the case discussions. For each case, salient lessons will be gleaned and sources of possible errors reviewed.

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for **AMA PRA Category 1 Credits**™.*

## DEMENTIA AND AGING

*Kent A/B/C, 4th Floor*

**Chair:** Dena Dubal, M.D., Ph.D., *University of California, San Francisco*

**Co-Chair:** Erik Roberson, M.D., Ph.D., *University of Alabama at Birmingham*

This SIG will provide updates on three highly topical issues in age-related dementia from three leaders in the field. Topics include recent advances in tau PET imaging and applications in Alzheimer's disease and non-Alzheimer dementias, as well as new insights into the mechanisms by which the C9ORF72 expansion causes neurodegeneration in FTD and ALS, and novel genetic insights into Alzheimer's disease. Limitations of genome-wide association studies (GWAS) will be discussed, and complementary approaches that can strengthen or expand upon GWAS findings will be explored. In addition, three submitted abstracts complementing these themes have been chosen for brief presentations.

LEADER IN THE FIELD PRESENTATION:
**3:30 - 3:50 PM**
**Tau PET: Progress and Challenges**
Gil Rabinovici, M.D., *University of California, San Francisco*

**3:50 – 4:00 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:00 – 4:10 PM**
**Preclinical Studies Targeting Toxic Tau Oligomers by Immunotherapy**
Rakez Kayed, Ph.D., *University of Texas*

LEADER IN THE FIELD PRESENTATION:
**4:10 – 4:30 PM**
**A Molecular Network Approach to Alzheimer's Disease Uncovers New Astrocyte Biology and Therapeutic Targets**
Phil DeJager, M.D., Ph.D., *Brigham and Women's Hospital*

**4:30 – 4:40 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:40 – 4:50 PM**
**RNA Destabilization in Amyotrophic Lateral Sclerosis and Frontotemporal Lobar Dementia**
Sami Barmada, M.D., Ph.D., *University of Michigan*

LEADER IN THE FIELD PRESENTATION:
**4:50 – 5:10 PM**
**A Small Molecule Strategy to Inhibit Development of Pathological Forms of Tau: In Vitro Studies to Human Trials**
Frank Longo, M.D., Ph.D., *Stanford University*

**5:10 – 5:20 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**5:20 – 5:30 PM**
**Characterization of Molecular Mechanism(s) Underlying ApoE4-Induced Brain Phospholipid Dysregulation in Alzheimer's Disease**
Dongming Cai, M.D., Ph.D., *Icahn School of Medicine at Mount Sinai*

## EDUCATION: THE ROLE OF QUALITY AND SAFETY IN NEUROLOGIC TRAINING

*Grand Ballroom 1&2, 3rd Floor*

**Chair:** Steven L. Lewis, M.D., *Rush University Medical Center*

Since the Institute of Medicine report "To Err is Human", quality and safety has been a hot topic in medicine, and there has been increasing involvement by neurologic training programs and trainees in assessing and (hopefully) improving quality of neurologic care and patient safety. This year's education SIG will discuss the current stance of our regulatory bodies with regard to training residents and fellows in quality and safety; review specifics of integration of quality and safety education in neurology training programs; discuss how to leverage institutional resources for quality and safety projects in residency; and discuss the role of the electronic health record in teaching quality and safety.

LEADER IN THE FIELD PRESENTATION:
**3:30 – 3:45 PM**
**Introduction**
Tracey Cho, M.D., *Massachusetts General Hospital*

LEADER IN THE FIELD PRESENTATION:
**3:45 – 4:00 PM**
**The ACGME and Quality and Safety**
Steven Lewis, M.D., *Rush University Medical Center*

LEADER IN THE FIELD PRESENTATION:
**4:00 – 4:15 PM**
**Quality and Safety in Residency Training**
Adam Webb, M.D., *Emory University School of Medicine*

LEADER IN THE FIELD PRESENTATION:
**4:15 – 4:30 PM**
**Leveraging Institutional Resources for Quality and Safety Projects in Neurology Residency**
Kathleen McKee, M.D., *Partners Neurology/Massachusetts General Hospital and Brigham and Women's Hospital*

LEADER IN THE FIELD PRESENTATION:
**4:30 – 4:45 PM**
**The Role of the Electronic Health Record in Teaching Quality and Safety**
Allison Weathers, M.D., *Rush University Medical Center*

**4:45 – 5:30 PM**
**Panel – Audience Discussion**

## HEADACHE AND PAIN

*Grand Ballroom 3&4, 3rd Floor*

**Chair:** Peter Goadsby, M.D., Ph.D., *University of California, San Francisco*

**Co-Chair:** K.C. Brennan, M.D., *University of Utah*

Headache, and migraine in particular, is singularly the most common neurological condition, and the treatment of the primary headache disorders is a major role for neurologists regardless of subspecialty. This SIG will provide an update on the increasingly complex and rapidly evolving medication and procedure options for migraine, and will provide a roadmap to the latest research that provides key insights into the understanding and treatment of headache.

# PROGRAM

LEADER IN THE FIELD PRESENTATION:
**3:30 – 4:00 PM**
**Effects of Obesity on the Trigeminal System: Preclinical Models**
Ana Recober-Montilla, M.D., *University of Pennsylvania*

**4:00 – 4:10 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:10 – 4:40 PM**
**Spreading Depolarizations in Migraine and Beyond**
KC Brennan, M.D., *University of Utah*

**4:40 – 4:50 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:50 – 5:20 PM**
**Spider Toxins Implicate Nav1.1 in Mechanical Pain**
Frank Bosmans, PharmD, Ph.D., *Johns Hopkins University School of Medicine*

**5:20 – 5:30 PM**
**Q&A**

## HEALTH SERVICES RESEARCH
*Grand Ballroom 7&8, 3rd Floor*

**Chair:** Lesli Skolarus, M.D., M.S., *University of Michigan Health System*

**Co-Chair:** Allison Willis, M.D., M.S., *University of Pennsylvania*

The topic of this session is research priorities and opportunities in health services research. Three health services research experts will separately present on their vision for research priorities in health services research in Neurology, crosscountry health care comparisons and designing stroke systems of care. Attendees will then have the opportunity to ask questions of this distinguished panel.

LEADER IN THE FIELD PRESENTATION:
**3:30 – 3:50 PM**
**International Comparisons in Healthcare: Utilization, Value, & Structure**
Kevin Kerber, M.D., M.S., *University of Michigan*

**3:50 – 4:00 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:00 – 4:08 PM**
**Using Telemedicine to Improve Access to Specialty Care for Underserved Patients with Neurodevelopmental Disorders**
Deepa Menon, M.D., *Kennedy Krieger Institute*

**4:08 – 4:10 PM**
**Q&A**

LEADER IN THE FIELD PRESENTATION:
**4:10 – 4:30 PM**
**Value-Based Healthcare Delivery in Geriatric Neurology**
Lidia Moura, M.D., MPH, *Masschusetts General Hospital*

**4:30 – 4:40 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**4:40 – 4:50 PM**
**Does Headache Neuroimaging Reduce Time to Glioma Diagnosis? A Cohort Study Using a Comprehensive Claims-Based Database**
James Burke, M.D., M.S., *University of Michigan*

LEADER IN THE FIELD PRESENTATION:
**4:50 – 5:10 PM**
**Community Engagement, Learning Collaboratives, and EMS Routing Policies to Optimize Acute Stroke Care in Chicago**
Shyam Prabhakaran, M.D., M.S., *Northwestern University*

**5:10 – 5:20 PM**
**Q&A**

DATA BLITZ PRESENTATION:
**5:20 – 5:30 PM**
**Trends and Regional Patterns of Care Associated with Hospice for US Stroke Inpatients, 2002-2011**
Benjamin George, M.D., M.P.H., *University of Rochester School of Medicine and Dentistry*

## INTERVENTIONAL NEUROLOGY
*Grand Ballroom 9&10, 3rd Floor*

**Chair:** Joseph Broderick, M.D., *University of Cincinnati College of Medicine*

**Co-Chair:** Johanna Fifi, M.D., *Icahn School of Medicine at Mount Sinai*

Interventional Neurology is a new subspecialty for neurologists, and is focused on catheter-based minimally invasive diagnosis and therapy of mainly neurovascular disorders. The field is rapidly advancing with new therapeutic devices and biological therapies such as stem cells being added to the armamentarium of the interventional neurologist. Scientific data to facilitate evidence-based practice has also steadily accumulated for these new therapies. The SIG in interventional neurology will address advances in flow diverter stents for treatment of wide-neck cerebral aneurysms, acute stroke intervention for ischemic stroke, update the audience on results from the recently completed first US intra-arterial stem cell clinical trial for stroke and address the diagnosis and management of neurovascular dural arteriovenous fistulas. These topics will substantially address the practice gaps in keeping up with the recent advances in the field for all neurologists.

LEADER IN THE FIELD PRESENTATION:
**3:30 – 3:50 PM**
**Endovascular Therapy for Acute Ischemic Stroke: Unanswered Questions and the Trials That Will Answer Them**
Joseph Broderick, M.D., *University of Cincinnati College of Medicine*

**Q&A**
**3:50 – 4:00 PM**

DATA BLITZ PRESENTATION:
**4:00 – 4:08 PM**
**CT Angiography Delays Groin Puncture in Mechanical Thrombectomy for Large Vessel Occlusion in Stroke**
Kunakorn Atchaneeyasakul, M.D., *University of Miami Miller School of Medicine*

**4:08 – 4:10 PM**
**Q&A**

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for **AMA PRA Category 1 Credits**™.*

**LEADER IN THE FIELD PRESENTATION:**
**4:10 – 4:30 PM**
**Endovascular Devices for Treatment of Intracranial Aneurysms: How Do They Work and Do They Improve Outcomes**
Johanna Fifi, M.D., *Icahn School of Medicine at Mount Sinai*

**4:30 – 4:40 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**4:40 – 4:48 PM**
**Reasons for Failed Mechanical Thrombectomy: Initial Data from Two Tertiary Care Centers**
Kaitlin Seibert, B.A., *The University of Toledo College of Medicine and Life Sciences*

**4:48 – 4:50 PM**
**Q&A**

**PANEL:**
**4:50 – 5:10 PM**
**Challenging Stroke Interventional Cases – What Would You Do?**
Panel

**5:10- 5:20 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**5:20 – 5:28 PM**
**Development of a Cerebral Angiography Calibration Model Based off of Relative Arterial Diameters**
Maximilian Bazil, *Icahn School of Medicine at Mount Sinai*

**Q&A**
**5:28 – 5:30 PM**

## NEUROMUSCULAR DISEASE

*Essex A/B, 4th Floor*

**Chair:** Laurie Gutman, M.D., *University of Iowa Hospitals and Clinics*

**Co-Chair:** Eric Sorenson, M.D., *Mayo Clinic*

This session will cover the latest in research, and therapeutics in key areas of Neuromuscular Diseases. It includes"senior" leader talks on ALS and other motor neuron diseases, Muscular Dystrophies and other myopathies and Peripheral Nerve disorders.

**LEADER IN THE FIELD PRESENTATION:**
**3:30 – 3:50 PM**
**Autonomic Disorders**
Amanda Peltier, M.D., *Vanderbilt University*

**3:50 – 4:00 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**4:00 – 4:08 PM**
**Microglial, pTDP43, and Neuronal Pathologies in the Brainstem and Spinal Cord of ALS Patients**
Matthew Cykowski, M.D., *Houston Methodist Hospital*

**4:08 – 4:10 PM**
**Q&A**

**LEADER IN THE FIELD PRESENTATION:**
**4:10 – 4:30 PM**
**Motor Neuron Diseases**
Nicholas Maragakis, M.D., *Johns Hopkins University*

**Q&A**
**4:30 – 4:40 PM**

**DATA BLITZ PRESENTATION:**
**4:40 – 4:48 PM**
**CAT-1004, an Oral Agent Targeting NF-kB: MoveDMD Trial Results in Duchenne Muscular Dystrophy (DMD)**
Joanne Donovan, M.D., Ph.D., *Catabasis Pharmaceuticals*

**4:48 – 4:50 PM**
**Q&A**

**LEADER IN THE FIELD PRESENTATION:**
**4:50 – 5:10 PM**
**Myasthenia Gravis**
Angela Vincent, M.D., *Somerville College of Oxford University*

**5:10 – 5:20 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**5:20 – 5:28 PM**
**Knockdown of SMN-Associated Long Non-Coding RNA as a Novel Therapeutic Strategy for SMA**
Charlotte Sumner, M.D., *Johns Hopkins School of Medicine*

**5:28 – 5:30 PM**
**Q&A**

## SLEEP DISORDERS AND CIRCADIAN RHYTHM

*Essex C, 4th Floor*

**Chair:** Louis Ptacek, M.D., *University of California, San Francisco*

**Co-Chair:** Phyllis Zee, M.D., Ph.D., *Northwestern University, Feinberg School of Medicine*

Sleep and circadian rhythm disturbances are pervasive in patients with neurological disorders. Given the ubiquitous influence of sleep and circadian timing on nearly all molecular and physiological processes, research and clinical practice in neurology need to consider the influence of the circadian cycle and sleep/wake states on the phenotypic expression and treatment of neurological disorders. The purpose of this SIG is to promote broader understanding of sleep and circadian phenotypes and the implications hese have on health of the nervous system.

