ANA2015

140TH ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION

CHICAGO, IL • SEPTEMBER 27-29, 2015

CHICAGO MARRIOTT DOWNTOWN MAGNIFICENT MILE • 2015.MYANA.ORG

FINAL PROGRAM

ANA
American Neurological Association
since 1875

THE 140TH 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 140th ANA Annual Meeting in Chicago, Illinois, September 27-29, 2015.

IMPORTANT DATES

- **Pre-conference Symposium:**
  September 26, 2015
- **Annual Meeting:**
  September 27-29, 2015
- **On-Site Registration Hours:**
  Saturday, September 26 3:00 PM–7:00 PM
  Sunday, September 27 6:00 AM–5:45 PM
  Monday, September 28 6:30 AM–5:45 PM
  Tuesday, September 29 6:30 AM–2:00 PM
- **Speaker Ready Room Hours:**
  Saturday, September 26 3:00 PM–7:00 PM
  Sunday, September 27 6:00 AM–5:45 PM
  Monday, September 28 6:30 AM–5:45 PM
  Tuesday, September 29 6:30 AM–2:00 PM
- **Poster Viewing Hours:**
  Sunday, September 27 11:00 AM–7:00 PM
  Poster presenters will be in attendance from 5:30 PM to 7:00 PM

  Monday, September 28 11:00 AM–7:00 PM
  Poster presenters will be in attendance from 5:30 PM to 7:00 PM
- **President's Reception:**
  Monday, September 28 7:30 PM–9:00 PM

LOCATION

Chicago Marriott Downtown Magnificent Mile
540 North Michigan Avenue
Chicago, IL 60611

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FROM THE CHAIR

DEAR COLLEAGUES,

The American Neurological Association (ANA) is delighted to report that the 140th Annual Meeting kicks off Sunday, September 27, and runs through Tuesday, September 29 in Chicago at the Marriott Downtown Magnificent Mile. We are excited about the scientific program as a whole, but are especially pleased to have a Saturday evening pre-meeting symposium on September 26, highlighting a talk by Mahlon R. DeLong, M.D., the William Timmie Professor of Neurology at Emory University School of Medicine, in a vibrant session with five talks on brain circuit disorders and neuromodulatory interventions. Other symposia will focus on the causes and triggers of MS, advances in neuroengineering, new insights into axonal pathology, debates on recent advances in stroke management and the latest demonstrations that prion type pathology may underlie a variety of neurodegenerative diseases.

The annual meeting features a wide range of educational sessions, including courses in career development, scientific symposia covering a broad spectrum of subspecialty areas and clinical updates in the special interest groups.

As an attendee you will also find a slate of up-to-the-minute presentations from disease-specific interest groups. The ANA continues to evolve to meet the needs of our ever-changing academic neurology community. We have a growing set of new members among neurologists from the U.S., an expanding roster of Ph.D. neuroscientiststs within neurology departments as well as colleagues from overseas. Our mission remains unchanged: Our goal is to develop an even stronger cadre of neuroscientists to participate in investigations of neurological illnesses and their therapies.

In addition to the outstanding programming, we are pleased to mention that our 2015 Annual Meeting affords each of us the opportunity to welcome and celebrate our colleagues from the Indian Academy of Neurology. We are thrilled to continue the tradition of recognizing colleagues and collaborators from overseas as was done in the last three years with the Mexican Academy of Neurology in 2014, the French Societe Francaise de Neurologie in 2013 and the 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 overseas.

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

With best regards,

Sam Pleasure, M.D., Ph.D.
Chair, Scientific Program Advisory Committee
University of California, San Francisco

SCHEDULE AT A GLANCE

SATURDAY, SEPTEMBER 26, 2015

3:00–7:00 PM	Registration Hours *5th Floor Registration*
6:00–10:00 PM	**Pre-Meeting Symposium:** Circuits and Circuit Disorders: Approaches to Neuromodulation *Marriott Ballroom, 4th Floor*

SUNDAY, SEPTEMBER 27, 2015

6:00 AM–5:45 PM	Registration Hours *5th Floor Registration*
7:00–9:00 AM	Continental Breakfast *Chicago Ballroom Foyer, 5th Floor*
7:30–9:00 AM	**Early Career Level Faculty Development Course I:** Finding Happiness: Panel Discussions in Lab, Translational and Clinical Research *Clark, 4th Floor* **Mid/Senior Career Level Faculty Development Course I:** MBA in 1.5 Hours – How an Academic Medical Center Actually Works *Avenue Ballroom, 4th Floor* **AUPN Chair Development Course I:** The Role of "Role Players" in the 21st Century Department *Addison, 4th Floor*
9:00–9:15 AM	Coffee Break *Chicago Ballroom Foyer, 5th Floor*
9:15–11:15 AM	**Symposium:** Causes/Triggers of Multiple Sclerosis *Chicago Ballroom A–E, 5th Floor*
11:00 AM–7:00 PM	**Poster Viewing** *Grand Ballroom, 7th Floor*
11:15–11:45 AM	**New Member Meet and Greet*** *Chicago Ballroom F, 5th Floor*
11:30 AM–1:00 PM	Lunch *Chicago Ballroom Foyer, 5th Floor*
11:45 AM–1:00 PM	**Interactive Lunch Workshops** 1. Current Ethical Dilemmas in Neurology *Purdue/Wisconsin, 6th Floor* 2. Emerging Therapeutic Targets for Demyelination and Remyelination *Los Angeles/Miami/ Scottsdale, 5th Floor* 3. Meet the NINDS* *Indiana/Iowa, 6th Floor* 4. Neurology Goes Global - International Issues in Diagnostics & Therapeutics *Northwestern/Ohio State, 6th Floor* 5. Non-lesional Drug-resistant Focal Epilepsy: Controversies in Diagnostic Evaluation and Surgical Management *Michigan/Michigan State, 6th Floor* 6. Novel Brain MRI and The Human Connectome: Insights and Implications for the Neurologist *Denver, Houston, Kansas City, 5th Floor*
1:15–3:15 PM	**Symposium:** Derek Denny-Brown Young Neurological Scholar Symposium *Chicago Ballroom A–E, 5th Floor*
3:15–3:30 PM	Coffee Break *Chicago Ballroom Foyer, 5th Floor*
3:30–5:30 PM	**Special Interest Group Symposia** 1. Behavioral Neurology *Indiana/Iowa, 6th Floor* 2. Cerebrovascular Disease *Los Angeles/Miami/ Scottsdale, 5th Floor* 3. Epilepsy *Purdue/Wisconsin, 6th Floor* 4. Movement Disorders *Denver, Houston, Kansas City, 5th Floor* 5. Multiple Sclerosis *Northwestern/Ohio State, 6th Floor* 6. Neurocritical Care *Michigan/Michigan State, 6th Floor* 7. Neuro-Oncology *Lincolnshire I/II, 6th Floor*
5:30–7:00 PM	**Poster Presentations & Reception I** *Grand Ballroom, 7th Floor*

MONDAY, SEPTEMBER 28, 2015

6:30 AM–5:45 PM	Registration Hours *5th Floor Registration*
7:00–9:00 AM	Continental Breakfast *Chicago Ballroom Foyer, 5th Floor*
7:30–9:00 AM	**Early and Mid/Senior Career Level Faculty (Combined) Development Course II:** Conflict Resolution/Negotiation *Marriott Ballroom, 4th Floor* **AUPN Chair Development Course II:** Optimizing the Use of Mid-level Providers in an Academic Neurology Practice *Avenue Ballroom, 4th Floor*
9:00–9:15 AM	Coffee Break *Chicago Ballroom Foyer, 5th Floor*
9:15–11:15 AM	**Symposium:** The Life and Death of Axons in Neurological Disease *Chicago Ballroom A–E, 5th Floor*
11:00 AM–7:00 PM	**Poster Viewing** *Grand Ballroom, 7th Floor*