**LEADER IN THE FIELD PRESENTATION:**
**3:30 – 4:05 PM**
**Regulation of Sleep During Sickness**
David Raizen, M.D., Ph.D., D.A.B.S.M., *University of Pennsylvania*

# PROGRAM

**DATA BLITZ PRESENTATION:**
**4:05 – 4:13 PM**
**Hyperglycemia Is Associated with Autonomic Dysfunction During Sleep in Patients with OSA**
Amanda Peltier, M.D., M.S., *Vanderbilt University*

**4:13 – 4:15 PM**
**Q&A**

**DATA BLITZ PRESENTATION:**
**4:15 - 4:23 PM**
**Sleep Disturbances Induced by Gastroesophageal Reflux Disease (GERD) in the US General Population**
Maurice Ohayon, M.D., D.S.C., Ph.D., *Stanford University*

**4:23 – 4:25 PM**
**Q&A**

**LEADER IN THE FIELD PRESENTATION:**
**4:25 – 5:00 PM**
**Aging and Sleep: A Bidirectional Relationship?**
Phyllis Zee M.D., Ph.D., *Northwestern University Feinberg School of Medicine*

**DATA BLITZ PRESENTATION:**
**5:00 – 5:08 PM**
**Novel Application of Brain-Targeting Polyphenol Compounds in Promoting Resilience Against Sleep Deprivation-Induced Cognitive Dysfunction**
Giulio Pasinetti, M.D., Ph.D., *Icahn School of Medicine at Mount Sinai*

**5:08 – 5:10 PM**
**Q&A**

**LEADER IN THE FIELD PRESENTATION:**
**5:10 – 5:30 PM**
**JZP-110, a Dopamine Norepinephrine Reuptake Inhibitor (DNRI), with Robust Wake-Promoting Effects and Low Abuse Potential**
Michelle Baladi, Ph.D., *Jazz Pharmaceuticals*

## 5:30 – 7:00 PM POSTER PRESENTATIONS & RECEPTION #2

*Harborside Ballroom, 4th Floor*

### POSTER CATEGORIES

**Autoimmune Neurology**
**Dementia and Aging**
**Education**
**Headache and Pain**
**Health Services Research**
**Interventional Neurology**
**Neuromuscular Disease**
**Sleep Disorders and Circadian Rhythm Education**
**Traumatic Brain Injury**

Full abstracts for all posters are available in the abstract supplement and mobile application.

## 7:30 – 9:00 PM ANA PRESIDENT'S RECEPTION

*Grand Ballroom 5&6, 3rd Floor*

# TUESDAY, OCTOBER 18

## 6:30 AM – 2:00 PM ANNUAL MEETING REGISTRATION

*Harborside Foyer, 4th Floor*

## 7:00 – 9:00 AM BREAKFAST

*Grand Ballroom Foyer, 3rd Floor*

## 7:30 – 9:00 AM FACULTY DEVELOPMENT COURSES

*Grand Ballroom 9&10, 3rd Floor*

### THE VIEW FROM THE NIH AND SUCCESSFUL GRANT WRITING

**Early/Mid Career Level Faculty Development Course III**

**Chair:** Amy Pruitt, M.D., *University of Pennsylvania, Perelman School of Medicine*

**Co-Chair:** David Greer, M.D., M.A., F.C.C.M., F.A.H.A., F.N.C.S., F.A.A.N., *Yale School of Medicine*

**Faculty:** Walter Koroshetz, M.D., *National Institutes of Health, NINDS*
Justin McArthur, M.B.B.S., M.P.H., F.A.A.N., *Johns Hopkins Medicine*

This course is designed to benefit those in the early to mid-stages of their career.

### NEUROSCIENCE SERVICE LINES IN ACADEMIC NEUROLOGY

**AUPN Chair Career Level Faculty Development Course I**

*Grand Ballroom 7&8, 3rd Floor*

**Chair:** L. John Greenfield, Jr., M.D., Ph.D., *University of Arkansas for Medical Sciences*

**Faculty:** Frances Jensen, M.D., F.A.C.P., *University of Pennsylvania, Perelman School of Medicine*
Joseph Broderick, M.D., *University of Cincinnati College of Medicine*
Brett M. Kissela, M.D., M.S., *University of Cincinnati College of Medicine*

This course is designed to benefit those in Chair positions or on a trajectory to advance into Chair positions.

## 9:00 – 9:15 AM COFFEE BREAK

*Grand Ballroom Foyer, 3rd Floor*

## 9:15 – 11:15 AM SYMPOSIUM: THE SOCIAL-EMOTIONAL BRAIN: FROM NEUROBIOLOGY TO NEUROLOGICAL DISEASE

*Grand Ballroom 5&6, 3rd Floor*

**Chair:** William Seeley, M.D., *University of California, San Francisco*

**Co-Chair:** William T. Dauer, M.D., *University of Michigan*

**PLEASE NOTE:** *Any session that has an asterisk (*) next to the session title, is not available for **AMA PRA Category 1 Credits**™.*

**Faculty:** Joseph E. LeDoux, Ph.D., *New York University*
Larry Young, Ph.D., *Emory University School of Medicine*
Virginia Sturm, Ph.D., *University of California, San Francisco*
Karen F. Berman, M.D., *National Institutes of Health, NIMH Intramural Research Program*

This symposium will outline the anatomical and neurochemical foundations of mammalian social and emotional behavior. Speakers will illustrate key principles gained from studying model organisms and human disease states that impact social-emotional functioning.

**9:20 –9:45 AM**
**Coming to Terms with Fear and Anxiety**
Joseph E. LeDoux, Ph.D., *New York University*

**9:45 – 10:10 AM**
**The Neurobiology of Social Bonding and Empathy: Implications for Autism**
Larry Young, Ph.D., *Emory University School of Medicine*

**10:10 – 10:35 AM**
**Neural Mechanisms of Socioemotional Disruption in Frontotemporal Dementia**
Virginia Sturm, Ph.D., *University of California San Francisco*

**10:35 – 11:00 AM**
**Neurogenetic Mechanism in Williams Syndrome: Translating between Genes, BRain, and Complete Human Behavior**
Karen F. Berman, M.D., *National Institutes of Health, NIMH Intramural Research Program*

**DATA BLITZ PRESENTATIONS:**

**11:00 – 11:02 AM**
**Relationship Between Orthographic and Phonological Treatments with Brain Atrophy in PPA**
Andrea Faria, M.D., Ph.D., *Johns Hopkins University School of Medicine*

**11:03 - 11:05 AM**
**Genetic Deletion of Sarm1 Attenuates Vincristine-Induced Neuropathy in a Mouse Model**
Stefanie Geisler, M.D., *Washington University in St. Louis*

**11:06 – 11:08 AM**
**Electrostimulation Effects in lvPPA and nfvPPA**
Kimberly Webster, M.D., M.S., *Johns Hopkins University School of Medicine*

**11:09 – 11:11 AM**
**Cerebellar tDCS to Agument Chronic Aphasia Treatment**
Rajani Sebastian, Ph.D., *Johns Hopkins University School of Medicine*

**11:12 - 11:15 AM**
**Dissociated Cognitive Impairments Associated with Midbrain and Hippocampal Microstructural Atrophy in Parkinson's Disease: A 7-Tesla MRI Study**
Kathleen Poston, M.D., M.S., *Stanford University*

## 11:45 AM – 1:00 PM LUNCH

*Grand Ballroom Foyer, 3rd Floor*

## 11:45 AM – 1:00 PM INTERACTIVE LUNCH WORKSHOPS

*These workshops are "lunch and learns"*

### 1: IMPACT OF CLINICAL TRIALS ON NEUROLOGIC PRACTICE

*Dover A/B, 3rd Floor*

**Moderator:** Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins University School of Medicine*

**Co-Moderator:** Daniel Woo, M.D., M.S., *University of Cincinnati*

**Faculty:** Robert Holloway, M.D., M.P.H., *University of Rochester*
Seemant Chaturvedi, M.D., *University of Miami Miller School of Medicine*

This session will review examples of clinical trials where the translation into community practice has been appropriate, excessive, or inadequate. In addition, obstacles to adoption of clinical trial results will be discussed, including therapeutic inertia, nihilism, and financial conflicts of interest. Finally, potential solutions will be outlined for greater acceptance of clinical trials within the neurology community.

### 2. TOPICS AND CASES IN NEUROINFECTIOUS DISEASE

*Kent A/B/C, 4th Floor*

**Moderator:** Avindra Nath, M.D., *National Institutes of Health*

**Co-Moderator:** Micheline McCarthy, M.D., Ph.D., *Miller School of Medicine, University of Miami*

**Faculty:** Carlos Pardo-Villamizar, M.D., *Johns Hopkins School of Medicine*
Anil Panackal, M.D., *National Institutes of Health, NIAID*
Larry Davis, M.D., *University of New Mexico*
Avindra Nath, M.D., *National Institutes of Health*

There will be 4 presentations. The session will present 3 new or updated topics linking infections with neurological disease. Two speakers will present viral infection-related topics and a third speaker will present on fungal infection in the nervous system.

The 4th presentation will be a directed discussion of interesting or challenging cases submitted by workshop attendees to the 4th speaker.

### 3. REFRACTORY STATUS EPILEPTICUS: MECHANISMS AND MANAGEMENT

*Grand Ballroom 1&2, 3rd Floor*

**Moderator:** Thomas Bleck, M.D., FANA, MCCM, *Rush University Medical Center*

**Co-Moderator:** Tobias Loddenkemper, Ph.D., M.D., *Boston Children's Hospital*

**Faculty:** Jaideep Kapur, M.B.B.S., Ph.D, *University of Virginia*
Michael Rogawski, M.D., Ph.D., *University of California, Davis*

Advances in our understanding of the mechanisms of refractory and super-refractory status epilepticus are only beginning to be translated into therapies. This session will examine why status fails initial treatments, and explore some options for management.

### 4. NEUROSARCOID: THE GREAT IMITATOR

*Dover C, 3rd Floor*

**Moderator:** Beau Ances, M.D., *Washington University in Saint Louis*

**Co-Moderator:** Walter Royal III, M.D., *University of Maryland*

**Faculty:** Barney Stern, M.D., *University of Maryland Baltimore*
David Clifford, M.D., *Washington University School of Medicine*

# PROGRAM

This session will look at neurosarcoid pathophysiology , neuroimaging, and potential treatment for neursarcoid. Often a number of neurologists see these kind of patients but are unsure if the diagnosis is correct and what treatment to provide. This session will help inform neurologists for assisting in the treatment of these complicated patients.

### 5: MEET THE NINDS*

*Grand Ballroom 3&4, 3rd Floor*

**Moderator:** Walter Koroshetz, M.D., *National Institute of Neurological Disorders and Stroke (NINDS)*

**Faculty:** Francesca Bosetti, Pharm.D., Ph.D.; Robin Conwit, M.D.; Adam Hartman, M.D.; Janet He, Ph.D.; Lyn Jakeman, Ph.D.; David Jett, Ph.D.; Beth-Ann Seiber, Ph.D.

This is your chance to get your questions answered by representatives from the National Institute of Neurological Disorders and Stroke (NINDS).

## 11:45 AM – 1:00 PM e-MENTORING PROGRAM LUNCH *

*Laurel B, 4th Floor*

Join the ANA President, Barbara G. Vickrey, M.D., M.P.H., *Chair of Neurology, Icahn School of Medicine at Mount Sinai*, and other members of the ANA Executive Board, Board of Directors, and E-Advising subcommittee, for a current status report of the new and improved e-Mentoring program. Attendees will have the opportunity to give input to the program design and implementation at the luncheon.

## 11:45 AM – 1:00 PM AUPN'S NETWORKING LUNCH FOR SMALL ACADEMIC DEPARTMENTS OF NEUROLOGY *

*Laurel A, 4th Floor*

**Moderator:** L. John Greenfield, Jr., M.D., Ph.D., *University of Arkansas for Medical Sciences*

This course is an opportunity in smaller academic departments of neurology to gather and learn best practices from shared experiences.

## 1:15 – 3:15 PM SYMPOSIUM: SELECTIVE NEURONAL DYSFUNCTION AND DEGENERATION

*Grand Ballroom 5&6, 3rd Floor*

**Chair:** Robert H. Brown, Jr., D.Phil, M.D., *University of Massachusetts Medical School*

**Co-Chair:** Margaret Elizabeth Ross, M.D., *Weil Cornell Medical College*

**Faculty:** Nicole Calakos, M.D., Ph.D., *Duke University School of Medicine*
William Dauer, M.D., *University of Michigan*
William Seeley, M.D., *University of California San Francisco*
D. James Surmeier, Ph.D., *Feinberg School of Medicine Northwestern University*

While neurodegeneration often involves final common pathways, the effects may occur first in particular neuronal populations, underlying the distinctive presentations of these disorders. The symposium will present current insights into the mechanisms leading to selective vulnerability of neuronal types in various neurodegenerative disorders of adults and children.

The F.E. Bennett Memorial Lecture will be presented in this symposium. Foster Elting Bennett, MD, established a lectureship in 1979 in the memory of his son. This award, which has been given to outstanding researchers and educators in neurology, is not limited to members of the American Neurological Association.

**1:20 – 1:45 PM**
**F.E. Bennett Memorial Lecture**
**Basis for Selective Vulnerability in Parkinson's Disease**
James Surmeier, Ph.D., *Feinberg School of Medicine Northwestern University*

**1:45 – 2:10 PM**
**Selective Circuit Vulnerability Underlying Frontotemporal Dementias and Alzheimer's Disease** William Seeley, M.D., *University of California, San Francisco*

**2:10 – 2:35 PM**
**Specific Dircuits Involved in Obsessive-Compulsive Behavior**
Nicole Calakos, M.D., Ph.D., *Duke University School of Medicine*

**2:35 - 3:00 PM**
**Selective Neuronal Vulnerability in Movement Disorders and Dystonia**
William Dauer, M.D., *University of Michigan*

**DATA BLITZ PRESENTATIONS:**

**3:00 – 3:02 PM**
**Medullary Neuronal Loss and Alpha-Synuclein Burden in Multiple System Atrophy**
Elizabeth Coon, M.D., *Mayo Clinic*

**3:03 – 3:05 PM**
**A Novel Type of Autoimmune Encephalitis Associated with Neurexin-3Antibodies**
Eugenia Martinez-Hernandez, M.D., Ph.D., *University of Barcelona*

**3:06 – 3:08 PM**
**Deep Brain Stimulation in Early Stage Parkinson's Disease: Long-Term Motor Benefit Through 5 Years**
Mallory Hacker, Ph.D., *Vanderbilt University*

**3:09 – 3:11 PM**
**Phase II, Placebo-Controlled, Double-Blinded, Crossover Study of Extended-Release Dalfampridine in Monophasic Transverse Myelitis**
Michael Levy, M.D., Ph.D., *Johns Hopkins University*

**3:12 – 3:15 PM**
**Intrathecal Autologous Adipose-Derived MSC Treatment for ALS: Results of Phase I and Design of Phase II Clinical Trials**
Nathan Staff, M.D., Ph.D., *Mayo Clinic*

## 3:15 PM MEETING ADJOURNMENT

# SPEAKER ABSTRACTS

## SATURDAY SPEAKER ABSTRACTS

### NEUROSCIENCE OF CONSCIOUSNESS AND COMA SYMPOSIUM

#### Consciousness: From Theory to Practice

Melanie Boly, M.D., Ph.D., *University of Wisconsin School of Medicine and Public Health*

Behavioral reports have traditionally been the gold standard for evaluating the presence of consciousness. However, it is becoming clear that consciousness can be present even in the absence of overt behavior and in unresponsive subjects. I will present neurophysiological evidence supporting the presence of consciousness in dissociated states from several domains. Measures of cortical integration and differentiation have recently proven to be the most reliable marker of consciousness irrespective of behavior and have been validated in a large number of different conditions. Recent work using both within-state, no-task paradigms and TMS-EEG shows that consciousness can be present during non REM sleep when the front of the brain shows high amplitude slow waves, as long as a posterior cortical hot zone is activated. Studies using different anesthetics have also shown that fully unresponsive subjects anesthetized with ketamine (as compared to propofol or xenon) retrospectively report intense dreams, which are again associated with high complexity responses to TMS, despite the occurrence of slow waves. Finally, high complexity responses can also be observed in some patients in a vegetative state suggesting, in line with previous findings using active paradigms, that completely unresponsive patients may retain consciousness.

#### Deciphering the Dynamics of the Unconscious Brain Under General Anesthesia

Emery N. Brown, M.D., Ph.D., *Edward Hood Taplin Professor of Medical Engineering and Computational Neuroscience, Picower Institute for Learning and Memory, Department of Brain and Cognitive Sciences, Institute for Medical Engineering and Science, Massachusetts Institute of Technology*

Warren M. Zapol, Professor Anaesthesia, *Department of Anesthesia, Critical Care and Pain Medicine, Massachusetts General Hospital , Harvard Medical School*

Using electrophysiological recordings, mathematical modeling and neural signal processing techniques, we study how anesthetics induce unconsciousness. We present findings from our human studies of general anesthesia using high-density EEG and intracranial electrodes to record neural activity during loss and recovery of consciousness due to standard anesthetics. The brain under general anesthesia is not turned off but rather is dynamic.[1] We show that a primary mechanism through which anesthetics induce unconsciousness is by producing highly structured oscillations. The nature of the oscillations depends on the specific receptors which the anesthetic targets and the neural circuits in which the receptors are located. Anesthetics in the same class produce similar oscillations. These oscillations are readily visible on the scalp EEG.[2] We show that the anesthetic responses (EEG oscillations) change systematically as a function of age. We present a neuro-metabolic model of burst suppression, the profound state of brain inactivation seen in deep states of general anesthesia. We show that our characterization of burst suppression can be used to design a closed-loop anesthesia delivery system for control of a medically-induced coma. We demonstrate that the state of general anesthesia can be rapidly reversed by activating specific brain circuits.[3] Our results show that it is now possible to have a detailed neurophysiological understanding of the brain under general anesthesia, and that this understanding can be used to precisely monitor and control anesthetic states.

1. Purdon PL, Pierce ET, Mukamel EA, et al. Electroencephalogram signatures of loss and recovery of consciousness from propofol. Proc Natl Acad Sci U S A 2013;110:E1142-51.
2. Purdon PL, Sampson A, Pavone KJ, Brown EN. Clinical Electroencephalography for Anesthesiologists: Part I: Background and Basic Signatures. Anesthesiology 2015;123:937-60.
3. Solt K, Cotten JF, Cimenser A, Wong KF, Chemali JJ, Brown EN. Methylphenidate actively induces emergence from general anesthesia. Anesthesiology 2011;115:791-803.

#### Neural Correlates of Consciousness: Progress and Problems

Christof Koch, Ph.D., *CSO*

There have been a number of advances in the search for the neural correlates of consciousness — the minimum neural mechanisms sufficient for any one specific conscious percept – since this term was introduced by Crick and Koch (1990). In this Review, we describe recent findings showing that the anatomical neural correlates of consciousness are primarily localized to a posterior cortical hot zone that includes sensory areas, rather than to a fronto-parietal network involved in task monitoring and reporting. We also discuss some candidate neurophysiological markers of consciousness that have proved illusory, and measures of differentiation and integration of neural activity that offer more promising quantitative indices of consciousness.

**References**

1. Crick F & Koch C (1990) Towards a neurobiological theory of consciousness. Seminars in the Neuroscience 2: 263–275.
2. Koch C, Massimini M, Boly M & Tononi G (2016) Neural correlates of consciousness – progress and problems. Nature Reviews Neurosci. 17: 307-321.

## SUNDAY SPEAKER ABSTRACTS

### DEREK DENNY BROWN YOUNG NEUROLOGICAL SCHOLAR SYMPOSIUM

#### Emerging Roles of RNA in Stroke

Glen Jickling, M.D., *University of California Davis*

My research is dedicated to improving our understanding and ability to treat patients with stroke. I have a multipronged approach, including developing personalized molecular diagnostic tools to improve a physician's ability to diagnose stroke, and developing novel therapies to reduce the impact of stroke.

To improve the diagnosis and risk stratification of stroke I have evaluated and described RNA based biomarkers. To date these have been directed to aid with the diagnosis of stroke and TIA, to determine the cause of stroke, and to identify patients at risk for tPA related hemorrhagic transformation. Determining the diagnosis of stroke quickly permits rapid referral and triage of patients to hospitals specializing in the care of stroke. Faster time to treatment reduces the amount of brain damaged and thus improves patient outcomes. I have also developed RNA biomarkers to determine the cause of stroke. Preventing stroke is based on knowing the cause of stroke and treating it. Currently more than 35% of patients with stroke never have a cause identified despite extensive investigation. The RNA biomarkers I have described assign a cause of stroke to these patients, and thus may permit better implementation of stroke prevention therapy.

Protecting the brain from stroke injury is also an active are of my investigation. Following ischemic brain injury, the immune system is activated and responds to damaged brain tissue. Aspects of this response may contribute to brain injury in stroke. Whether modulating the immune system and the cellular response to ischemia to limit brain injury is an area I am currently investigation. I also have described the peripheral immune response that occurs in patients with ischemic stroke at the transcriptome level. I currently am evaluating several of these genes and associated pathways as potential novel treatment targets to modulate the immune system and reduce its damaging effects on the brain following stroke. This may lead to new therapies to reduce brain injury following stroke, and thus improve patient outcomes.

**References**

1. Jickling GC, Zhan X, Stamova B, Ander BP, Tian Y, Liu D, Sison SM, Verro P, Johnston SC, Sharp FR. Ischemic transient neurological events identified by immune response to cerebral ischemia. Stroke. 2012 Apr;43(4):1006-12.
2. Jickling GC, Xu H, Stamova B, Ander BP, Zhan X, Tian Y, Liu D, Turner RJ, Mesias M, Verro P, Khoury J, Jauch EC, Pancioli A, Broderick JP, Sharp FR. Signatures of cardioembolic and largevessel ischemic stroke. Ann Neurol. 2010 Nov;68(5):681-92
3. Jickling GC, Stamova B, Ander BP, Zhan X, Tian Y, Liu D, Xu H, Johnston SC, Verro P, Sharp FR. Profiles of lacunar and nonlacunar stroke. Ann Neurol. 2011 Sep;70(3):477-85.
4. Jickling GC, Ander BP, Stamova B, Zhan X, Liu D, Rothstein L, Verro P, Khoury J, Jauch EC, Pancioli AM, Broderick JP, Sharp FR. RNA in blood is altered prior to hemorrhagic transformation in ischemic stroke. Ann Neurol. 2013 Aug;74(2):232-40.
5. Jickling GC, Ander BP, Zhan X, Noblett D, Stamova B, Liu D. microRNA Expression in Peripheral Blood Cells following Acute Ischemic Stroke and Their Predicted Gene Targets. PLos One. 2014 Jun 9;9(6):e99283. PMID: 24911610

## Understanding the Role of C9orf72 in Neurodegeneration and Neuroinflammation

Robert Baloh, M.D., Ph.D., *Cedars-Sinai Medical Center*

The overall goal of my laboratory is to understand the molecular mechanisms of neuromuscular diseases using *in vitro* and animal modeling, based on insights from human genetics, to develop novel therapeutic agents. We are particularly interested in amyotrophic lateral sclerosis (ALS), and inherited neuropathy (Charcot-Marie-Tooth disease). The molecular pathways defined by genes mutated in hereditary neuromuscular diseases provide insight into molecular pathogenesis, and are potential candidates for therapeutic manipulation. Our current focus is on defining the molecular mechanism and therapeutic strategies for *C9orf72* related ALS and frontotemporal degeneration.

Amyotrophic lateral sclerosis (ALS) is an incurable neurodegenerative disease that affects motor neurons in the brain and spinal cord, and leads to death from respiratory failure within 3-5 years of onset. No effective treatments are available. Expansions of a hexanucleotide repeat in a noncoding region of the *C9orf72* gene account for ~40% of familial ALS cases, and are found in up to 10% of sporadic ALS patients. Our laboratory developed iPSC lines from 4 patients with *C9orf72* mutations, demonstrated that they have an in vitro disease phenotype including RNA foci, altered gene expression and electrophysiology, and showed that treatment with antisense oligonucleotides normalized phenotypic abnormalities. Subsequently we developed one of the first BAC transgenic mouse models of *C9orf72* related ALS, and demonstrated the therapeutic potential of ASOs in cells from these mice. Finally we recently demonstrated that myeloid cells including microglia express high levels of *C9orf72*, and that decreased *C9orf72* expression in patients may directly alter immune function and contribute to neurodegeneration. We continue to both examine the therapeutic utility of antisense oligonucleotides in *C9orf72* related ALS, and whether loss of *C9orf72* function synergizes with toxic protein aggregation to cause neurodegeneration

**References**

1. Sareen D, Gire-O'Rourke J, Muhammad AK, Grant S, Simpkinson M, Bell S, Carmona S, Ornelas L, Sahabian A, Gendron T, Petrucelli L, Baughn M, Ravits J, Harms MB, Rigo F, Bennett CF, Svendsen CN, Baloh RH. Targeting RNA Foci in iPSC-Derived Motor Neurons from ALS Patients with a C9ORF72 Repeat Expansion. Science Transl Med 23 October 2013. PMCID: PMC4090945
2. O'Rourke JG, Bogdanik L, Muhammad AKMG, Gendron TF, Kim KJ, Austin A, Cady J, Liu E, Zarrow J, Grant S, Ho R, Bell S, Daughrity L, Carmona S, Simpkinson M, Wu K, Dickson DW, Harms MB, Petrucelli L, Lee EB, Lutz CM, Baloh RH. C9orf72 BAC transgenic mice display typical pathologic features of ALS/FTD. Neuron, 2 December 2015.
3. O'Rourke JG, Bogdanik L, Yáñez A, Lall D, Wolf AJ, Muhammad AKMG, Ho R, Carmona S, Vit JP, Zarrow J, Kim KJ, Bell S, Harms MB, Miller TM, Dangler CA, Underhill DM, Goodridge HS, Lutz CM, Baloh RH. *C9orf72* is required for proper macrophage and microglial function in mice. Science, 2016 Mar 18;351(6279):1324-9.