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

MONDAY, SEPTEMBER 28, 2015 *(continued)*

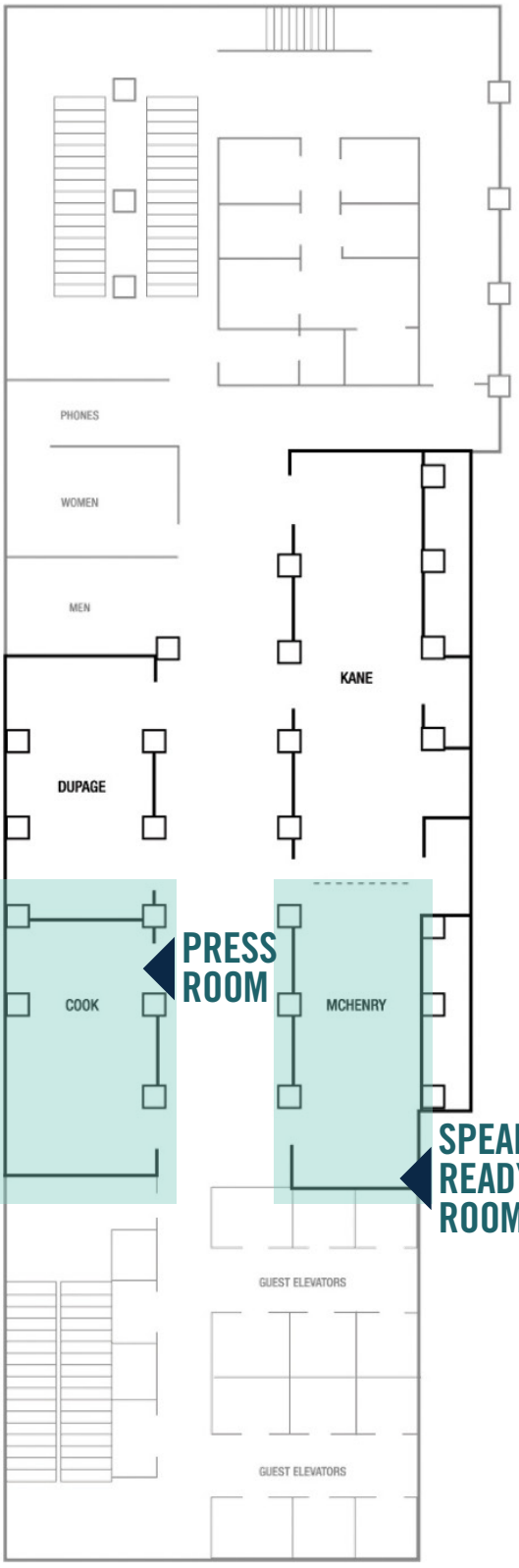
11:15–11:45 AM	**Executive Session of Membership*** *(All Members are encouraged to attend)* *Chicago Ballroom A–E, 5th Floor*
11:30 AM–1:00 PM	Lunch *Chicago Ballroom Foyer, 5th Floor*
11:45 AM–1:00 PM	**Interactive Lunch Workshops** 1. Challenges and Innovation in Prehospital Stroke Care *Indiana/Iowa, 6th Floor* 2. Current State of Stem Cell Therapies *Los Angeles/Miami/Scottsdale, 5th Floor* 3. Emerging CNS Infections *Denver, Houston, Kansas City, 5th Floor* 4. ICU Neuropathy *Michigan/Michigan State, 6th Floor* 5. Meet the Neurology Department Chairs* *Purdue/Wisconsin, 6th Floor* 6. Translational Research Opportunities at NINDS *Northwestern/Ohio State, 6th Floor*
11:45 AM–1:00 PM	**Additional Workshops** 1. Summary of the ABPN MOC Program: Life-long Learning for Neurologists and Psychiatrists* *Lincolnshire I/II, 6th Floor* 2. 15th Annual Women of the ANA Lunch Program: Work Life Balance- Is it Attainable? *Chicago Ballroom FGH, 5th Floor*
1:15–3:15 PM	**Symposium:** Harnessing Plasticity: Technology for Motor and Cognitive Neurorehabilitation After Stroke and Neurotrauma *Chicago Ballroom A–E, 5th Floor*
3:15–3:30 PM	Coffee Break *Chicago Ballroom Foyer, 5th Floor*
3:30–5:30 PM	**Special Interest Group Symposia** 1. Autoimmune Neurology *Denver/Houston/Kansas City, 5th Floor* 2. Case Studies *Lincolnshire I/II, 6th Floor* 3. Dementia and Aging *Northwestern/Ohio State, 6th Floor* 4. Education *Addison, 4th Floor* 5. Headache and Pain* *Clark, 4th Floor* 6. Health Services Research *Michigan/Michigan State, 6th Floor* 7. Interventional Neurology *Indiana/Iowa, 6th Floor* 8. Neuromuscular Disease *Los Angeles/Miami/Scottsdale, 5th Floor* 9. Sleep Disorders and Circadian Rhythm *Purdue/Wisconsin, 6th Floor*
5:30–7:00 PM	**Poster Presentation & Reception II** *Grand Ballroom, 7th Floor*
7:30–9:00 PM	**President's Reception** *Chicago Ballroom, 5th Floor*

TUESDAY, SEPTEMBER 29, 2015

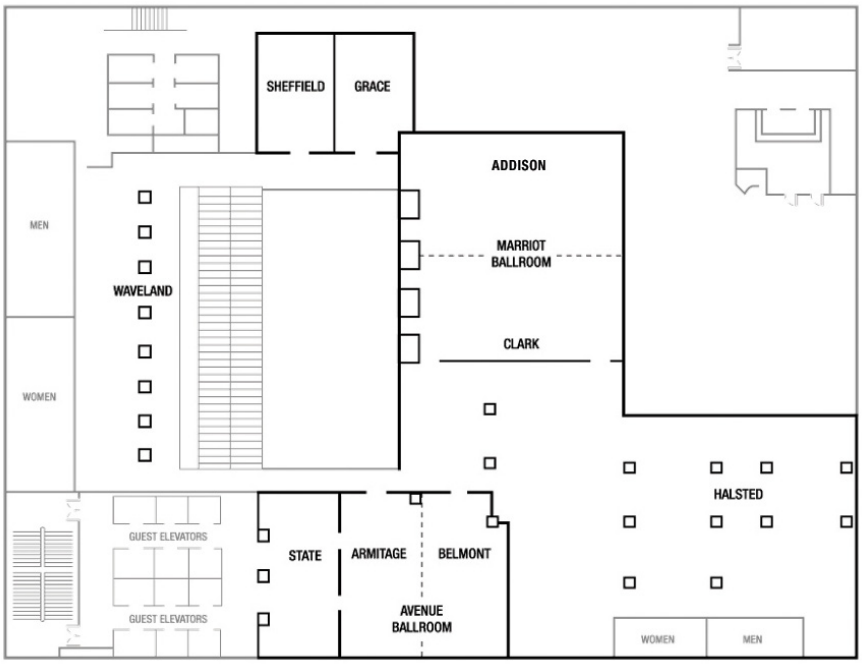
6:30 AM–2:00 PM	Registration Hours *5th Floor Registration*
7:00–9:00 AM	Continental Breakfast *Chicago Ballroom Foyer, 5th Floor*
7:30–9:00 AM	**Early Career Level Faculty Development Course III:** Grant Writing and Getting Funded *Clark, 4th Floor* **Mid/Senior Career Level Faculty Development Course III:** Becoming a Department Chair: Pitfalls, Challenges and Rewards *Avenue Ballroom, 4th Floor* **AUPN Chair Development Course III:** Chairing a Department: If You Can't Laugh You're Done *Addison, 4th Floor*
9:00–9:15 AM	Coffee Break *Chicago Ballroom Foyer, 5th Floor*
9:15–11:15 AM	**Presidential Symposium:** Evolving Concepts in Prion Biology: How Many Neurodegenerative Diseases are Caused by Prions *Chicago Ballroom A–E, 5th Floor*
11:30 AM–1:00 PM	Lunch *Chicago Ballroom Foyer, 5th Floor*
11:45 AM–1:00 PM	**Interactive Lunch Workshops** 1. A Clinician's Approach to Chronic Traumatic Encephalopathy *Denver/Houston/Kansas City, 5th Floor* 2. Epilepsy Genetics: Discoveries Through Team Science *Los Angeles/Miami/Scottsdale, 5th Floor* 3. Meet the Editors* *Michigan/Michigan State, 6th Floor* 4. Sex Disparities in Neurologic Research *Northwestern/Ohio State, 6th Floor* 5. Use of Retinal Imaging in the Diagnosis and Management of Alzheimer's and Other Neurological Diseases *Purdue/Wisconsin, 6th Floor*
11:45 AM–1:00 PM	**Additional Workshops** 1. ANA Liasons Lunch* *Clark, 4th Floor* 2. AUPN'S Networking Lunch for Small Academic Departments of Neurology* *Addison, 4th Floor*
1:15–3:15 PM	**Symposium:** Controversies in Stroke Therapeutics *Chicago Ballroom A–E, 5th Floor*
3:15 PM	Meeting Adjournment

FLOOR PLANS

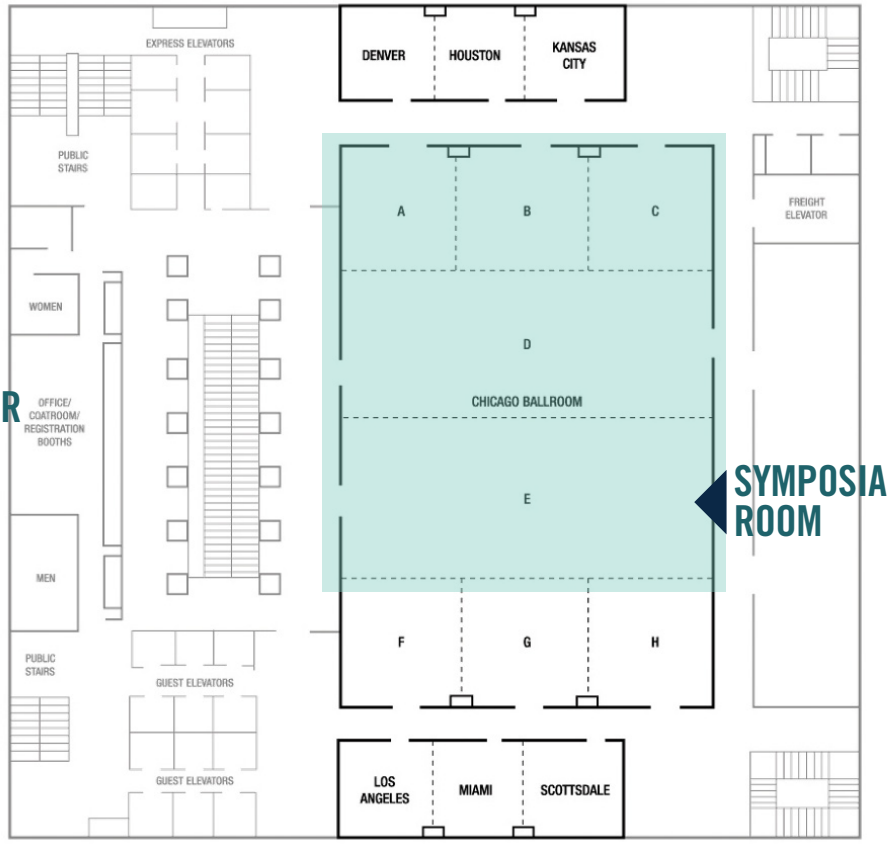
3RD FLOOR MEETING ROOMS



4TH FLOOR MEETING ROOMS



5TH FLOOR MEETING ROOMS



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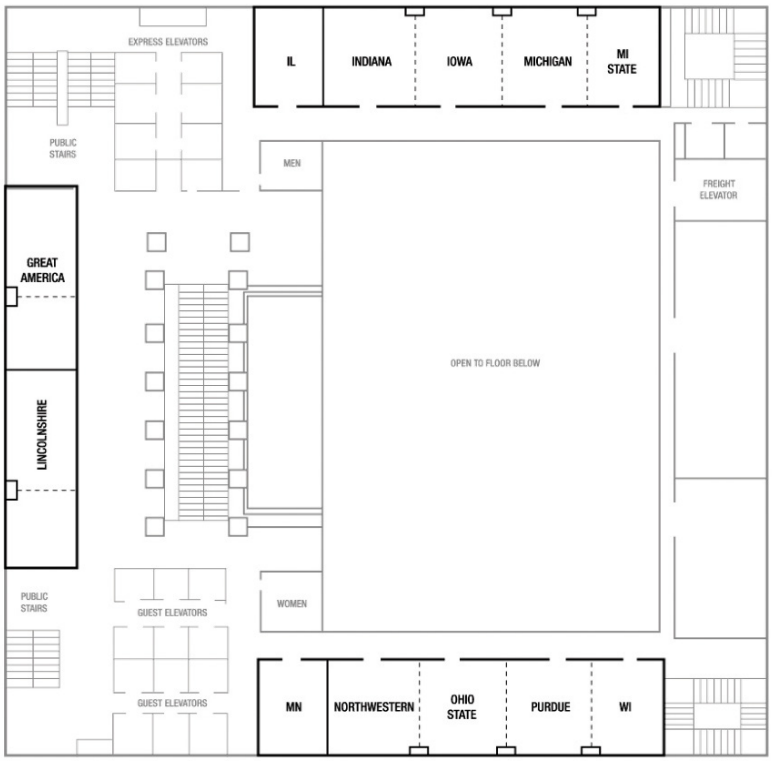
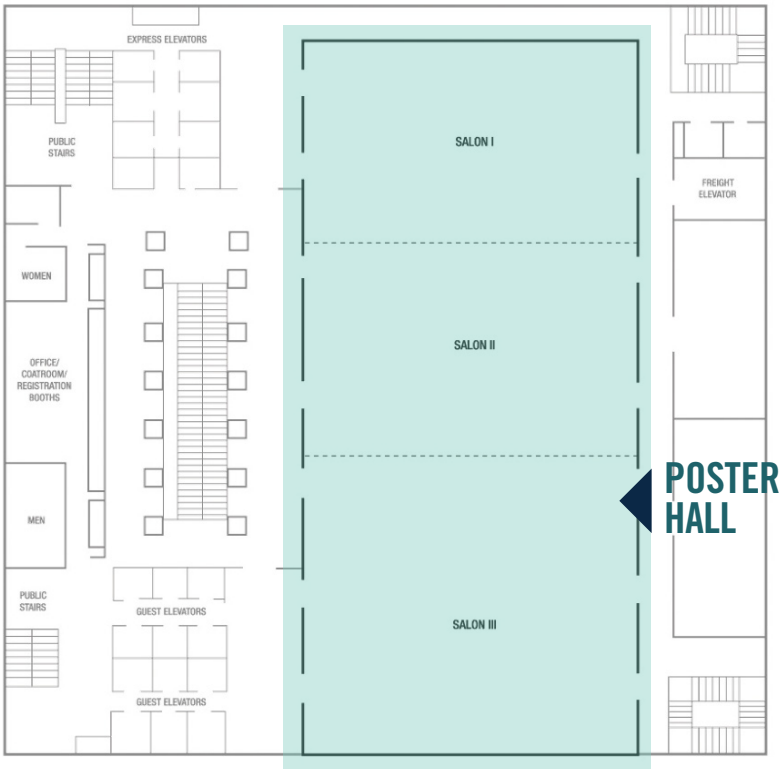
6TH FLOOR MEETING ROOMS



7TH FLOOR MEETING ROOMS

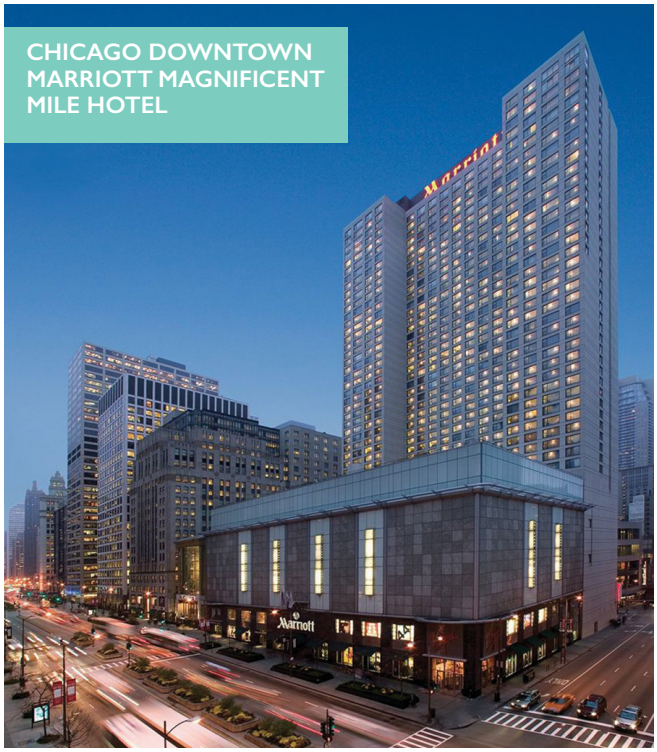


POSTER HALL

GENERAL INFORMATION

HOTEL INFORMATION

Chicago Marriott Downtown Magnificent Mile
540 North Michigan Avenue
(Driveway Entrance on 541 North Rush Street)
Chicago, IL 60611
Main Phone: (312) 836-0100
Check-In Time: 4:00 PM
Check-Out Time: 12:00 PM



ON-SITE REGISTRATION HOURS

5th Floor Registration

Saturday, September26	3:00 PM–7:00 PM
Sunday, September 27	6:00 AM–5:45 PM
Monday, September 28	6:30 AM–5:45 PM
Tuesday, September 29	6:30 AM–2:00 PM

POSTER VIEWING HOURS

Grand Ballroom, 7th Floor

Sunday, September 27 11:00 AM–7:00 PM
Poster presenters will be in attendance from 5:30 PM to 7:00 PM

Monday, September 28 11:00 AM–7:00 PM
Poster presenters will be in attendance from 5:30 PM to 7:00 PM

The 2015 ANA Abstracts will be available in the *Annals of Neurology* Journal
The supplement is:
Annals of Neurology
Volume 78, Supplement 19, 2015

SPEAKER READY ROOM

McHenry, 3rd Floor

Saturday, September 26	3:00 PM–7:00 PM
Sunday, September 27	6:00 AM–5:45 PM
Monday, September 28	6:30 AM–5:45 PM
Tuesday, September 29	6:30 AM–2:00 PM

LUNCH

Chicago Ballroom Foyer, 5th Floor

Sunday, September 27	11:30 AM–1:00 PM
Monday, September 28	11:30 AM–1:00 PM
Tuesday, September 29	11:30 AM–1:00 PM

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

PRESS ROOM

Cook, 3rd Floor

Saturday, September 26	3:00 PM–7:00 PM
Sunday, September 27	6:00 AM–5:45 PM
Monday, September 28	6:30 AM–5:45 PM
Tuesday, September 29	6:30 AM–2:00 PM

WIRELESS CONNECTION

Guest Room Wi-Fi: All guest rooms booked under the ANA block will be provided with complimentary high-speed wireless internet. To connect to the WI-FI use the instructions provided to you in your hotel room.

Meeting Room Wi-Fi: To connect, turn on Wi-Fi in the device. Look to the network SSID: **ANA2015**. A splash page will pop up for Guest-Tek. Enter the Passcode: **ANA2015** (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.

MOBILE APP

The ANA is pleased to continue the mobile application for the 2015 Annual Meeting.

How to download: For phones with iTunes or Google Play: Visit the app store or Google Play on your phone and search for **ANA Meetings**. For all other web-enabled devices, including those listed above and Blackberry, enter http://m.core-apps.com/ana_annual2015 to be automatically directed to the proper download version for your phone. Once downloaded, select the ANA 2015 Annual Meeting to access the meeting information.

Should you have any questions please contact support@core-apps.com.

CONTINUED

CONTINUING MEDICAL EDUCATION:
ACCREDITATION & DESIGNATION STATEMENT(S)

American Neurological Association 140th 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 American Neurological Association designates this live activity for a maximum of 24.25 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

The Circuits and Circuit Disorders: Approaches to Neuromodulation Symposium
This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the American Neurological Association and American Medical Association. The ANA is accredited by the ACCME to provide continuing medical education for physicians.

The American Neurological Association designates this live activity for a maximum of 3.0 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

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

EVALUATIONS ONLINE

Within a week following the event, you will receive an email containing a link to the ANA evaluation site. 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, Robin Geary, at rgeary@myana.org or **(856) 793-0804**.

CONSENT TO USE OF PHOTOGRAPHIC IMAGES

Registration and attendance at, or participation in, ANA meetings and other related activities constitutes an attendee's authorization of ANA's use and distribution (both now and in the future) of the attendee's image or voice in photographs, video recordings, electronic reproductions, audio recordings and other media throughout the world and royalty free.

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 2015 Annual Meeting.