## Cervical Dystonia: A Neural Integrator Disorder

Aasef Shaikh, M.D., Ph.D., *Case Western*

Medicalentereural integrators assure that eyes are held steady in straight-ahead and eccentric gaze positions. The abnormal role of the ocular motor neural integrator leads to centripetal drifts of the eyes resulting in gazeevoked nystagmus. About a decade ago a neural integrator, analogous to that in the ocular motor system, was proposed for the control of head movements. We recently identified a counterpart of gazeevoked eye nystagmus for head movements; where the head did not stay steady in eccentric positions on the trunk. The findings lead to a novel pathophysiological explanation in cervical dystonia. We proposed that the abnormalities of head movements in cervical dystonia originate in a malfunctioning head neural integrator, likely as a result of impaired cerebellar, basal ganglia, or peripheral feedback. After briefly recapitulating the history of the neural integrator for eye movements, this presentation will develop the idea of the head movement neural integrator. I will finally discuss the putative role of the head neural integrator in cervical dystonia. I hypothesize that changing the activity in an impaired head neural integrator, by modulating feedback, could treat dystonia.

**References:**

1. Shaikh AG, Zee DS, Crawford JD, Jinnah HA. Cervical dystonia: a neural integrator disorder. Brain. 2016 Jun 20. pii: aww141. [Epub ahead of print]
2. Shaikh AG, Zee DS, Jinnah HA. Oscillatory head movements in cervical dystonia: Dystonia, tremor, or both? Mov Disord. 2015 May;30(6):83442.
3. Shaikh AG, Wong AL, Zee DS, Jinnah HA. Keeping your head on target. J Neurosci. 2013 Jul 3;33(27):1128195.
4. Klier EM, Wang H, Constantin AG, Crawford JD. Midbrain control of threedimensional head orientation. Science. 2002 Feb 15;295(5558):13146.

## Genomic studies of developmental epilepsies identify importance of synaptic activation response pathways

Alex R. Paciorkowski, M.D., *University of Rochester Medical Center*

Recent genomic studies of individuals with severe early life epilepsies have identified pathogenic sequence variations in the kinase *SIK1*. Patients with *SIK1* syndrome have a spectrum of developmental epilepsies, including early myoclonic encephalopathy, Ohtahara

syndrome, and infantile spasms. *SIK1* acts to regulate the transcriptional activity of *MEF2C*, another causative gene for developmental epilepsies. Our studies have identified that epilepsy causing mutations in *SIK1* interfere with *MEF2C* transcriptional activity, and are associated with downstream abnormalities in *ARC*-related synaptic activation response as well as neuronal morphology. These findings highlight the ability of next-generation genomics to identify causative genes for developmental disorders, and the importance of synaptic activation response pathways in pathogenesis.

### Peripheral Neuropathic Changes in Pachyonychia Congenita

Michael Polydefkis, M.D., M.H.S., *Johns Hopkins University School of Medicine*

Pachyonychia congenita (PC) is an autosomal dominant keratoderma caused by a mutation in any one of the keratin genes *KRT6A, KRT6B, KRT6C, KRT16,* or *KRT17*. Plantar hyperkeratosis, thickened nails, oral plaques and intense pain (in the feet) are the distinct characteristics of PC [1]. Pressure on affected areas of soles is painful resulting in patients limiting their activity. Shaving PC-associated callus only partially alleviates pain, and 'over shaving' is also paradoxically painful. Little is known regarding the origin, nature or underling mechanisms of pain in these patients.

In the present study, we investigated the histopathology and distribution of cutaneous nerve fibers, subsets of epidermal nerve fibers, mechano-receptors, myelinated nerves, and blood vessels in patients with PC.

Plantar biopsies from 10 genetically confirmed PC patients (with a mutation in *KRT6A*) were performed at the ball of the foot (affected skin) and the arch (unaffected) and were compared to biopsies from corresponding locations in 10 control subjects. Tissue was processed to visualize intraepidermal nerve fibers (IENF) (PGP9.5), subsets of IENF (CGRP, SP, TH), myelinated nerve fiber (NFH), blood vessels (CD31), Meissner's corpuscles and Merkel cells (MC). Structures were quantified using stereology or validated quantification methods.

After adjusting for multiple comparisons, we observed that PC-affected plantar skin had significantly lower sweat gland innervation (SGNFD) and reduced numbers of Meissner's corpuscles compared to PC-unaffected or anatomically matched control skin. In contrast, Merkel cell densities, and blood vessel counts were higher in PC-affected skin compared to either control or PC-unaffected skin. There were no differences in myelinated nerve fiber densities, SP or CGRP between the groups. There were no differences between PC-unaffected and control subject skin in any of the markers studied. Pressure pain thresholds, a functional measure for Merkel cell function, were lower in PC-affected skin compared to PC unaffected and anatomically matched control skin. Additionally, MC densities in callused plantar skin from healthy runners with callus and one subject with a non-painful palmoplantar keratoderma (*AQP5* mutation) were similar to PC-unaffected and control skin consistent with callus alone not being sufficient to increase MC number.

MC-neurite complexes function as slowly adapting type I mechanoreceptors [2]. Their function involves Piezo2 mechanically gated ion channels expressed in both the MC and associated myelinated nerve fibers [3] and Piezo2 is required for MC mechanotransduction [4]. Piezo2 knockdown in MC reduces capsaicin-induced mechanical allodynia [5], suggesting that MC may play an important role in mechanical allodynia, and potentially explaining how increased MC densities contribute to pain in PC-affected skin. The observations that PC-affected skin contains alterations in peripheral nerve structures broadens the perception of PC as a dermatological condition to a multisystem disorder with neuropathic pain as a prominent feature. These observations open the door to novel treatment approaches and suggest that neuropathic pain treatments may have a role in PC.

**References:**

1. Eliason MJ, Leachman SA, Feng BJ, Schwartz ME, Hansen CD. A review of the clinical phenotype of 254 patients with genetically confirmed pachyonychia congenita. J Am Acad Dermatol 2012;67(4):680-686.
2. Iggo A, Muir AR. The structure and function of a slowly adapting touch corpuscle in hairy skin. J Physiol 1969;200(3):763-796.
3. Coste B, Mathur J, Schmidt M, Earley TJ, Ranade S, Petrus MJ, Dubin AE, Patapoutian A. Piezo1 and Piezo2 are essential components of distinct mechanically activated cation channels. Science 2010;330(6000):55-60.
4. Woo SH, Ranade S, Weyer AD, Dubin AE, Baba Y, Qiu Z, Petrus M, Miyamoto T, Reddy K, Lumpkin EA, Stucky CL, Patapoutian A. Piezo2 is required for Merkel-cell mechanotransduction. Nature 2014;509(7502):622-626.
5. Ikeda R, Cha M, Ling J, Jia Z, Coyle D, Gu JG. Merkel cells transduce and encode tactile stimuli to drive Abeta-afferent impulses. Cell 2014;157(3):664-675.

## RETHINKING THE BLOOD BRAIN BARRIER: BRAIN DRAINS AND INNATE IMMUNITY IN NEUROLOGIC DISEASE SYMPOSIUM

### CSF CLEARANCE AND EFFLUX PATHWAYS FROM BRAIN REVISITED

Helene Benveniste, M.D., Ph.D., *Stony Brook University School of Medicine*

Cerebrospinal fluid (CSF) is produced in the choroid plexus, brain/spinal cord parenchyma and offers buoyance and protection of the brain from mechanical damage. CSF also serves to provide a constant fluid flow via the ventricular system and through brain parenchyma. Specifically, CSF has been observed to circulate inside the ventricular system and onto the surface of the brain and spinal cord 1, 2. Under normal conditions the production of CSF and reabsorption of CSF into the general circulation is in equilibrium and a major driver of CSF transport is inspiratory respiratory effort2. Recent studies using optical imaging and intrathecal small molecular weight fluorescently tagged dyes showed that CSF circulates through brain parenchyma via a unique macroscopic system designated the 'glymphatic pathway'3. Key components of the glymphatic pathway include the peri-vascular space and aquaporin 4 water channels positioned on glial end-feet. CSF is convectively driven through the glymphatic pathway (and brain parenchyma) from the peri-arterial space, across AQP4 channels into the interstitial fluid space and is thought to exit into the central peri-venous spaces for ultimate exit via lymph vessels on the head/neck and general circulation3. A major role of CSF circulation is removal of waste products including soluble amyloid beta and tau protein. Our analysis of CSF circulation using contrast enhanced MRI4 suggests that parenchymal CSF access and circulation is largely dependent on altered states of arousal but not 'unconsciousness' per se. I will summarize our findings regarding the effect of various anesthetics on CSF transport of paramagnetic contrast agents in central nervous system and also share our findings on how CSF effluxes during altered states of consciousness where the glymphatic pathway is inaccessible. A major challenge will be to develop neuroimaging and computational approaches to translate these findings into live human brain.

**References:**

1. Battal B, Kocaoglu M, Bulakbasi N, Husmen G, Tuba Sanal H, Tayfun C. Cerebrospinal fluid flow imaging by using phase-contrast MR technique. The British journal of radiology. 2011 Aug;84(1004):758-65.
2. Dreha-Kulaczewski S, Joseph AA, Merboldt KD, Ludwig HC, Gartner J, Frahm J. Inspiration Is the Major Regulator of Human CSF Flow. J Neurosci. 2015 Feb 11;35(6):2485-91.
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## Modulating the BBB for the Enhanced Delivery of Therapeutics.

Lorraine Iacovitti, Ph.D., *Thomas Jefferson University*

The presence of the blood-brain barrier (BBB) in vertebrates is a major limitation to the delivery of therapeutic agents into brain tissue. Blood brain barrier integrity and active filtration of intra-vascular contents has long proved to be a significant barrier in the treatment of many disease states. Both chemotherapeutic and cellular therapies for disease states such as glioblastoma, Parkinson's disease, Alzheimer's disease, epilepsy and stroke have been limited by the presence of the BBB (1, 2). This barrier has required the use of invasive techniques such as direct injection into brain tissue, or potentially morbid techniques for BBB modulation such as high-dose mannitol administration. The development of a technique which demonstrates rapid onset of BBB opening and equally rapid reconstitution following discontinuation, with ease of repeatability would be enormously beneficial, facilitating the delivery of disease-modifying agents (various types of stem cells, viral vectors, etc.) directly to the region of interest. Furthermore, development of trans-vascular delivery with BBB modulation would obviate the need to inject or continually infuse agents directly into brain tissue, as is more commonly being investigated now.

The sphenopalatine ganglion (SPG) is the main source of parasympathetic input to brain blood vessels. Stimulation of the SPG causes vasodilation of brain vessels, and increases permeability of the BBB in the rat in a rapid and reversible fashion (3, 4). The therapeutic potential of this transient effect on the BBB is significant, but has not yet been demonstrated in the literature. We will discuss our recent studies on SPG stimulation and modulation of the BBB in rats with experimental stroke (middle cerebral artery occlusion; MCAO). We will show enhanced delivery of intra-arterially administered bone marrow mesenchymal stem cells and other therapeutics after SPG stimulation and discuss the possible underlying mechanisms and clinical implications of our results.

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## Implications of the Meningeal Lymphatic Pathways

Kari Alitalo, M.D., Ph.D. and collaborators, Wihuri Research Institute and Translational Cancer Biology Center of Program, Biomedicum Helsinki, 00014 University of Helsinki, Finland

Nearly all tissues in our bodies are highly vascularized by blood and lymphatic vessels. In peripheral organs, lymphatic drainage contributes to the regulation of interstitial fluid (ISF) homeostasis and immune surveillance by draining excess fluid, macromolecules and immune cells into lymph nodes (LNs) and systemic circulation (1). However, the mammalian central nervous system (CNS) parenchyma and its meningeal linings have mostly been considered devoid of a lymphatic vascular system. The early studies of Mascagni in 1787 were later refuted (2) and although "… the connection from the brain to the draining nodes..." was noted by Cserr and Knopf (3), the lymphatic vessel system inside the cranium was missed. This, and the presence of the blood-brain barrier were thus thought to make the CNS an immune privileged site. Cerebrospinal fluid (CSF) is mainly produced in the choroid plexus and has a similar composition as the ISF, which is dynamically exchanged with CSF and later on cleared from brain into subarachnoid space via the perivascular drainage ("glymphatic") system. CSF was suggested to be reabsorbed via arachnoid granulations directly into the bloodstream, as well as via cranial nerve sheaths into peripheral lymphatic drainage and further on into systemic circulation. However, previous research in this area has mostly studied CSF and ISF separately

Last year, we, and Dr. Jonathan Kipnis and collaborators, reported the surprising finding of a lymphatic vessel network in the dura mater of the mouse brain (4, 5). We showed that dural lymphatic vessels absorb brain ISF as well as CSF from the adjacent subarachnoid space and transport fluid and macromolecules into deep cervical LNs (dcLNs) via foramina at the base of the skull. In a transgenic mouse model expressing a VEGF-C/D trap and displaying complete aplasia of the dural lymphatic vessels, macromolecule clearance from the brain was attenuated and transport from the subarachnoid space into dcLNs was abrogated (4). Furthermore, surgical abruption of these vessels resulted in alteration of meningeal T-cell distribution (5). Surprisingly, brain ISF pressure and water content were unaffected (4).

Overall, these findings indicate that the mechanism of CSF flow into the dcLNs is directly via an adjacent dural lymphatic network, which may be important for the clearance of fluid, macromolecules and immune cells from the brain. Importantly, these results call for a reexamination of the role of the lymphatic system in CNS physiology and disease, particularly with respect to CNS immune privilege and macromolecule clearance. Further studies into meningeal lymphatic vessels may shed new light into the etiology of neurological diseases and provide novel treatment options for cerebral edema as well as different neurodegenerative and neuroinflammatory diseases, including Alzheimer's disease and multiple sclerosis.