PROGRAM

SATURDAY, SEPTEMBER 26

3:00 – 7:00 PM **REGISTRATION**
5th Floor Registration

6:00 – 10:00 PM **SYMPOSIUM: CIRCUITS AND CIRCUIT DISORDERS: APPROACHES TO NEUROMODULATION**

6:00 – 7:00 PM **BUFFET DINNER**
Halstead, 4th Floor

7:00 – 10:00 PM **SYMPOSIUM**
Marriott Ballroom, 4th Floor

Co-Chairs: Roger N. Rosenberg, M.D., *University of Texas Southwestern Medical Center; Editor, JAMA Neurology*
Clifford Saper, M.D., Ph.D., *Harvard Medical School; Editor, Annals of Neurology*

7:00 – 7:25 PM
Circuits, Circuit Disorders and Neurologic Signs and Symptoms
Mahlon DeLong, M.D., *Emory University*

7:30 – 7:55 PM
Circuit Mechanisms of Dystonia: Insights from Combined Cortical and Basal Ganglia Recordings in Humans Undergoing Deep Brain Stimulator Implantation
Phillip A. Starr M.D., Ph.D., *University of California San Francisco*

8:00 – 8:25 PM
Deep Brain Stimulation for Tourette Syndrome and Related Disorders
Jonathan W. Mink, M.D., Ph.D., *University of Rochester*

8:30 – 8:55 PM
Therapeutic Modulation of Cingulate-Cortical Pathways in Major Depression
Helen S. Mayberg, M.D., *Emory University*

9:00 – 9:25 PM
Chemogenetics: A Robust Translational Platform for Cell-type Specific Neuromodulation
Bryan L. Roth, M.D., Ph.D., *University of North Carolina*

9:30 – 10:00 PM
General Open Discussion

The 2014 Lasker-DeBakey Clinical Medical Research Award recipients are Mahlon R. DeLong, Emory University, and Alim-Louis Benabid, Universite Joseph Fourier, for developing deep brain stimulation (DBS) of the subthalamic nucleus as effective therapy for Parkinson's disease (PD) patients. DBS has been has been shown to be effective to reduce bradykinesia and tremors, and in general restores motor ability in patients with PD. DBS has made it possible to reduce the dose of Levodopa (L-dopa) and its side-effects. It has been administered to over 100,000 patients and is widely credited to be the most important scientific advance in therapy for PD since the introduction of L-dopa in the 1960s.

DBS has been applied more recently for other neurological and psychiatric disorders including: tardive dyskinesias, cluster headaches, Tourette syndrome, epilepsy, obesity, depression, anorexia, addictions, and obsessive compulsive disorders. As Dr. DeLong has emphasized, "DBS is not disease specific, but rather circuit-specific, since the same target may be used to treat a variety of movement disorders."

This symposium is a tribute to Dr. DeLong and his colleagues for the achievement of DBS and features Dr. DeLong, who will review the concepts of circuits and circuit disorders and the research which led to the clinical use of DBS for tremor, Parkinson's disease, and a wide variety of neurologic and psychiatric disorders. Philip Starr will present his work on circuit mechanisms of dystonia and results of DBS. Jonathan Mink will focus on results of recent studies of DBS in Tourette syndrome and obsessive compulsive disorder, while Helen Mayberg will discuss DBS as an emerging experimental treatment strategy for patients with intractable major depression. Bryan Roth will summarize a chemogenetic platform known as designer receptors exclusively activated by designer drugs (DREADDs) that provides remote control of neuronal activity in a cell-type specific and non-invasive manner and its use to control neurons in freely moving animals.

SUNDAY, SEPTEMBER 27

6:00 AM – 5:45 PM **REGISTRATION**
5th Floor Registration

7:00 – 9:00 AM **CONTINENTAL BREAKFAST**
Chicago Ballroom Foyer, 5th Floor

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

EARLY CAREER LEVEL FACULTY DEVELOPMENT COURSE I:
Finding Happiness: Panel Discussions in Lab, Translational and Clinical Research
Clark, 4th Floor

Chairs: David Greer, M.D., M.A., F.C.C.M., F.A.N.A., F.N.C.S., *Yale University*
Joachim M. Baehring, M.D., D.Sc., *Yale University*

Faculty: Susanne Muehlschlegel, M.D., M.P.H., F.N.C.S., *University of Massachusetts Medical School*
Ralph Josefowicz, M.D., *University of Rochester School of Medicine*
Babar Kokhar, M.D., M.B.A., *Yale School of Medicine*
Charlotte Sumner, M.D., *Johns Hopkins University*
Gilmore O'Neil, M.D., *Biogen Idec, Inc.*

This course will provide samples of the different careers in neurology, ranging from the clinician-scientist to the clinician-educator, from work-life balance to a career in industry/biomedicine. The speakers will give brief talks to explain how they've found their career paths, the bumps along the way, and how they've found happiness in the process.

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

MID/SENIOR CAREER LEVEL FACULTY DEVELOPMENT COURSE I:

MBA in 1.5 Hours: How an Academic Medical Center Actually Works

Avenue Ballroom, 4th Floor

Chairs: Amy Pruitt, M.D., *University of Pennsylvania*
Roy Hamilton, M.D., M.S., *University of Pennsylvania*

Faculty: Teresa Costantinidis, *University of California, San Francisco*

In today's healthcare climate, leadership in neurology requires a skill set that includes not only clinical acumen or research expertise, but also managerial, financial, and administrative competency. Changing trends triggered by national health care reform further underscore the need to maintain a balance between clinical care and business savvy. However, while these skills are becoming increasingly important for advancing the practice of neurology—and medicine in general—they are typically not included in medical or neurologic training. This session will discuss basic tools and strategies employed in the business arena that neurologists can use to enhance their leadership skills.

AUPN CHAIR DEVELOPMENT COURSE I

The Role of "Role Players" in the 21st Century Department

Addison, 4th Floor

Chair: David Fink, M.D., *University of Michigan*

Faculty: David Lee Gordon, M.D., F.A.A.N., F.A.N.A., F.A.H.A., *University of Oklahoma*
Steven L. Small, M.D., Ph.D., *University of California, Irvine*

Changes in healthcare delivery and reimbursement, a progressively competitive marketplace, and shrinking research revenue have forced academic medicine to address demands for improved clinical performance and productivity. The combination of these forces has driven a move away from the ideal of a triple-threat academician towards a division of labor along the core missions - patient care, education, and research. This course will consist of a short presentation about mission-based hiring (David Lee Gordon, University of Oklahoma), a short presentation about the incorporation of general and community neurologists into an academic practice (Steven L. Small, University of California, Irvine), and comments from a panel of chairs who have dealt with these issues followed by an open discussion session.

9:00 – 9:15 AM COFFEE BREAK

Chicago Ballroom Foyer, 5th Floor

9:15 – 11:15 AM SYMPOSIUM: CAUSES/TRIGGERS OF MULTIPLE SCLEROSIS

Chicago Ballroom A–E, 5th Floor

Chairs: Robert T. Naismith, M.D., *Washington University in St. Louis*
David A. Hafler, M.D., *Yale New Haven Hospital*

The cause of multiple sclerosis (MS) has remained an enigma, hypothesized to be triggered by an aberrant immunologic response to an environmental insult in a genetically susceptible individual. Recent investigations have provided important insights into factors that lead to the initiation and progression of MS. Leading experts will provide a comprehensive and practical review interlinking the roles of relevant genes, environmental triggers and immune responses resulting in MS as a clinical syndrome.

Learning Objectives: Having completed this symposium, participants will be able to:

1. Identify genetic variants associated with risk of developing MS.
2. Enumerate the environmental factors associated with MS development and disease severity.
3. Identify how the immune system responds to limit injury from inflammatory demyelination

9:20 – 9:45 AM
Presentation from the Adams Awardee
Genetic Variation Leading to Risk of MS
David A. Hafler, M.D., *Yale School of Medicine and The Broad Institute of MIT*

9:50 – 10:15 AM
Environmental Determinants of MS Risk
Alberto Ascherio, M.D., Dr.P.H., *Harvard School of Public Health*

10:20 – 10:45 AM
Environmental Factors Interacting with Genes
Lisa F. Barcellos, Ph.D., M.P.H., *University of California at Berkeley*

10:50 – 11:15 AM
No Quiet Surrender: Molecular Guardians in MS Brain
Larry Steinman, M.D., *Stanford University*

11:00 AM – 7:00 PM POSTER VIEWING

Grand Ballroom, 7th Floor

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

11:15 – 11:45 AM NEW MEMBER MEET AND GREET*

Chicago Ballroom F, 5th Floor

11:30 AM – 1:00 PM LUNCH

Chicago Ballroom Foyer, 5th Floor

Boxed lunches are available to be taken into Interactive Lunch Workshops

PROGRAM

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

These workshops are "lunch and learns".

CURRENT ETHICAL DILEMMAS IN NEUROLOGY

Purdue/Wisconsin, 6th Floor

Moderator: Zachary Simmons, M.D., *Penn State Hershey Medical Center*

Faculty: Daniel Larriviere, M.D., J.D., *Ocshner Neuroscience Institute*
Zachary Simmons, M.D., *Penn State Hershey Medical Center*
David M. Greer, M.D., *Yale University School of Medicine*
Satish V. Khadilkar, *M.B.B.S, M.D., D.M., D.N.B.E., F.I.A.N., Grant Medical College and Sir J.J. Group of Hospitals*

This program features three topics for discussion that are relevant to clinical neurologists and that pose ethical dilemmas:

1. Physician-Assisted Suicide: Ethically Permissible?
2. Right-to-Try: Patient Autonomy vs. Physician Nonmaleficence.
3. The Role of fMRI in Blurring the Line Between Consciousness and Unconsciousness

EMERGING THERAPEUTIC TARGETS FOR DEMYELINATION AND REMYELINATION

Los Angeles/Miami/Scottsdale, 5th Floor

Moderator: Jun Li, M.D., Ph.D., *Vanderbilt University*
Co-Moderator: Thomas Lloyd, M.D., Ph.D., *Johns Hopkins University*

Faculty: Stephen Hauser, M.D., *University of California, San Francisco*
Rhonda Voskuhl, M.D., *University of California, Los Angeles*
Brian Popko, Ph.D., *University of Chicago*

This workshop will discuss the following specific areas:

1. How do genetic factors render susceptibility to demyelination and how will these factors be modified?
2. Role of membrane trafficking in demyelination
3. Molecular targets for therapeutic development against CNS/PNS demyelination

MEET THE NINDS*

Indiana/Iowa, 6th Floor

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

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

NEUROLOGY GOES GLOBAL - INTERNATIONAL ISSUES IN DIAGNOSTICS & THERAPEUTICS

Northwestern/Ohio State, 6th Floor

Moderators: Farrah Mateen, M.D., Ph.D., *Massachusetts General Hospital, Harvard Medical School*
Shri Mishra, M.D., M.S., A.B.M.S., F.A.A.N., F.N.A.A., F.A.N.A., *David Geffen School of Medicine, University of California*
Robert Brown, D.Phil, M.D., *University of Massachusetts Medical School*

Faculty: Shri Mishra, M.D., M.S., A.B.M.S., F.A.A.N., F.N.A.A., F.A.N.A., *David Geffen School of Medicine, University of California*
Farrah Mateen, M.D., Ph.D., *Massachusetts General Hospital, Harvard Medical School*
Ildefonso Rodriguez Levya, M.D., *Hospital Central Dr. Ignacio Morones Prieto, San Luis Potosi, Mexico*
Arbinda Mukherjee, *M.D., D.M., President, Indian Academy of Neurology, Vivekanand Institute, Kolkata*

Neurological diseases are a leading cause of death and disability globally. Most people with neurological disorders – including epilepsy, dementia, stroke, meningitis, and traumatic brain injury – live in low- and middle-income countries. The consequences of neurological diseases in resource-limited settings can have especially profound socioeconomic and health consequences. This program will engage the international participants of the ANA, from low-, middle-, and high-income countries, including India, Mexico, and the U.S. Collaborations among neurologists and neuroscientists will be discussed. The proposed speakers will be researchers and career advocates of global neurology, working collectively in Africa, Asia, and the Americas.

NON-LESIONAL, DRUG-RESISTANT FOCAL EPILEPSY: CONTROVERSIES IN DIAGNOSTIC EVALUATION AND SURGICAL MANAGEMENT

Michigan/Michigan State, 6th Floor

Moderator: Gregory Cascino, M.D., *Mayo Clinic of Rochester*
Co-Moderator: Thomas Bleck, M.D., *Rush Medical College*

Faculty: Lara E. Jehi, M.D., *Cleveland Clinic*
Gregory A. Worrell, M.D., Ph.D., *Mayo Clinic*
Nitin Tandon, M.D., *University of Texas Health Science Center at Houston Medical School*
P. Satish Chandra, M.B.B.S., D.M., *Director / Vice-Chancellor, National Institute of Mental Health and Neuro Sciences (NIMHANS)*

Patients with drug-resistant focal epilepsy and normal magnetic resonance imaging studies are less favorable candidates to be rendered seizure-free following surgical treatment. Potential issues include difficulty localizing the epileptogenic zone, involvement of functional cerebral cortex, and ineffective operative strategies. Identification of patients with non-lesional focal epilepsy and a surgically remediable epileptic syndrome may require chronic intracranial EEG and additional functional neuroimaging studies. The rationale for the present interactive lunch workshop is to identify the controversies in the diagnostic evaluation and surgical management of non-lesional drug-resistant focal epilepsy.

NOVEL BRAIN MRI AND THE HUMAN CONNECTOME: INSIGHTS AND IMPLICATIONS FOR THE NEUROLOGIST

Denver/Houston/Kansas City, 5th Floor

Moderator: Sudha Seshadri, M.D., *Boston University School of Medicine*

Faculty: Maurizio Corbetta, M.D., *Washington University School of Medicine*
Meredith N. Braskie, Ph.D., *University of Southern California*

President Obama announced the BRAIN (Brain Research through Advancing Innovative Neurotechnologies) Initiative as 'a bold new research effort to revolutionize our understanding of the human mind and uncover new ways to treat, prevent, and cure brain disorders like Alzheimer's, stroke, schizophrenia, autism, epilepsy, and traumatic brain injury.' A key component of this initiative is the Human Connectome Project. Many neurologists are familiar with diffusion weighted imaging on brain MRI and some use tractography in clinical research settings but have only a vague understanding of the details and vast potential of this project. This ILW will be an opportunity to interact with leading clinician-investigators in this field, to ask questions, share your own ideas on its promise and pitfalls. There will be an introduction to Brain Connectome studies, their scope and clinical translational, especially in stroke, dementia and TBI research and treatment.

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

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

Chicago Ballroom A–E, 5th Floor

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

The Derek Denny-Brown Young Neurological Scholar Symposium is an opportunity for young researchers to share groundbreaking research in the field of Neurology. This symposium will feature sessions from the three 2015 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 2015, 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 two (2) Physician Scientist – Basic and one (1) Physician Scientist – Clinical.

The 2015 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.

Learning Objectives: Having completed this symposium, participants will be able to:

1. Identify the impact dietary reversal model had on the mice
2. List de novo causes of epilepsy recently identified through collaborative efforts across many institutions
3. Describe how the blood-brain barrier is altered in distinct ways at different stages of lesion formation, and in particular how these alterations are reflected in magnetic susceptibility changes detectable using ultra-high-field (7 tesla) MRI
4. Evaluate if tau reduction–based therapeutic approaches could have even broader indications than AD and could also include forms of epilepsy

1:20 – 1:45 PM
Presentation of Derek Denny-Brown: Young Neurological Award in Basic Science
The Genetics of Epilepsy: A Complex Architecture
Annapurna Poduri, M.D., M.P.H., *Harvard Medical School*

1:48 – 2:13 PM
Presentation of Derek Denny-Brown: Young Neurological Award in Basic Science
Tau, Hyperexcitability, and Alzheimer's Disease
Erik Roberson, M.D., Ph.D., *University of Alabama at Birmingham*

2:16 – 2:41 PM
Presentation of Derek Denny-Brown: Young Neurological Award in Clinical Science
Advanced Imaging of Lesion Repair in Multiple Sclerosis
Daniel Reich, M.D., Ph.D., *National Institute of Health, National Institute of Neurological Disorders*

2:44 – 2:59 PM
Presentation from the Grass Awardee
Longevity Factor Klotho Enhances Cognition and Confers Resilience Against Neurodegenerative Pathologies
Dena Dubal, M.D., Ph.D., *University of California, San Francisco*

3:03 – 3:15 PM
Presentation from the Wolfe Research Prize Awardee
Dietary Reversal of Neuropathy in a Murine Model of Prediabetes
Lucy Hinder, Ph.D., *University of Michigan*

3:15 – 3:30 PM COFFEE BREAK

Chicago Ballroom Foyer, 5th Floor

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

BEHAVIORAL NEUROLOGY

Advances in Understanding Large-scale Human Brain Networks and Their Dysfunction in Neurological Diseases
Indiana/Iowa, 6th Floor

Chair: Marilu Gorno Tempini, M.D., Ph.D., *University of California, San Francisco*
Co-Chair: Brad Dickerson, M.D., *Massachusetts General Hospital*

In this session, the speakers will review several major human brain networks subserving cognitive function and recent advances in our understanding of their functional neuroanatomy and connections.

Abnormalities of these networks in patients with neurological disorders, including aphasia and neglect, will be discussed.

LEADER IN THE FIELD PRESENTATION
3:30 – 3:50 PM
Connections Required to Connect: Networks Underlying Recognition and Sharing in Others Emotions
Argye Hillis, M.D., M.A., *Johns Hopkins University School of Medicine*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION
4:00 – 4:08 PM
Modulating Large-Scale Neurocognitive Networks Using Targeted Noninvasive Stimulation
Joel Voss, Ph.D, *Northwestern University Feinberg School of Medicine*

4:08 – 4:10 PM
Q&A

PROGRAM

LEADER IN THE FIELD PRESENTATION
4:10 – 4:30 PM
Connections and Network Coherence in PPA
Marsel Mesulam, M.D., *Northwestern University Medical School*

4:30 – 4:40 PM
Q&A

DATA BLITZ PRESENTATION
4:40 – 4:48 PM
Changes in Functional Connectivity Between Speech-Specific Cortical Regions in Developmental Stuttering
David Rosenfield, M.D., *Houston Methodist Hospital/Weill Cornell College of Medicine*

4:48 – 4:50 PM
Q&A

LEADER IN THE FIELD PRESENTATION
4:50 – 5:10 PM
Lesion Topography and Functional Connectivity Differentially Predicts Sensorimotor and Cognitive Deficits After Stroke
Maurizio Corbetta, M.D., *Washington University School of Medicine*

5:10 – 5:20 PM
Q&A

DATA BLITZ PRESENTATION
5:20 – 5:28 PM
Convergent Excitability Defects in Prefrontal Corticothalamic Pyramidal Neurons Link Genes to Behavior in Mouse Models of Autism
Audrey Brumback, M.D., Ph.D, *University of California, San Francisco*

5:28 – 5:30 PM
Q & A

CEREBROVASCULAR DISEASE

Los Angeles/Miami/Scottsdale, 5th Floor

Chair: Anthony Kim, M.D., M.S., *University of California, San Francisco*
Co-Chair: Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins University*

This session brings together leading stroke experts for focused presentations on recent new evidence and controversies in stroke as well as for data-blitz presentations by the top cerebrovascular abstracts from the meeting.