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# MONDAY SPEAKER ABSTRACTS

## BEYOND THE GENOME: TOWARD PRECISION MEDICINE IN NEUROLOGY

### Beyond the Genome: Insights into the Causes of Neurodevelopmental Disorders

Evan Eichler, Ph.D., *University of Washington*

Autism is a devastating neurodevelopmental disorder that afflicts approximately 1% of live births. It is characterized by deficits in language, social interaction, and repetitive behaviors. I will summarize our findings regarding the discovery of genetic mutations and their contribution to autism spectrum disorder (ASD) and intellectual disability (ID). Our analysis of children with ASD/ID suggests that between 8-14% of disease is caused by inherited or *de novo* deletions and duplications of large segments of the genome involving multiple genes. I will present evidence from exome and molecular inversion probe sequencing of more than 10,000 children with simplex autism and show how these data may be used to pinpoint specific genes. The emerging data strongly argue that the development of the human brain is particularly sensitive to the timing and expression of many different genes; multiple genetic perturbations within specific neurodevelopmental pathways related to long-term potentiation, chromatin remodeling, and WNT signaling appear particularly important; and that the maternal and paternal contributions differ significantly. I will present data on how grouping patients based on a specific gene can be used to predict clinical subtypes of autism. Next-generation exome and genome sequencing data provide a powerful path forward for understanding the genetic architecture of these diseases but the heterogeneity demands an unprecedented level of global cooperation and networking. Going forward, a major challenge will be developing systems to understand the function of these neurocognition and neurobehavioral genes in both primate and nonhuman primate model organisms.

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### Beyond reasonable doubt: proving pathogenicity for ALS gene mutations

Christopher E Shaw, M.D., *Maurice Wohl Clinical Neuroscience Institute, King's College London*

A first draft of the Human Genome Mapping Project was published in 2000. It took 15 years and cost $3.3 billion to sequence one person's entire genetic code. Now this can be done in 1 week for around $1,000. The revolution in affordable genomics is already having a major impact with a proliferation of new ALS genes and provided important insights into disease mechanisms. This has helped families define their genetic risk and raised the prospect of gene-specific therapies but are all the ALS genes real and are all of the mutations in these genes pathogenic?

In this talk I will discuss our current practice for gene discovery and the bioinformatics and functional evaluation of potentially pathogenic gene variants. This validation process is essential if these discoveries are to influence diagnostic and predictive gene testing, pre-implantation genetic diagnosis and strategies for gene therapy.

### Genetics and Integrative Genomics in Human Neuropsychiatric Disease

Daniel H. Geschwind M.D.,Ph.D., *University of California, Los Angeles*

Advances in genetics and genomics have begun to deliver on their promise to expand our understanding of nervous system function in health and disease. One of the interesting challenges that perhaps paradoxically has emerged from these successes is an understanding of the profound genetic heterogeneity and complexity of most nervous system disorders. For example in Autism Spectrum Disorder (ASD), over a hundred of probable risk loci have been identified, none of which account for more than 1% of cases. This has led to what we consider to be a central question highly relevant to precision health approaches: will we have to treat each rare genetic form as a unique condition, or will there be convergence on a smaller number of shared pathways? I will highlight advances in the genetics of ASD and discuss integrative genomic approaches such as genome-wide transcriptional profiling that inform this important issue and that do suggest a substantial level of convergence. These studies provide a quantitative framework for functional investigation of disease mechanisms with a goal of accelerating therapeutic development.

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### The Expanding RNA World Reveals New Perspectives on Neuromuscular Diseases

Maurice Swanson, Ph.D., *University of Florida*
Jodi L. Bubenik, Łukasz J. Sznajder James D. Thomas, Ruan S. Oliveira, Marina M. Scotti and Maurice S. Swanson, *Department of Molecular Genetics and Microbiology, Center for NeuroGenetics, University of Florida*

The functional repertoire of RNA continues to grow including recent discoveries on surprising roles for noncoding, micro and circular RNAs in gene expression. Moreover, studies on repetitive sequences have also highlighted unusual effects of disease-associated mutations on more conventional RNAs. For example, the expansion of simple sequence repeats, or microsatellites, in the coding and noncoding regions of genes occurs in >40 neurological and neuromuscular diseases. For noncoding regions, expansions result in several dominantly inherited diseases, including myotonic dystrophy (DM) and ***C9orf72***-linked ALS/FTD, and current evidence indicates that these mutations are pathogenic at the RNA level. While the RNA-mediated pathogenesis model proposes that RNA expansions sequester RNA binding proteins (RBPs), these repeats are also translated into toxic peptides by repeat-associated non-AUG (RAN) translation. Here, we focus on DM as a model for RNA-mediated disease and discuss recent studies on mouse DM models together with human transcriptome analysis.

Research support provided by the NIH (NS058901, NS098819).

## PRESIDENTIAL SYMPOSIUM

### Research to Identify Neurologic Disease in Populations

Lewis Morgenstern, M.D., *University of Michigan*

It is important to understand neurologic disease in communities in order to make new discoveries, determine research priorities, and plan for ways to optimize neurologic health outcomes. Community-based research takes years to initiate and requires close collaboration with diverse members of society to achieve high quality results. We report the development and results of a community-based study of stroke, sleep disordered breathing, peripheral neuropathy and dizziness. The study illustrates what can be gained by careful observation of neurologic disease and therapy in a population that is not influenced by tertiary referral bias or selection into clinical trials. The data gathering is a crucial initial step for behavioral, medical and public health intervention studies to prevent and treat neurologic disease in this community and beyond.

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### Designing and Testing Interventions to Improve Neurologic Health Outcomes at the Population Level

Barbara G. Vickrey, M.D., M.P.H., *Icahn School of Medicine at Mount Sinai*

Health services research aims to complete the translation of basic science research into measurable improvements in population health. Investigators study mechanisms for translating evidence about what works in clinical care under optimal conditions, into actual practice settings. Patients in these settings have more heterogeneous clinical presentations than are typical of clinical trial participants and have broader social and economic contexts to their health and health care. The Chronic Care Model is a theoretical model of optimal chronic care delivery developed in the 1980s, and it has been the basis of a series of care coordination interventions that have been tested in randomized trials for neurological care, including dementia care management and others. Additional research has focused on understanding how to adapt and refine such models, so that – like efforts to develop biomarkers to predict who will benefit from specific drug or surgical therapies –scarce resources can be allocated to those who will benefit from them, and so that adaptations can be made to enhance effectiveness for subgroups. Critical issues in building sustainable, population care models include the role of specialists, how to improve access to subspecialty care when appropriate (eg, in Parkinson's disease), and how to optimize coordination of subspecialty care within a population health care team. As an example, access to subspecialists for complex pediatric epilepsy has many non-clinical (eg, parent, provider, payer) barriers.

Of particular concern in developing re-engineered care models to improve quality of neurological care is that these new interventions and approaches aim to eliminate disparities, a high national priority. In the dementia collaborative care model trial, the model was even more beneficial among caregivers with low education compared to those with higher education, thereby overcoming educational disparities in care quality. Research on cardiovascular risk reduction has shown that interventions need to span traditional healthcare systems and the home and community, to redress disparities. Given the role of many academic medical centers in providing care in the public safety net system – usually through its training programs - academic neurology is well-positioned to lead and apply research on racial and ethnic disparities in neurologic care, with the ultimate goal of eliminating these disparities.

Finally, the Affordable Care Act has created an enormous, 'sea-change' opportunity to introduce evidence-based care models into sustainable programs within academic medical centers and elsewhere, through the dramatic shift in payment incentives.

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### Placing Neurology at the Lead of Better Health

S. Claiborne Johnston, M.D, Ph.D., M.P.H., *The University of Texas at Austin*

The current healthcare system is dysfunctional in many ways, with too little emphasis on prevention and subjugation to fee-for-service revenue. Abrupt change is required to address relentlessly raising costs without commensurate improvements in health. Academic neurology is particularly well situated to help guide a transition in the health care system. A disciplined approach to gathering, analyzing, and interpreting complex data will be required in order to develop and sustain a system that rewards better outcomes, with the ultimate goal of aligning with society's interest in the efficient promotion of health.

### The Future of Teleneurology

Ray Dorsey, M.D., *University of Rochester Medical Center*

Telehealth, the provision of care at a distance, is rapidly re-shaping health care. Since its introduction in an academic paper in 1999, telestroke has re-shaped care for a common acute neurological condition enabling access to expert neurological care and treatment for millions. In less than two decades, telestroke has become mainstream and is now part of standard stroke care around the world.

While telehealth has transformed care for acute neurological conditions, applications to chronic neurological conditions are still in their infancy. Current care models currently ask individuals with often disabling conditions, impaired mobility, and overburdened caregivers to travel to major urban centers to receive care. By using technology to bring expert neurological care to individuals (rather than patients to care), academic neurologists can deliver patient-centered care where it is needed most. A recent randomized controlled trial of telemedicine for Parkinson disease highlights the benefits and limitations of this approach. Just as telestroke has expanded the reach of academic medical centers, virtual clinic visits can do the same for chronic conditions and lay the foundation for the research and education missions of academic neurology.

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# TUESDAY SPEAKER ABSTRACTS

## THE SOCIAL-EMOTIONAL BRAIN: FROM NEUROBIOLOGY TO NEUROLOGICAL DISEASE

### Coming to Terms With Fear

Joseph LeDoux., Ph.D., *Center for Neural Science, NYU; Emotional Brain Institute , NYU and Nathan Kline Institute.*

Fear is a fundamental part of human life, and plays a central role in psychiatric disorders. One of the main ways that fear has been related to brain mechanisms is through studies of Pavlovian fear conditioning. Research on Pavlovian fear conditioning has been very successful in revealing the brain's so-called fear system. The field has now matured to the point where a sharper conceptualization of what is being studied could be very useful as we go forward. Terms like "fear conditioning" and "fear system" blur the distinction between processes that give rise to conscious feelings of fear and non-conscious processes that control defense responses elicited by threats. The fear conditioning procedure allows exploration of how the brain learns about and later detects and responds to threats, not how the brain feels fear. While mechanisms that detect and respond to threats contribute indirectly to conscious feelings of fear, they are not the same as those that give rise to conscious fear. This is an important distinction since symptoms based on conscious and non-conscious processes may be vulnerable to different predisposing factors and may also be treatable with different therapeutic approaches in people who suffer from uncontrolled fear or anxiety. A conception of aversive conditioning in terms of circuits that detect and respond to threats non-consciously, but that contribute to conscious fear, is proposed as way forward. Key to this conception is a new set of terms that avoid the implication that the circuits are responsible for conscious feelings of fear. Thus, circuits that detect and respond to threats are conceived as defensive survival circuits; these work non-consciously in humans and other animals. Activation of defensive survival circuits results in the expression of defensive responses in the body, and a hosts of changes in the brain. Within the brain, the collective consequence of activating a defensive survival circuit is the establishment of a defensive motivational state. This global state organizes future brain functions, including actions, but also functions non-consciously. In species with the cognitive where-with-all to be able to monitor brain activities in relation to the self, a conscious feeling of fear can arise from the coalescence in awareness of (a) sensory information about an external stimulus; (b) long-term semantic and episodic memories that identify the present stimulus as a threat to one's self, and (c) cognitive monitoring of defensive motivational state information triggered within the brain and in external behavior. The nervous systems of many organisms create these global motivational states that are part of the quest to survive danger. Only an organisms that can be conscious of its own brain's activities in relation to a sense of self can consciously experience fear when a defensive motivational state is helping it to stay alive.

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## The Neurobiology of Social Bonding and Empathy: Implications for Autism

Larry Young, Ph.D., *Emory University*

The socially monogamous prairie vole provides an opportunity to examine the neurobiological and genetic mechanisms underlying complex social behaviors, including social bonding and empathy-related behaviors. Oxytocin receptor (OXTR) signaling in the nucleus accumbens (NAcc) is critical for pair bond formation between mates. Diversity in expression patterns within the brain contribute to diversity in social behaviors across and within species. In prairie voles, oxytocin links the neural encoding of the social signature of the partner with the rewarding aspects of mating through interactions with dopamine and by coordinating communication across a neural network linking social information with reward. Genetic polymorphisms robustly predict natural variation in OXTR expression in the striatum (King et al., 2015), which predict pair bonding behavior and resilience to neonatal social neglect (Barrett et al., 2015). We have also explored the capacity of prairie vole to display empathy-like behavior, specifically consoling. Prairie voles increase their partner-directed grooming toward mates that have experienced an unobserved stressor. This consoling response is abolished blocking oxytocin receptor antagonist into the anterior cingulate cortex, a region involved in human empathy (Burkett et al., 2016). Finally, Loss of a bonded partner results in the development of depressive-like "grieving" behavior. Infusion of oxytocin into the NAcc prevents social loss-induced depression. Studies using intranasal oxytocin and behavioral genetics suggest that the role of oxytocin on social attachment and social cognition is conserved from rodent to man. In humans, intranasal oxytocin enhances eye gaze into the eyes of others, the ability to infer the emotions of others from facial cues, empathy, and socially reinforced learning. Thus the oxytocin system may be a viable target for drugs to improve social functioning in autism. Melanocortin agonists, in particular, evoke endogenous oxytocin release, facilitate social bonding, and activate oxytocin-dependent neural networks via enhancing oxytocin receptor signally, and thus represent a novel therapeutic strategy for improving social function in autism spectrum disorders (Modi et al., 2015).