LEADER IN THE FIELD PRESENTATION
3:30 – 3:50 PM
Atrial Cardiopathy and Cryptogenic Stroke
Hooman Kamel, M.D., *Weill Cornell Medical College*

DATA BLITZ PRESENTATION
3:50 – 4:00 PM
Diet and Carotid Atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Carotid MRI Study
Jennifer Dearborn, M.D., M.P.H., *Yale University School of Medicine*

LEADER IN THE FIELD PRESENTATION
4:00 – 4:20 PM
Evidence-based Primary Prevention of Stroke: Published Guidelines and Recent Advances
James Meschia, M.D., *Mayo Clinic*

DATA BLITZ PRESENTATION
4:20 – 4:30 PM
Regulatory T cell Transplantation Attenuates Hemorrhage Transformation in Stroke Mice After Thrombolytic Treatment with rtPA
Xiaoming Hu, M.D., Ph.D., *University of Pittsburgh*

LEADER IN THE FIELD PRESENTATION
4:30 – 4:50 PM
The Era of Endovascular Stroke Therapy: A Vascular Neurologist's Perspective
Shyam Prabhakaran, M.D., M.S.,*Northwestern University, Feinberg School of Medicine*

DATA BLITZ PRESENTATION
4:50 – 5:00 PM
Obesity Paradox in Patients Hospitalized for Acute Ischemic Stroke: An Analysis of Nationwide Inpatient Sample Data
Urvish Patel, M.D., *Icahn School of Medicine at Mount Sinai*

LEADER IN THE FIELD PRESENTATION
5:00 – 5:20 PM
Management of Intracranial Hemorrhage in Patients on Warfarin and NOACs
Jose Biller, M.D., F.A.C.P., F.A.A.N., F.A.H.A., *Loyola University Health System*

DATA BLITZ PRESENTATION
5:20 – 5:30 PM
Increased Blood Pressure Variability after Endovascular Thrombectomy is Associated with Worse Outcome
Alicia Bennett, D.O., *University of Utah*

EPILEPSY

Big Data: From Understanding Epilepsy to Individualized Patient Management
Purdue/Wisconsin, 6th Floor

Chair: Robert Knowlton, M.D., M.S.P.H., *University of California, San Francisco*
Co-Chair: Lara Jehi, M.D., *Cleveland Clinic Foundation*

The session consists of a series of "paired" talks first discussing the use of "big data" to understand epilepsy and next illustrating its use to manage individual patients. Topics to be tackled vary from diagnostic imaging (network studies to source localization), to electrophysiology (high- and low-frequency analyses for seizure prediction to the application of electrical neurostimulation in direct patient care) to epilepsy surgery (meta-analyses to predictive modeling). The goal is to provide an overview of the current hot areas of research and future directions within the field of epilepsy.

LEADERS IN THE FIELD PRESENTATIONS
3:30 – 3:50 PM
Introduction: Source Modeling as Example
Robert Knowlton, M.D., M.S.P.H., *University of California, San Francisco*

3:50 – 4:10 PM
Seizure Predication and Detection
Gregory Worrell, M.D., Ph.D., *Mayo Clinic, Rochester*

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

4:10 – 4:30 PM
Applying Electrocorticography and Evoked Potentials to Surgical Planning
Dileep Nair, M.D., *Cleveland Clinic Foundation*

4:30 – 4:35 PM
Break

4:35 – 4:55 PM
Metanalyses in Epilepsy Surgery Outcomes
Frank Gilliam, M.D., M.P.H., *Penn State, Milton S. Hershey Medical Center*

4:55 – 5:15 PM
Individualized Surgery Outcome Prediction
Lara Jehi, M.D., *Cleveland Clinic Foundation*

5:15 – 5:30 PM
Panel Q&A/Adjourn

MOVEMENT DISORDERS

Update on Movement Disorders: Insights on Circuit Modulation and Molecular Cellular Mechanisms
Denver/Houston/Kansas City, 5th Floor

Chair: Nicole Calakos, M.D., Ph.D., *Duke University*
Co-Chair: Peter Todd, M.D., Ph.D., *University of Michigan*

Rapidly developing technologies in the areas of neuromodulation, imaging, human genetics, and molecular and cellular methods to study human disease are helping accelerate our understanding of movement disorders. In this update, presenters will discuss the latest findings that advance our understanding of the mechanisms and therapeutic opportunities for movement disorders.

LEADER IN THE FIELD PRESENTATION

3:30 – 3:50 PM
Genetic, Optogenetic and Chemogenetic Interrogation and Correction of Parkinsonian Activity in the Subthalamic Nucleus
Mark Bevan, Ph.D., *Northwestern University*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION

4:00 – 4:08 PM
DTI-Based Biomarker of Clinical Severity in Essential Tremor
Ludy Shih, M.D., *BIDMC, Harvard Medical School*

4:08 – 4:10 PM
Q&A

LEADERS IN THE FIELD PRESENTATIONS

4:10 – 4:30 PM
Helen Bronte-Stewart, M.D., M.S., *Stanford University Medical Center*

4:30 – 4:40 PM
Q&A

4:40 – 4:55 PM
Repeat Associated Non-AUG (RAN) Translation: a New Contributor to Human Neurodegenerative Disease
Peter J. Todd, M.D., Ph.D., *University of Michigan*

4:55 – 5:00 PM
Q&A

5:00 – 5:15 PM
Phenotypic Screening Strategies for Neurodegeneration
Vikram Khurana, M.D., Ph.D., *Massachusetts General Hospital, Harvard Medical School*

5:15 – 5:20 PM
Q&A

DATA BLITZ PRESENTATION

5:20 – 5:28 PM
In Vivo Multiphoton Imaging in a Mouse Model of Parkinsonism Demonstrates Progressive Aggregation of Alpha-Synuclein and Selective Degeneration of Lewy Inclusion-Bearing Neurons
Vivek K. Unni, M.D., Ph.D., *Oregon Health & Science University*

5:28 – 5:30 PM
Q&A/Adjourn

MULTIPLE SCLEROSIS

Northwestern/Ohio State, 6th Floor

Chair: Ari Green, M.D., M.C.R., *University of California, San Francisco*
Co-Chair: Robert Naismith, M.D., *Washington University in St. Louis*

This session will focus on multiple sclerosis (MS) as a neurodegenerative disease. 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 PRESENTATIONS

3:30 – 3:45 PM
Evaluation of Serum JCV-Test and INDEX Values in Natalizumab-Treated Individuals
Clementine Karageorgiou, M.D., Ph.D., *Iatriko Hospital of Athens, Maroussi, Athens, Greece*

3:45 – 4:00 PM
Phase II Trial of Dalfampridine to Improve Visual Function in Chronic Optic Neuritis Due to MS
Robert Naismith, M.D., *Washington University in St. Louis*

4:00 – 4:15 PM
Demyelination and Iron Accumulation in Subcortical Gray Matter (GM) in Multiple Sclerosis (MS)
Vasily Yarnyk, Ph.D., *University of Washington*

4:15 – 4:30 PM
A Multiple Sclerosis Disease-Risk Variant in EVI5 Links Susceptibility to the S1P Pathway
Alessandro Didonna, Ph.D., *University of California, San Francisco*

4:30 – 4:45 PM
In Vivo Quantitative Evaluation of Cortical Gray Matter Damage in Multiple Sclerosis
Jie Wen, Ph.D., *Washington University in St. Louis*

4:45 – 5:00 PM
The Perivascular Macrophage is a Molecularly Distinct Antigen Presenting Cell at the Blood-Brain Barrier
Gregory Wu, M.D., Ph.D., *Washington University in St. Louis*

PROGRAM

5:00 – 5:15 PM
Integrated Investigation of Multiple Sclerosis Risk Factors in First-Degree Relatives to Pave the Way for Primary Prevention
Zongqi Xia, M.D., Ph.D., *Brigham and Women's Hospital and Harvard Medical School*

5:15 – 5:30 PM
Q&A/Adjourn

NEUROCRITICAL CARE

Michigan/Michigan State, 6th Floor

Chair: J. Claude Hemphill III, M.D., M.A.S., F.N.C.S., F.A.N.A., *University of California, San Francisco*
Co-Chair: Paul Nyquist, M.D., *Johns Hopkins University*

Neurocritical Care is more than stroke and head trauma. This session will focus on the clinical and scientific interface of topics related to electricity, immunity, and metabolism that impact patients with a range of acute neurological conditions.

LEADER IN THE FIELD PRESENTATION
3:30 – 3:50 PM
New Onset Refractory Status Epilepticus, "NORSE" Syndrome and Immune Encephalopathy
Paul Nyquist, M.D., *Johns Hopkins University*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION
4:00 – 4:10 PM
Surgical Treatment of Traumatic Subdural Hematoma: Weekday vs. Weekend Admission
Kavelin Rumalla, *University of Kansas Medical Center*

LEADER IN THE FIELD PRESENTATION
4:10 – 4:30 PM
Managing Edema in Ischemic Stroke: From the Molecular to the Mechanical
W. Taylor Kimberly, M.D., Ph.D., *Massachusetts General Hospital, Harvard Medical School*

4:30 – 4:40 PM
Q&A

DATA BLITZ PRESENTATION
4:40 – 4:50 PM
Quantification of Brain Edema Changes After 23% Hypertonic Saline in Liver Failure
Andrew Naidech, M.D., M.S.P.H., *Northwestern University*

LEADER IN THE FIELD PRESENTATION
4:50 – 5:10 PM
ICU-Acquired Weakness: Why Did This Happen to My Patient?
J. Claude Hemphill III, M.D., M.A.S., F.N.C.S., F.A.N.A., *University of California, San Francisco*

5:10 – 5:20 PM
Q&A

DATA BLITZ PRESENTATION
5:20 – 5:30 PM
Engineering a Regeneration Permissive Environment Allowing for Recovery After Complete Spinal Cord Transection
Jeffrey Hakim, Ph.D., *Mayo Clinic*

5:30 PM
Q & A / Adjourn

NEURO-ONCOLOGY

Lincolnshire I/II, 6th Floor

Chair: Amy Pruitt, M.D., *University of Pennsylvania*
Co-Chair: John Joseph Laterra, M.D., Ph.D., *Johns Hopkins Hospital, Kennedy Krieger Institute*

This session will focus on recent developments in cancer therapeutics, their potential neurotoxicities and their applications to nervous system malignancies.