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## Neurogenetic Mechanisms in Williams Syndrome: Translating between Genes, Brain, and Complex Human Behavior

Karen Faith Berman, M.D., *National Institutes of Health, NIMH*

Williams syndrome (WS), a rare neurogenetic, developmental disorder caused by hemizygous microdeletion of approximately 1.6 megabases on chromosomal band 7q11.23, has a unique profile of striking behavioral features: remarkable hypersociability combined with differential impact on cognitive functions-some mildly affected while others, particularly visuospatial construction, are severely impaired. Because the genes involved are known, WS affords a privileged setting for investigating how genes are translated in the brain to produce cognitive and behavioral features.

Using multimodal imaging (PET, MRI, MRS, fMRI, DTI), we have identified several fundamental aspects of the brain phenotype in Williams syndrome: 1) underlying the syndrome's hallmark visuospatial construction impairment, is a neurostructural anomaly and adjacent neural hypofunction in the dorsal visual processing stream, as well as hippocampal involvement; 2) underlying the syndrome's hallmark social cognition and personality features, are structural and functional anomalies in the insula, in the orbitofrontal cortex, and in a fronto-amygdalar circuit found to be dysfunctional in WS; and 3) underlying several of these gray matter structural and functional abnormalities are altered white matter axonal tracts.

Identification of these brain phenotypes in WS has motivated experiments aimed at linking specific genes in 7q11.23, such as LIMK1 and GTF2I, to the neural and behavioral features of the syndrome. First, extremely rare individuals with small deletions that include only a subset of the genes deleted in classic WS-LIMK1 and genes extending telomerically, but not GTF2I have the visuospatial deficits but not the hypersociability found in people with classic WS 7q11.23 deletions. In these individuals, we found reduced gray matter volume and activation in several dorsal stream regions, as was seen in classic WS. Because these individuals did not have GTF2I deleted, disruption of this gene does not appear to be necessary to produce the observed visuospatial deficits, hypofunction, and reduced gray matter volume. Moreover, when analyses were restricted to six individuals with only LIMK1 and ELN deleted, we a found a similar pattern. Because ELN plays little part in neural organization, our findings strengthen the evidence for an important role of LIMK1 in the dorsal stream abnormalities and associated visuospatial impairment in WS. Second, and further implicating LIMK1, We found that allelic variation in this gene is associated with reduced gray matter volume in the dorsal stream in a large cohort of healthy individuals (N=244, P<001). In contrast, the WS hypersocial personality and its underlying neural substrate appear to be linked to the GTF2I hemideletion.

The incisive approach afforded by this known genetic abnormality and the specific cognitive/behavioral profile it produces in WS provides a unique opportunity for the study of neurogenetic mechanisms of social function. Further work longitudinally documenting the developmental trajectory of these brain phenotypes in children with WS is underway.

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### Neural Mechanisms of Socioemotional Disruption in Frontotemporal Dementia

Virginia Sturm, Ph.D., *University of California, San Francisco*

Changes in emotion are common in neurodegenerative diseases. In frontotemporal dementia (FTD), there is progressive deterioration of the neural systems that support social behavior, empathy, speech, and language. Disruption of emotion is a hallmark feature of behavioral variant frontotemporal dementia (bvFTD), a clinical subtype of FTD that targets brain regions that support emotion generation and resting autonomic physiology. In our research, we use a laboratory-based approach to assess emotional reactivity, empathy, and emotion regulation in patients with FTD and Alzheimer's disease (AD). We quantify patients' autonomic nervous system activity, facial expression, and subjective experience in response to affective stimuli and use structural and functional neuroimaging techniques to determine how emotional responding relates to neural integrity. Our studies have revealed areas of preservation, impairment, and enhancement in specific emotion systems in FTD and AD. Alterations in emotion physiology and behavior reflect patterns of brain atrophy and functional connectivity impairment, and they relate to clinical symptoms. By refining our understanding of the neurobiology of emotion, this work not only advances current models of emotion dysfunction in FTD but also helps to elucidate the biological bases of affective symptoms that are common across neurological and psychiatric disorders.

**References**


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2. Sturm VE, Sollberger M, Seeley WW, et al. Role of right pregenual anterior cingulate cortex in self-conscious emotional reactivity. Soc Cogn Affect Neurosci 2013;8:468-474.
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## SELECTIVE NEURONAL DYSFUNCTION AND DEGENERATION SYMPOSIUM

### What Drives the Pattern of Pathology in Parkinson's Disease?

James Surmeier, Ph.D., *Northwestern University*

Parkinson's disease (PD) is the second most common neurodegenerative disease. One of the key features of the disease is a selective pattern of Lewy pathology and neurodegeneration that includes, but is not limited to, the substantia nigra pars compacta. It is widely believed that this pattern of pathology is caused by a prion-like process. However, connectome-mapping studies show that the pattern of LP in PD is not consistent with this simple model, arguing that if LP propagates in PD, it must be gated by cell- or region-autonomous mechanisms. In this talk, I will discuss the evidence for and against the spreading prion model, as well as evidence that vulnerable neurons share anatomical and physiological characteristics that render them more susceptible to both alpha-synuclein pathology and death.

### Circuit-Selective Function and Dysfunction of Basal Ganglia in Habit and Compulsion

Nicole Calakos, M.D., Ph.D., *Duke University Medical Center*

Habits are a form of normal healthy adaptive learning that emerge from repeated experience and positive reinforcement. Habitual responding can enable routine, previously experienced goal-directed actions to be performed with relatively little cognitive attention, freeing up attentional demands for novel stimuli and challenges. The advantages of habitual responding have also been tapped into as a source for behavior modification in the lay press. Pathological compulsive behaviors share some similarities with habitual responding but are more rigid and persist despite significant adverse consequences. Compulsive behaviors are commonly observed in diseases such as Obsessive Compulsive Disorder, addiction, and some forms of autism.

The striatal circuitry of the basal ganglia is implicated in both the healthy adaptive response of habit formation and pathological compulsive behaviors. But where do the neural substrates for these two behaviors diverge? And do they actually share any similarities at the neural level, as widely hypothesized? In this presentation, new opportunities to answer these questions using contemporary neurophysiological approaches and mouse models will be discussed. A key element for appreciating neural circuit substrates for habit and compulsion is to discriminate between the two classes of striatal projection neurons. Within the striatum of the basal ganglia, there are two output pathways, the striatonigral and striatopallidal, that are widely known to exert opposing influences on movement, especially with gross manipulations such as lesions, deep brain stimulation, and opto- or chemo-genetic techniques. However, how striatal circuit adaptations lead to more subtle behaviors, as in habit or compulsion is less well understood. We will discuss our recent findings which reveal that long-lasting and circuit-selective reconfigurations in how the striatal circuitry responds to incoming activity exist in sensorimotor regions of the dorsal striatum and can discriminate goal-oriented from habitual and compulsive mice. Our findings are also significant for providing a previously technically inaccessible view of the features of activity between basal ganglia circuits that predict behavior. Notably, such a view has led us to discover a novel mechanism by which striatal plasticity may influence behavior - which is by modulating the relative latency to fire action potentials between direct and indirect pathway neurons. These findings update models of basal ganglia function to include not only rate but relative timing as a mechanism driving behavior.

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## Selective Neuronal Vulnerability in Movement Disorders and Dystonia

William Dauer, M.D., *University of Michigan Medical School*

Neurological diseases are defined largely by their distinctive patterns of cell and circuit dysfunction and degeneration. The factors that determine this selective vulnerability are poorly understood, however. This lecture will outline the conceptual considerations important in addressing this biological question, and highlight recent advances in defining the cell types and mechanisms of selective vulnerability in primary dystonia.

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2. Pappas, S. S., Darr, K., Holley, S. M., Cepeda, C., Mabrouk, O. S., Wong, J. M., LeWitt, T. M., Paudel, R., Houlden, H., Kennedy, R. T., Levine, M. S., and Dauer, W. T. (2015). Forebrain deletion of the dystonia protein torsinA causes dystonic-like movements and loss of striatal cholinergic neurons. Elife 4,
3. Liang, C. C., Tanabe, L. M., Jou, S., Chi, F., and Dauer, W. T. (2014). TorsinA hypofunction causes abnormal twisting movements and sensorimotor circuit neurodegeneration. J Clin Invest 124, 3080-3092.
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## Frontotemporal Dementia Neuroanatomy: Onset, Spread, and Implications for Treatment

William W. Seeley, MD, *Professor of Neurology and Pathology, University of California, San Francisco*

The anatomy of neurodegenerative disease can be understood in terms of two key aspects: onset and progression. Mechanisms controlling onset timing and location remain mysterious, and each disease features striking heterogeneity in its onset sites. Network analyses have revealed that each clinical syndrome reflects degeneration of a specific large-scale network. Each vulnerable network, in turn, is anchored by a key "epicenter" whose functional-anatomical connections govern vulnerability of other regions, perhaps because prion-like corruptive templating begets trans-synaptic disease protein spread. In behavioral variant frontotemporal dementia (bvFTD), disease begins within a "salience network", anchored by the anterior cingulate and frontoinsular cortices, regions specialized for social-emotional-autonomic processing. Patients lose the capacity for adaptive, real-time behavioral guidance, possibly in part because salience-driven viscero-autonomic cues and responses are late, degraded, or lacking modulation. Within the salience network hubs, Layer 5 von Economo neurons and fork cells show a particular predilection for disease protein aggregation and cell death, providing a cellular focus for bvFTD selective vulnerability research akin to the motor neuron focus leveraged by amyotrophic lateral sclerosis researchers.

How can selective vulnerability studies help us on the road toward FTD treatments? Human clinical and neuropathological research has and will continue to play a major role in uncovering potential pathogenic mechanisms and prioritizing therapeutic targets. The finer details of FTD neuroanatomy can be productively deployed in the analysis of disease models at all levels, from human induced pluripotent stem cell-derived neurons to transgenic or disease protein-inoculated rodents. Focusing these critical research tools on the most relevant neural systems may prove critical for enhancing the signals a model can provide about therapeutic avenues. Nonetheless, the most important disease model will remain the living patient, in whom determining therapeutic efficacy will require more elegant tools, crafted using theoretical principles and data, to detect onset and measure network-based spread.

# NOTES

# 2016 AWARD INFORMATION

## RAYMOND D. ADAMS LECTURESHIP

### Daniel Geschwind, M.D., Ph.D.

Dr. Daniel Geschwind is the Gordon and Virginia MacDonald Distinguished Professor of neurology, psychiatry and human genetics at the UCLA School of Medicine, and the Senior Associate Dean and Associate Vice Chancellor of Precision Medicine in the UCLA Health System and David Geffen School of Medicine.. He is director of the Neurogenetics Program and the Center for Autism Research and Treatment (CART) and co-director of the Center for Neurobehavioral Genetics in the Semel Institute at UCLA. Dr. Geschwind obtained an A.B. in psychology and chemistry at Dartmouth College, followed by 2 years as a research associate at the Boston Consulting Group. He subsequently obtained his M.D./Ph.D. at Yale School of Medicine (AOA) prior to completing his internship, residency (Neurology), and postdoctoral fellowship at UCLA, after which he founded the neurogenetics program in 1997.

Dr. Geschwind's laboratory aims to develop a mechanistic understanding of neuropsychiatric diseases, namely autism and neurodegenerative diseases, and their relationship to the range of normal human higher cognitive function and behavior. The lab's approach relies heavily on computational and bioinformatic methods in addition to wet laboratory experimentation. In this area the lab has pioneered the application of gene expression and network methods in neurologic and psychiatric disease, working in collaboration with dozens of other laboratories to connect molecular pathways to nervous system function. The over-arching goal is to develop new therapeutics for nervous system disorders such as autism or dementia for which disease-altering therapies are not currently available.

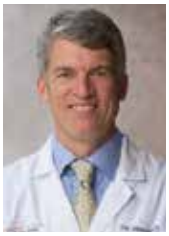
Dr. Geschwind is also a strong advocate for data and biomaterial sharing, having provided scientific leadership for the Autism Genetic Resource Exchange (AGRE). He has served on numerous scientific advisory boards, including the Faculty of 1000 Medicine, the Executive Committee of the American Neurological Association, the Scientific Advisory Board for the Allen Institute for Brain Science, the NIMH Advisory Council and the NIH Council of Councils. He has published over 390 papers and serves on the editorial boards of Biological Psychiatry, Cell, Current Opinion in Genetics & development, Human Molecular Genetics, Neurobiology of Disease, Neuron and Science. He received the Derek Denny-Brown Neurological Scholar Award from the American Neurological Association in 2004, an NIMH MERIT Award, the Scientific Service Award from Autism Speaks in 2007, the Ruane Prize for Child and Adolescent Psychiatric Research from the Brain and Behavior foundation in 2012, the Taking on Tomorrow Innovation Award (Research/Scientific Breakthrough in Autism) -Boston Children's Hospital in 2013. He is a member of the American Academy of Physicians, and is an elected member of the National Academy of Medicine, USA.

## F.E. BENNETT MEMORIAL LECTURESHIP

### D. James Surmeier, Ph.D.

Dr. D. James Surmeier is the Nathan Smith Davis Professor and Chair of the Department of Physiology at the Feinberg School of Medicine at Northwestern University and Director of the Morris K. Udall Parkinson's Disease Research Center of Excellence at Northwestern University. Dr. Surmeier received his Ph.D. in Physiology and Biophysics from the University of Washington in 1983. In 1998, he moved to the Department of Physiology at Northwestern University and assumed his current position as Chair in 2001. Dr. Surmeier's research program focuses physiological determinants of Parkinson's and Huntington's diseases. His pursuit of the mechanisms underlying selective neuronal vulnerability in Parkinson's disease has led to the identification of factors underlying the selective vulnerability of substantia nigra dopaminergic neurons – neurons whose loss underlies the cardinal motor symptoms of Parkinson's disease.

## SORIANO LECTURESHIP



### S. Claiborne Johnston, M.D., Ph.D., M.P.H.

S. Claiborne "Clay" Johnston, M.D., Ph.D. Clay Johnston is the inaugural Dean of the Dell Medical School at The University of Texas at Austin. His ambitious vision includes building a world-class medical school by creating a vital, inclusive health ecosystem that supports new and innovative models of education, health care delivery and discovery – all with a focus on improving health and making Austin a model healthy city. Clay is also a Neurologist, specializing in stroke care and research. He was formerly at the University of California, San Francisco where he served as Associate Vice Chancellor of Research and founding director of the Center for Healthcare Value. Clay is a graduate of Amherst College, completed medical school at Harvard University and received a Ph.D. in epidemiology from the University of California, Berkeley.

## DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR AWARD

### DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR AWARD IN BASIC SCIENCE

**Robert Baloh, M.D., Ph.D.**

Dr. Baloh received a dual MD-Ph.D. degree from Washington University, and subsequently completed the Harvard Neurology Residency Program, serving as Chief Resident in his final year. Afterwards he returned to Washington University for a fellowship in neuromuscular diseases, and to develop his laboratory program focused on understanding the molecular mechanisms of neurodegenerative diseases.

In 2011 Dr. Baloh moved to Cedars-Sinai Medical Center to head the newly formed Division of Neuromuscular Medicine. He is currently an Associate Professor of Neurology at Cedars-Sinai and UCLA. He maintains an active clinical practice, and a basic and translational laboratory research program.

### DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR AWARD IN CLINICAL SCIENCE

**Glen Jickling, M.D., MS.c., F.R.C.P.C.**

Dr. Glen Jickling is an Assistant Professor at the University of California Davis, in the Department of Neurology. His research interests are the genomics and genetics of cerebrovascular disorders. He completed his M.D.and Neurology residency at the University of Alberta and a Stroke fellowship at University of California. He studies include RNA as markers of cerebrovascular disease, microRNA regulation of RNA in stroke, and the role of the immune response in ischemic brain injury. He has described RNA as a marker to identify atrial fibrillation in stroke, to identify TIA, and to predict tPA related hemorrhagic transformation.

### DEREK DENNY-BROWN YOUNG NEUROLOGIAL SCHOLAR AWARD IN CLINICAL SCIENCE

**Alex R. Paciorkowski, M.D.**

A native of Morristown, NJ, Dr. Paciorkowski received his B.A. from New York University majoring in Latin American Studies. After completing medical school at the University of Connecticut, he remained at UConn for pediatric and medical genetics residencies. Dr. Paciorkowski then relocated to Washington University in St. Louis to obtain child neurology training, and was a research fellow through the Neurological Sciences Academic Development Award. After a post-doc in Seattle, since 2012 he has been a faculty member at the University of Rochester. Dr. Paciorkowski's laboratory focuses on discovering mutations in novel genes for developmental brain disorders, followed by the study of these mutations using a variety of in vitro neuroscience methods. He also focuses on developing new computational methods for understanding phenotype-genotype correlations. Dr. Paciorkowski's clinical work is devoted to improving the care of individuals with neurogenetic disorders.

## DISTINGUISHED NEUROLOGY TEACHER AWARD

**David Lee Gordon, M.D., F.A.A.N., F.A.N.A., F.A.H.A.**

Dr. David Lee Gordon has served as Professor and Chair of the Department of Neurology at the University of Oklahoma College of Medicine (OUCOM) since 2007. He received his medical degree from University of Miami, completed neurology residency at Mt. Sinai Hospital, and completed fellowship in cerebrovascular diseases at University of Iowa. He has special interest in stroke, migraine, and medical education. He has been named to "Best Doctors in America®" on multiple occasions and has received numerous teaching awards, including the OUCOM Stanton L. Young Master Teacher Award and the American Academy of Neurology Clerkship Directors Teaching Award.

## THE GRASS FOUNDATION-ANA AWARD IN NEUROSCIENCE

**Aasef Shaikh, M.D., Ph.D.**

Aasef Shaikh is a neurologist and neuroscientist from Daroff-Dell'Osso Ocular Motility Laboratory, Case Western Reserve University, University Hospitals Cleveland, and Cleveland VA Medical Center. His research focuses on the application of control systems engineering to approach complex disorders of the vestibular system, eye movements, and head movements. The overarching goal is to discover novel network connections and leverage their influence to modulate the motor circuits artificially for the treatment of intractable neurological conditions. Dr. Shaikh has received research support from National Institutes of Health, Dystonia Medical Research Foundation, and Human Frontiers Science Program. He was the recipient of the prestigious American Academy of Neurology Alliance Founders Award.

## WOLFE NEUROPATHY RESEARCH PRIZE

**Michael Polydefkis, M.D., M.H.S.**

Dr. Polydefkis is a Professor of Neurology at Johns Hopkins. He grew up on the south side of Chicago and after graduating from Brown University attended medical school at Johns Hopkins. Michael completed internal medicine, neurology and neuromuscular training at Johns Hopkins. His research focuses on using human skin biopsies to develop novel measures of peripheral nerve disease and sensitive outcome measures for clinical trials.

# 2016 AWARD INFORMATION

## TRAVEL AWARDS

Each year the ANA selects the top abstracts submitted by fellows, residents, students, or junior faculty to receive a travel award to attend the Annual Meeting and present their work. For more information, please visit the main ANA website.

***Poster numbers listed with an S will be presented on Sunday, October 16; poster numbers listed with an M will be presented on Monday, October 17.***

Ye Hu, M.D., *University of Miami*
**S147 Relation between Fatigue Severity Scale (FSS) and Pupillary Indices in Patients with Multiple Sclerosis (MS)**

Francesca Cignarella, Ph.D., *Washington University in St. Louis*
**S149 Effects of Intermittent Fasting in an Animal Model of Multiple Sclerosis**

Michaela Liedtke, M.D., *Stanford University School of Medicine*
**S154 Impact of NEOD001 on Neuropathy and Organ Biomarkers in Patients with Light Chain Amyloidosis and Persistent Organ Dysfunction: Results from the Expansion Phase of a Phase 1/2 Study**

Harrison Bai, M.D., *Hospital of the University of Pennsylvania*
**S155 Clinical Features and Prognostic Factors of 476 Patients with Spinal Astrocytoma: An Integrated Analysis from Multi-Institutional Data and the Literature**

Hao Zhou, M.D., *Xiangya Hospital, Central South University*
**S156 MR Imaging Features Predict Survival and Molecular Profile in Diffuse Lower Grade Gliomas**

Donna Tippett, M.P.H., M.A., CCC-SL, *Johns Hopkins University School of Medicine*
**S158 Differentiating between Subtypes of Primary Progressive Aphasia and Mild Cognitive Impairment on a Modified Version of the Frontal Behavioral Inventory**

Virginia Sturm, Ph.D., *University of California, San Francisco*
**S159 Emotion Network Breakdown Relates to Baseline Autonomic Dysfunction in Behavioral Variant Frontotemporal Dementia**

Jun-Sang Sunwoo, M.D., *Soonchunhyang University Seoul Hospital*
**S160 Altered MicroRNA Expression in a Mouse Model of Autism Spectrum Disorder**

Farwa Ali, M.B.B.S., *Mayo Clinic*
**S161 Bismuth Neurotoxicity: A Rare Cause of Rapidly Progressive Dementia**

Nazan Aksan, Ph.D., *University of Iowa*
**S162 Relevance of Inhibitory Control and Neurocognitive Function to Tactical Self-Regulation in the Context of Driving**

Alexis Simpkins, M.D., Ph.D., *National Institutes of Health*
**S163 Early Changes in ADC Volumes Are Associated with Absolute Neutrophil Count During Ischemic Stroke**

Muhammad Shah Miran, M.D., *CentraCare St. Cloud Hospital*
**S165 Severe Stenosis of Internal Carotid Artery Is Associated with Higher Neutrophil-Lymphocyte Ratio: A Concept of Cerebrovascular Stress**

Paul Jarvis, M.D., *University of Vermont*
**S166 Localized Hippocampal Knockout of TSC1 Gene in Mice Leads to In Vivo Cognitive Dysfunction and Electrophysiological Changes without Seizures**

Madeline Fields, M.D., *Ichan School of Medicine at Mount Sinai*
**S167 Palinacousis**

Richa Tripathi, M.D., *Detroit Medical Center/ Wayne State University*
**S168 Generalized Tonic Clonic and Psychogenic Non Epileptic Spells Audio Recordings in the Epilepsy Monitoring Units: Identification and Inter-Rater Agreement**

Luigi Maccotta, M.D. Ph.D, *Washington University School of Medicine*
**S169 Seizure Control Does Not Change Abnormal Functional Connections in Temporal Lobe Epilepsy**

Oren Levy, M.D. Ph.D, *Columbia University Medical Center*
**S171 KTKEGV-Motif Mutations in Alpha-Synuclein Cause Neuronal Death and Defects in Neurite Regrowth After Mechanical Injury**

Sam Horng, M.D., Ph.D, *Icahn School of Medicine at Mount Sinai*
**S172 Claudin-4 is Required for Astrocytic Tight Junction Formation and Protects Against Inflammatory Lesion Size and Severity in the Central Nervous System**

David Bargiela, MBBS, *Brigham and Women's Hospital*
**S173 Selection of First-Line Therapy in Multiple Sclerosis Using Risk-Benefit Decision Analysis**

Shila Azodi, M.D., *National Institutes of Health*
**S174 Longitudinal Spinal Cord Measurements in Neuroinflammatory Diseases**

Monique Anderson, BA, *National Institutes of Health*
**S176 HTLV-I Viral Antigens Detected in Exosomes Isolated from HAM/TSP Patient CSF by Novel Nanotrap Technology**

Dana Marafie, M.D., *Baylor College of Medicine*
**S178 Prevalence and Character of Severe Epileptic Encephalopathy in Patients Affected by MECP2 Duplication Syndrome**

Renee Nelson, BS in progress, *Tulane University*
**S180 Gender Differences in Symptom Manifestation and Survival Patterns in Multiple System Atrophy**

Giovanni Battistella, Ph.D., *Icahn School of Medicine at Mount Sinai*
**S181 Polygenic Risk of Spasmodic Dysphonia Is Associated with Functional Connectivity Alterations in the Sensorimotor Cortex**

Franziska Hoche, M.D., *Massachusetts General Hospital and Harvard Medical School*
**S183 Development of a Brief Ataxia Rating Scale for Children (BARS-c)**

CONTINUED

Yasuyoshi Kimura, M.D.,Ph.D., *Graduate School of Medicine, Osaka University*
**S185 An Extragenic Safe Harbor Permits Suicide Gene Expression Serving as a Safety Switch for Cell Therapy of Neurological Disorders**

Jennifer Purks, BS, *Georgetown University*
**S187 Applying Natural Language Processing (NLP) to Verbatim Patient-Reported Outcomes**

Michelle Fullard, M.D., *University of Pennsylvania*
**S188 Predictors of Neurobehavioral Outcomes in Bilateral Subthalamic Nucleus Deep Brain Stimulation Surgery**

Nancy Song, M.D., *Dartmouth-Hitchcock Medical Center*
**S189 A Novel Presentation of Post-Surgical Edema Associated with DBS Implantation with Review of the English Literature**

Arpita Hazra, M.D., *Detroit Medical Center*
**S191 Temporal Trends and Outcomes of Acute Ischemic Stroke in Patients with Cancer of Brain and Nervous System**

Marianna Spatola, M.D., *Institut d'Investigacions Biomdiques August Pi i Sunyer (IDIBAPS)*
**M101 New Clinical and Immunological Features of Anti-GABAA Receptor Encephalitis**

Remi Kessler, B.A., *Johns Hopkins University*
**M103 Anti-AQP4 Titer Is Not Predictive of Disease Course in Neuromyelitis Optica Spectrum Disorder**

Brendan Lucey, M.D., *Washington University School of Medicine*
**M104 Effect of Sleep Deprivation and Sodium Oxybate on CSF A40 and A42 Kinetics**

Benjamin George, M.D., M.P.H., *University of Rochester School of Medicine and Dentistry*
**M105 Trends and Regional Patterns of Care Associated with Hospice for US Stroke Inpatients, 2002-2011**

Sarah Crisp, MB, BChir, Ph.D., Institute of Neurology, *University College London*
**M109 Pathological Mechanisms of Glycine Receptor Antibodies**

Michael Levy, M.D., Ph.D, *Johns Hopkins University*
**M111 Phase II, Placebo-Controlled, Double-Blinded, Crossover Study of Extended-Release Dalfampridine in Monophasic Transverse Myelitis**