LEADER IN THE FIELD PRESENTATION
3:30 – 3:50 PM
Targeting ID01 to Increase Immunotherapeutic Efficacy in Brain Cancer
Derek Wainwright, Ph.D., *Northwestern University Feinberg School of Medicine*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION
4:00 – 4:08 PM
Prevalence of Chemotherapy Induced Peripheral Neuropathy
Noah Kolb, M.D., *University of Utah*

4:08 – 4:10 PM
Q&A

LEADER IN THE FIELD PRESENTATION
4:10 – 4:30 PM
CNS-Immune Reconstitution Inflammatory Syndrome
Avindra Nath, M.D., *National Institute of Neurological Disorders and Stroke, National Institutes of Health*

4:30 – 4:40 PM
Q&A

DATA BLITZ PRESENTATION
4:40 – 4:48 PM
Everolimus for Subependymal Giant Cell Astrocytoma Associated with Tuberous Sclerosis Complex
David Franz, M.D., *Cincinnati Children's Hospital Medical Center*

4:48 – 4:50 PM
Q&A

LEADER IN THE FIELD PRESENTATION
4:50 – 5:20 PM
PD-1/PD-L1 and CTLA-4 as Immunotherapeutic Targets in CNS Tumors
Rimas V. Lukas, M.D., *University of Chicago*

5:20 – 5:30 PM
Q&A/Adjourn

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

5:30 – 7:00 PM POSTER PRESENTATIONS AND RECEPTION #1

Grand Ballroom, 7th Floor

POSTER CATEGORIES

Behavioral Neurology	Poster #S101 – S118
Cerebrovascular Disease	Poster #S201 – S257WIP
Epilepsy	Poster #S301 – S324WIP
Movement Disorders	Poster #S401 – S463WIP
Multiple Sclerosis	Poster #S501 – S517WIP
Neurocritical Care	Poster #S601 – S617
Neuro-Oncology	Poster #S701 – S707
Regulatory Science	Poster #S801

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

MONDAY, SEPTEMBER 28

6:30 AM – 5:45 PM REGISTRATION

5th Floor Registration

7:00 – 9:00 AM CONTINENTAL BREAKFAST

Chicago Ballroom Foyer, 5th Floor

7:30 – 9:00 AM FACULTY DEVELOPMENT COURSES

EARLY AND MID/SENIOR CAREER LEVEL (COMBINED) FACULTY DEVELOPMENT COURSE II

Conflict Resolution/Negotiation

Marriott Ballroom, 4th Floor

Chairs: David Greer, M.D., M.A., F.C.C.M., F.A.H.A., F.N.C.S., F.A.A.N., *Yale University*
Roy Hamilton, M.D., M.S., *University of Pennsylvania*

Faculty: Marcia Martinez-Helfman, J.D., M.S.W., *University of Pennsylvania*

The ability to resolve professional disagreements and to negotiate effectively are essential skills for career advancement in academia. However, the value of these skills is generally under-recognized and formal training in these areas is often neglected in professional development. This session will specifically focus on the kinds of conflict and opportunities for negotiation that arise in academic neurology and introduce participants to important tools for engaging in effective dialogue in these challenging scenarios.

AUPN CHAIR DEVELOPMENT COURSE II

Optimizing the Use of Mid-Level Providers in an Academic Neurology Practice

Avenue Ballroom, 4th Floor

Chair: David Fink, M.D., *University of Michigan*

Faculty: Gretchen E. Tietjen, M.D., *University of Toledo*
David C. Good, M.D., *Pennsylvania State University*

One often touted response to increasing patient demand for neurology services is for departments to hire mid-level providers as "physician extenders". Dr. Gretchen Tietjen (University of Toledo) and Dr. David Good (Penn State) will present different models for the use of mid-levels, and discuss the pros and cons of physician extenders in an academic practice environment. The session will include ample time for open discussion and sharing of experiences from all participants.

9:00 – 9:15 AM COFFEE BREAK

Chicago Ballroom Foyer, 5th Floor

9:15 – 11:15 AM SYMPOSIUM: THE LIFE AND DEATH OF AXONS IN NEUROLOGICAL DISEASE

Chicago Ballroom A–E, 5th Floor

Co-Chairs: Ahmet Hoke, M.D., Ph.D., *Johns Hopkins University*
Charlotte Sumner, M.D., *Johns Hopkins University*

Axonal degeneration with loss of neuronal connectivity is a principal cellular event underlying clinical disability in many chronic neurodegenerative disorders as well as acute traumatic injuries of both the central nervous system (CNS) and peripheral nervous system (PNS). In this symposium, advances in our understanding of the molecular mechanisms of axonal maintenance and degeneration will be highlighted along with potential opportunities for novel therapeutics. The symposium co-chairs Dr. Charlotte Sumner and Dr. Ahmet Hoke both are clinical scientists who care for patients with peripheral nerve diseases and undertake research to understand mechanisms of axonal degeneration. The session participants are all widely recognized experts who have contributed fundamental insights regarding the function of axons, why they degenerate during neurological disease and how they might be protected using novel therapeutics.

Learning Objectives: Having completed this symposium, participants will be able to:

1. Explain how deficits of axonal transport cause axonal degeneration and mutations of specific axonal transport proteins cause several disorders of the PNS and CNS
2. Describe the molecular mechanisms that trigger axonal degeneration that are both intrinsic and extrinsic to neurons and could serve as novel therapeutic targets in neurological diseases
3. Evaluate the weaknesses in both preclinical and clinical trial design such that better trials could be conducted in the future

PROGRAM

9:20 – 9:45 AM
Presentation from the Bennett Awardee
Autophagy and Mitophagy in Neuronal Homeostasis and Neurodegeneration
Erika Holzbaur, Ph.D., *University of Pennsylvania*

9:50 – 10:15 AM
Molecular Mechanism of Axon Degeneration After Injury and Relevance to Disease
Michael Coleman, Ph.D., *Babraham Institute, United Kingdom*

10:20 – 10:45 AM
Myelinating Glia and the Metabolic Support of Axon Function
Klaus-Armin Nave, Ph.D., *Max Planck Institute for Experimental Medicine*

10:50 – 11:15 AM
Developing Therapies Aimed at Preventing Distal Axonal Degeneration
Ahmet Hoke, M.D., Ph.D., *Johns Hopkins University*

11:00 AM – 7:00 PM POSTER VIEWING
Grand Ballroom, 7th Floor

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

11:15 – 11:45 AM EXECUTIVE SESSION OF MEMBERSHIP*
Chicago Ballroom A–E, 5th Floor

All ANA members invited to attend for a year in review and an opportunity to discuss any potential bylaws changes.

11:30 AM – 1:00 PM LUNCH
Chicago Ballroom Foyer, 5th Floor

Boxed lunches are available to be taken into Interactive Lunch Workshops

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

CHALLENGES AND INNOVATION IN PREHOSPITAL STROKE CARE
Indiana/Iowa, 6th Floor

Moderator: Andrew Southerland, M.D., M.Sc., *University of Virginia Health System*

Faculty: Nerses Sanossian, M.D., F.A.H.A., *University of Southern California*
Sherita Chapman Smith, M.D., *Virginia Commonwealth University*
Christopher T. Richards, M.D., M.S., *Northwestern University*
Kameshwar Prasad, M.B.B.S., M.D., D.M., M.M.Sc., F.R.C.P., *All India Institute of Medical Sciences*

The emphasis on more rapid diagnosis and treatment of acute stroke is motivating research and innovation in the prehospital setting. Numerous national and international initiatives have identified the need for new approaches to prehospital stroke care, including the American Heart Association/American Stroke Association (AHA/ASA) *Target:Stroke* program and international SITS-WATCH study to improve acute stroke treatment times and outcomes (Fonarow et al. Stroke 2011, www.clinicaltrials.gov NCT01811901). Given the recent trial evidence in favor of endovascular stroke therapy there is an even greater need for timely prehospital diagnosis and triage. For patients living in rural and underserved areas, the infrastructure for prehospital stroke care is limited by a geographic disparity of proximity to primary stroke centers and access to neurological expertise. Innovative initiatives aimed at improving of the accuracy and timing of stroke diagnosis and treatment in the prehospital setting are underway, including improvements in prehospital stroke recognition, mobile telestroke and mobile stroke units, and prehospital stroke treatment trials.

CURRENT STATE OF STEM CELL THERAPIES
Los Angeles/Miami/Scottsdale, 5th Floor

Moderator: Sean Savitz, M.D., *University of Texas Health Science Center at Houston*

Faculty: Erin Furr Stimming, M.D., *University of Texas Health Science Center at Houston*
Anthony Windebank, M.D., *Mayo Clinic*
Sean Savitz, M.D., *University of Texas Health Science Center at Houston*

The purpose of this session is to demonstrate how Cell based therapies are being investigated as a new investigation treatment approach for various neurological disorders. This session will provide an update on current FDA approved clinical trials, testing cell therapies for different neurological disorders. There will be a specific focus on stroke, traumatic brain injury, ALS, and MS.

EMERGING CNS INFECTIONS
Denver/Houston/Kansas City, 5th Floor

Moderator: Avindra Nath, M.D., *National Institute of Neurological Disorders and Stroke, National Institutes of Health*
Co-Moderator: Thomas Bleck, M.D., *Rush Medical College*

Faculty: James Sejvar, M.D., *Center for Disease Control and Prevention*
Avindra Nath, M.D., *National Institute of Neurological Disorders and Stroke, National Institutes of Health*
Michael Wilson, M.D., *University of California, San Francisco*
Usha Kant Misra, M.B.B.S., M.D., M.N.A.M.S., D.M., N.A.S.I., *Sanjay Gandhi Post Graduate Institute of Medical Sciences*

The recent years have seen an emergence of enterovirus related acute myelitis, measles, dengue, chickenguniya and other new arboviruses in the United States. They pose unique challenges in diagnosis and treatment and underlying pathogenic mechanisms are not entirely clear. A discussion of these infections is of great importance to academic neurologists and those in practice.

ICU NEUROPATHY
Michigan/Michigan State, 6th Floor

Moderator: Paul Nyquist, M.D., *Johns Hopkins University*

Faculty: Christopher Klein, M.D., *Mayo Clinic*
Brian Crum, M.D., *Mayo Clinic*

We will present a case of ICU Neuropathy and review the EMG as well as clinical pathological characteristics of this disorder.