Jung-Ah Lim, M.D., *Seoul National University Hospital*
**M112 Tocilizumab Is Effective in Autoimmune Limbic Encephalitis Refractory to Weekly Rituximab**

Dongming Cai, M.D., Ph.D, *Icahn School of Medicine at Mount Sinai*
**M114 Characterization of Molecular Mechanism(s) Underlying ApoE4-Induced Brain Phospholipid Dysregulation in Alzheimer's Disease**

Justin Chandler, M.D., *University of Rochester School of Medicine and Dentistry*
**M115 Hospital Tax Status and Carotid Artery Stent Utilization in US Hospitals Performing Carotid Revascularization**

Benjamin Murdock, Ph.D, *University of Michigan*
**M118 NK and NKT Cells Are Increased in the Peripheral Blood of ALS Patients**

Tae-Joon Kim, M.D., *Seoul National University Bundang Hospital*
**M120 Anti-LGI1 Encephalitis Is Associated with Unique HLA Class II Subtypes**

Jesse Cohen, BA, *Johns Hopkins Medicine*
**M121 A Costly Condition: Assessing the Burden of Autoimmune Encephalitis at a Tertiary Institution from 2006-2015**

Claudia Santos BS, *University of Rhode Island / Lifespan*
**M122 Cardiac Vagal Tone Is Altered in Pre-Clinical Alzheimer's Disease**

Kimberly Webster, M.A., M.S., *Johns Hopkins University*
**M123 Electrostimulation Effects in lvPPA and nfvPPA**

Whitney Wharton, Ph.D., *Emory University*
**M126 Fewer Neurofibrillary Tangles and Slower Conversion to AD in MCI Patients Taking RAS Acting Antihypertensives**

Andrea Faria, M.D., Ph.D., *Johns Hopkins University*
**M127 Relationship Between Orthographic and Phonological Treatments with Brain Atrophy in PPA**

Thomas Wingo, M.D., *Emory University*
**M128 A Genetic Study of a Large Pedigree with Late-Onset Alzheimer's Disease**

Suman Jayadevm, M.D., *University of Washington*
**M129 Alzheimer Disease Associated Presenilin 2 N1411 Mutation Leads to Loss of Gamma-Secretase Function and Enhanced Pro-Inflammatory Behavior in Microglia**

Eric Landsness, M.D., Ph.D, *Washington University in St. Louis*
**M130 Ziprasidone as a Potential Abortive Treatment for Status Migrainosus**

Daniela Menichella, M.D., Ph.D, *Northwestern University Feinberg Medical School*
**M133 Chemogenic Silencing of Nav 1.8 Nociceptors Reverses Neuropathic Pain and Small Fiber Degeneration in Painful Diabetic Neuropathy (PDN)**

Phillip O'Brien, Ph.D., *University of Michigan*
**M135 Amelioration of Peripheral Neuropathy in HFD-STZ Mice Through Dietary Reversal**

Christopher Cashman, BA, *Johns Hopkins School of Medicine*
**M136 Direct Motor Neuron Transplantation Maintains the Regenerative Capacity of Chronically Denervated Peripheral Nerves**

Lauren Bowen, M.D., *National Institutes of Health/NINDS*
**M139 Motor Neuron Disease with HIV infection: Activation of HERV-K and Response to Anti-Retroviral Therapy**

Matthew Cykowski, M.D., *Houston Methodist Hospital*
**M140 Microglial, pTDP43, and Neuronal Pathologies in the Brainstem and Spinal Cord of ALS Patients: Relationship to Site of Symptom Onset and Disease Duration**

Kristy Hwang, BS, *Oakland University William Beaumont SOM*
**M147 Predicting Brain Amyloidosis Using Peripheral Blood-Based Gene Expression Measures**

Emer McGrath, M.B., Ph.D., *Massachusetts General Hospital*
**M148 Blood Pressure Trends in Mid-To-Late Life and Risk of Dementia**

Michelle Caunca, BS, *University of Miami Miller School of Medicine*
**M149 Carotid Intima-Media Thickness, Plaque, and Cognition: The Northern Manhattan Study**

# 2016 AWARD INFORMATION

Yohannes Woldeam, M.D., *Stanford University School of Medicine*
**M150 Migraine and Complex Regional Pain Syndrome: A Case-Referent Clinical Study**

Hsinlin Cheng, M.D., Ph.D., *Massachusetts General Hospital*
**M152 Age-Dependent Painful Neuropathy in Metabolic Syndrome**

Mfon Umoh, BAS, *Emory University*
**M153 Is C9orf72 ALS Different from Sporadic Disease? A Comparative Study in an ALS Clinic Population**

David Brandman, M.D., *Brown University*
**M154 Closed Loop Intracortical Brain Computer Interface Control in a Person with ALS Using a Filtered Gaussian Process Decoder**

Raghav Govindarajan, M.D., *University of Missouri, Columbia*
**M155 Canine Degenerative Myelopathy-An Emerging Disease Model for ALS**

Stephanie Geisler, M.D., *Washington University in Saint Louis*
**M156 Genetic Deletion of Sarm1 Attenuates Vincristine-Induced Neuropathy in a Mouse Model**

Kaitlin Seibert, BA, *The University of Toledo College of Medicine and Life Sciences*
**M161 Reasons for Failed Mechanical Thrombectomy: Initial Data from Two Tertiary Care Centers**

Maliheh Mohamadpour, M.D., *Brain and Mind Research Institute*
**M304 Parieto-Occipital delta alpha Spectral Power in Electroencephalogram Indexes Post-Traumatic Confusion and Predicts Recovery After Traumatic Brain Injury**

Zeeshan Mansuri, M.D., M.P.H., *Drexel University*
**S103 Burden and Outcomes of Acute Ischemic Stroke in Patients with Major Depression**

Ravi Chopra, BA, *University of Michigan*
**S106 Increased Dendritic Excitability and Calcium-Dependent PKC Activation: A Novel Mechanism Underlying Purkinje Neuron Dendritic Degeneration in Cerebellar Ataxias**

Christine Ashton, BA, *Beth Israel Deaconess Medical Center*
**S108 Spatio-Temporal Analysis and Modeling of Gait in Parkinson's Disease**

Jennifer Graves, M.D., Ph.D, M.A.S., *University of California, San Francisco*
**S109 Vitamin D Genetic Risk Score Is Strongly Associated with Vitamin D Levels and Relapse Rate in Pediatric MS Patients**

Andrea Schneeider, M.D., Ph.D, *Johns Hopkins University School of Medicine*
**S110 Hospitalization Risk Following Acute Stroke: The Atherosclerosis Risk in Communities (ARIC) Study**

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**S112 Widespread Migration and Integration of Neurons in the Early Postnatal Human Frontal Cortex**

Jochen Meyer, Ph.D., *Baylor College of Medicine*
**S113 Dynamic Participation Patterns of Superficial Cortex During Absence Seizures**

Aman Deep, M.D., *Barrow Neurological Institute*
**S114 Why Do Patients with Parkinson's Disease Fall?: A Single Center Experience**

Serena Bianchi, Ph.D., *Icahn School of Medicine at Mount Sinai*
**S115 White Matter Abnormalities in Distinct Phenotypes and Putative Genotype of Spasmodic Dysphonia**

Veronique VanderHorst, M.D., Ph.D., *Beth Israel Deaconess Medical Center/Harvard Medical School*
**S116 A Translational Approach to Analyze Spatial and Temporal Gait Parameters: Normal and Parkinsonian Gait Signatures in Mice and Human**

Tritia Yamasaki, M.D., Ph.D., *University of Kentucky*
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Jennifer Orthmann-Murphy, M.D., Ph.D., *Johns Hopkins University*
**S120 In Vivo Imaging of Cortical Demyelination and Remyelination in a Mouse Model of Multiple Sclerosis**

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**S121 Differences Between Lacunar Infarcts and Deep Intracerebral Hemorrhage: A Nested Case-Control Study from the Framingham Heart Study**

Norma Castillo, BA, *University of Illinois at Chicago*
**S122 Large Vessel Occlusion Predicted by Stroke Screening Scales**

Mona Bahouth, M.D., *Johns Hopkins School of Medicine*
**S123 Normalization of BUN/Creatinine Ratio in Acute Ischemic Stroke Patients Is Associated with Less Infarct Expansion**

Jangsup Moon, M.D., Ph.D., Biomedical Research Institute, Seoul National University Hospital, College of Medicine
**S124 HLA-B*40:02 and HLA-DRB1*04:03 as Genetic Markers for Oxcarbazepine Induced Maculopapular Eruption**

Hye Rim Shin, M.D., SNUH
**S125 Cyclicity and Clustering of Spontaneous Recurrent Seizures in Mouse Pilocarpine Model of Temporal Lobe Epilepsy**

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**S127 Distinct Modes of Seizure-Like Hyperexcitability as Revealed in Drosophila Sodium Channel Mutants**

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**S129 Framingham Risk Scores Predict a 2 Year Worsening in Motor Impairment in Parkinson Disease**

Elizabeth Coon, M.D., *Mayo Clinic*
**S130 Use of Selective Serotonin Reuptake Inhibitors Does Not Improve Survival in Multiple System Atrophy**

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**S131 Preclinical Investigations of Sex-Specific Risk for Mild Cognitive Impairment in Parkinson's Disease: Androgen Effects on the Subthalamic Nucleus**

Lauren Heusinkveld, BS, *Vanderbilt University Medical Center*
**S133 If We Build It, Will They Come? Motivating Factors and Barriers for Participation in the DBS in Early PD Pivotal Trial**

Christina Tamargo, *Vanderbilt University Medical Center*
**S134 Effects of Optimal Drug Therapy versus Deep Brain Stimulation on Employment in Early Stage Parkinson's Disease**

Maxim Turchan, BA, *Vanderbilt University*
**S136 A Novel Spasticity Diagnostic Algorithm - A Case Study of the Kappa Paradox**

CONTINUED

Kelly Mills, M.D., *Johns Hopkins University*
**S138 Cognitive Impairment in Parkinson's Disease: Association Between Patient-Reported and Clinically Measured Outcomes in the Parkinson's Disease Biomarker Program**

Peter Todd, M.D, Ph.D., *University of Michigan*
**S142 Repeat Associated Non-AUG Initiated Translation from GC Rich Repeats in Neurodegenerative Disease**

Peter Kosa, Ph.D., *National Institutes of Health*
**S144 Development of a Sensitive Outcome for Economical Drug Screening for Progressive Multiple Sclerosis**

Tina Roostaei, M.D., M.P.H., *Centre for Addiction and Mental Health*
**S146 Convergent Effects of a Functional C3 Polymorphism on Cognitive Impairment, Brain Atrophy and Demyelination in Multiple Sclerosis**

Jereemiah Scharf, M.D., Ph.D., *Massachusetts General Hospital*
**S107 Rare Recurrent NRXN1 Deletions and CNTN6 Duplications Increase Risk for Tourette Syndrome**

Mallory Hacker, Ph.D., *Vanderbilt University*
**S105 Deep Brain Stimulation in Early Stage Parkinson's Disease: Long-Term Motor Benefit Through 5 Years**

Jong Park, BS, *Boston Children's Hospital*
**S128 Disrupting Abducens Nerve Development Causes Duane Retraction Syndrome**

Eugenia Martinez- Hernandez, M.D., Ph.D., *Institut d'Investigacions Biomdiques August Pi i Sunyer, Hospital Clinic, University of Barcelona*
**M110 A Novel Type of Autoimmune Encephalitis Associated with Neurexin-3 Antibodies**

Kathleen Poston, M.D., M.S., *Stanford University*
**S101 Dissociated Cognitive Impairments Associated with Midbrain and Hippocampal Microstructural Atrophy in Parkinson's Disease: A 7-Tesla MRI Study**

Rajani Sebastian, Ph.D., *Johns Hopkins University School of Medicine*
**S157 Cerebellar tDCS to Agument Chronic Aphasia Treatment**

# ACADEMIC NEUROLOGY REPRESENTATIVES FROM ITALY

### ACADEMIC NEUROLOGY REPRESENTATIVES FROM ITALY

**SUNDAY, OCTOBER 16 11:45 AM- 1:00 PM**

**Gennarina Arabia, M.D., M.Sc.**
Associate Professor of Neurology
*University of Catanzaro School of Medicine*

**Carla Colosimo, M.D.**
Chairman, Dept. of Neurology
*Santa Maria University Hospital*

Participating in the Interactive Lunch Workshop titled "Are Alzheimer's and Parkinson's Diseases of Childhood?"

**MONDAY, OCTOBER 17 11:45 AM- 1:00 PM**

**Antonio Toscano, M.D.**
Professor of Neurology
*University of Messina*

Participating in the Interactive Lunch Workshop titled "Advances in Understanding and Treatment of Mitochondrial Disorders Affecting the Nervous System"

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**Online Registration:**
http://2016.myana.org/

**Meeting Contacts**
Phone: (856)-380-6889
Hours: Monday–Friday 9:00 am– 5:00 pm ET
Registration Email: meetings@myana.org

**Meeting Location**
Baltimore Marriott Waterfront Hotel
700 Aliceanna Street
Baltimore, MD 21202

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**142nd American Neurological Association Annual Meeting**
October 15 – 17, 2017
San Diego, CA

**143rd American Neurological Association Annual Meeting**
October 21 – 23, 2018
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