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

MEET THE NEUROLOGY DEPARTMENT CHAIRS*

Purdue/Wisconsin, 6th Floor

Faculty: Frances Jensen, M.D., *University of Pennsylvania*
Jeffrey Loeb, M.D., Ph.D., *University of Illinois*
Jose Biller, M.D., F.A.C.P., F.A.A.N., F.A.H.A., *Loyola University Health System*
David Holtzman, M.D., *Washington University in St. Louis*

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

TRANSLATIONAL RESEARCH FUNDING OPPORTUNITIES AT NINDS

Northwestern/Ohio State, 6th Floor

Moderator: Dane Chetkovitch, M.D., *Northwestern University*

Faculty: Amir Tamiz, Ph.D., *National Institute of Neurological Disorders and Stroke, National Institutes of Health*

This session will be about all the funding programs that have recently been launched at NINDS to support therapy and device development to a neurology/neuroscience focused audience.

11:45 AM – 1:00 PM SUMMARY OF THE ABPN MOC PROGRAM: LIFE-LONG LEARNING FOR NEUROLOGISTS AND PSYCHIATRISTS*

Lincolnshire I/II, 6th Floor

Faculty: Larry Faulkner, M.D., *President and CEO, American Board of Psychiatry and Neurology*

Dr. Faulker will lead the session off 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 four-part ABPN MOC program components

11:45 AM – 1:00 PM 15TH ANNUAL WOMEN OF THE ANA LUNCH PROGRAM: WORK LIFE BALANCE—IS IT ATTAINABLE?

Chicago Ballroom FGH, 5th Floor

Co-Chairs: Kathleen Digre, M.D., *University of Utah*
Cynthia Comella, M.D., F.A.N.A., *Rush University Medical Center*

Panelists: Cynthia Comella, M.D., F.A.N.A., *Rush University Medical Center*
Kathleen Shannon, M.D., *Rush University*
Deborah Hall, M.D., *Rush University*
Zoe Arvanitakis, M.D., F.A.N.A., *Rush University*

Over the last several years there has been increasing interest in women achieving some kind of balance in work and life. The practice of medicine seems harder to achieve balance and sometimes it seems to be either life or work. We will explore the questions about work/life balance by understanding what factors seem important for balance and our panel (made up of women academic neurologists at various stages of their careers) will contribute and discuss their insights. We will also ask all of the attendees to contribute ideas to the discussion and share the wisdom of years of experience.

1:15 – 3:15 PM SYMPOSIUM: HARNESSING PLASTICITY: TECHNOLOGIES FOR MOTOR AND COGNITIVE NEUROREHABILITATION AFTER STROKE AND NEUROTRAUMA

Chicago Ballroom A–E, 5th Floor

Chairs: Leigh R. Hochberg, M.D., Ph.D., *Massachusetts General Hospital, Harvard Medical School, Brown University*
Craig Powell, M.D., Ph.D., *University of Texas Southwestern Medical Center*

Neurotechnologies – devices that record from or stimulate the nervous system – are poised to become widely available therapies for the treatment of neurologic disorders causing paralysis or cognitive dysfunction. Increasingly specific brain regions can be targeted by technologies including transcranial direct current stimulation (tDCS), transcranial alternating current stimulation (tACS), transcranial magnetic stimulation (TMS), deep brain stimulation (DBS), intracortical microstimulation (ICMS), and optogenetic techniques in the laboratory. Stimulation of the peripheral nervous system (Functional Electrical Stimulation) can also be achieved for a variety of therapeutic purposes, including the reanimation of paralyzed limbs. In addition, new recording methods and computational approaches to understanding cortical function are allowing us to "decode" real-time neuronal ensemble activity, both in the laboratory and in clinical trials. In this exciting symposium, experts in neuromodulation, cortical plasticity and neuroprosthetics will discuss basic and clinical aspects of how we can both stimulate and record from the nervous system toward rehabilitating and restoring patients' neurological function.

Learning Objectives: Having completed this symposium, participants will be able to:
1. Identify neuromodulation strategies that may improve recovery of function after stroke
2. Determine if neuromodulation may improve function after acquired brain injury
3. Pinpoint how frequently Functional Electrical Stimulation has been used to improve mobility after spinal cord injury
4. Select computational approaches to studying cortical function that may lead to better control of external devices for people with paralysis

1:20 – 1:45 PM
Noninvasive Brain Stimulation for Stroke Recovery
Heidi Schambra, M.D., *Columbia University*

1:50 – 2:15 PM
Functional Electrical Stimulation for Reanimation of Paralyzed Limbs
P. Hunter Peckham, Ph.D., *Case Western University*

2:20 – 2:45 PM
Toward Clinically-viable Brain-machine Interfaces
Krishna Shenoy, Ph.D., *Stanford University*

2:50 – 3:15 PM
Modulating Plasticity to Repair the Injured Brain
Randolph Nudo, Ph.D., *University of Kansas*

PROGRAM

3:15 – 3:30 PM **COFFEE BREAK**
Chicago Ballroom Foyer, 5th Floor

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

AUTOIMMUNE NEUROLOGY
Denver/Houston/Kansas City, 5th Floor

Chair: Sean Pittock, M.D., *Mayo Clinic*
Co-Chair: Jeffrey Gelfand, M.D., M.A.S., *University of California, San Francisco*

This session will highlight advances in autoimmune neurology, an exciting and rapidly expanding field within neurology. This session will cover a range of autoimmune disorders affecting the central nervous system, peripheral nervous system and muscle, highlighting emerging concepts about pathogenesis and approaches to treatment.

LEADER IN THE FIELD PRESENTATION:
3:30 – 3:50 PM
Recent Advances in Autoimmune Myopathy
Andrew Mammen, M.D., Ph.D., *National Institutes of Health*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION:
4:00 – 4:08 PM
Autoantibodies in the CSF of Anti-GABAB Receptor Encephalitis Patients Block Activation of GABAB Receptors In Vitro
Ankit Jain, B.S.., *Perelman School of Medicine at the University of Pennsylvania*

4:08 – 4:10 PM
Q & A

LEADER IN THE FIELD PRESENTATION:
4:10 – 4:30 PM
Autoimmune Encephalitis: How Understanding Pathogenesis Will Improve Treatment
Josep Dalmau, M.D., Ph.D., *University of Pennsylvania, USA and University of Barcelona, IDIBAPS, and ICREA, Spain*

4:30 – 4:40 PM
Q & A

DATA BLITZ PRESENTATION:
4:40 – 4:48 PM
GAD Autoimmunity: Syndromes, Comorbidities, and Coexisting Antibodies in 121 Patients
Helena Ariño, *Hospital Clinic, University of Barcelona*

4:48 – 4:50 PM
Q & A

LEADER IN THE FIELD PRESENTATION:
4:50 – 5:10 PM
Designing Clinical Trials for Autoimmune CNS Disorders: The NMO Experience
Bruce Cree MD, Ph.D., *University of California, San Francisco*

5:10 – 5:20 PM
Q&A

DATA BLITZ PRESENTATION:
5:20 – 5:28 PM
Clinical Relevance of Voltage Gated Potassium Channel (VGKC)-Complex Antibodies in Children
Yael Hacohen, M.R.C.P.C.H., *University of Oxford*

5:28 – 5:30 PM
Q & A/Adjourn

CASE STUDIES
Cases in the Interface Between Neurology and Internal Medicine
Lincolnshire I/II, 6th Floor

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

3:30 – 5:30 PM
Case Presentations (Case Numbers 1-6)

DEMENTIA AND AGING
Northwestern/Ohio State, 6th Floor

Chair: Mike Greicius, M.D., M.P.H., *Stanford University*
Co-Chair: Dena Dubal, M.D., Ph.D., *University of California, San Francisco*

This symposium will provide updates on recent discoveries and future directions in aging and age-related dementias from leaders in the field. Dr. Ray Kelleher will discuss recent advances in understanding how presenilin mutations cause familial AD. Relevance of these mechanisms to AD phenotypes will be discussed. Dr. Erik Roberson will describe new insights into the role of tau in synaptic and network dysfunction in models of AD and FTD. Our final speaker, Dr. David Bennett, will discuss novel molecular systems approaches in probing cognitive resilience in aging and AD. In addition, three submitted abstracts complementing these themes will be chosen for brief presentations. The symposium will conclude with a question and answer session involving all the speakers.

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
3:30 – 3:50 PM
Loss of Presenilin Function in Familial Alzheimer's Disease Pathogenesis
Raymond Kelleher III, M.D., Ph.D., *Harvard Medical School*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION
4:00 – 4:10 PM
Single Cell qPCR Analysis of Frontal Cortex Retrotransposition in Alzheimer's Disease
Alex Bryant, M.D., *Laboratory of Dr. Fred Gage, Salk Institute*

LEADER IN THE FIELD PRESENTATION
4:10 – 4:30 PM
Targeting NMDA Receptor Dysfunction in Frontotemporal Dementia
Erik Roberson, M.D., Ph.D., *University of Alabama at Birmingham*

4:30 – 4:40 PM
Q&A

DATA BLITZ PRESENTATION
4:40 – 4:50 PM
CG4721 as a Novel Gene Controls Aging Through Insulin Signaling Pathway
Mohammad Jodeiri Farshbaf, M.D., *New Mexico State University*

LEADER IN THE FIELD PRESENTATION
4:50 – 5:10 PM
Resilience to Cognitive Aging: It's Not All About Neuropathology
David Bennett, M.D., *Rush University Medical Center*

5:10 – 5:20 PM
Q&A

DATA BLITZ PRESENTATION
5:20 – 5:30 PM
Human Neural Stem Cells Expressing IGF-1: A Novel Cellular Therapy for Alzheimer's Disease
Eva Feldman, M.D., Ph.D., *University of Michigan*

5:30 PM
Q & A / Adjourn

EDUCATION
Addison, 4th Floor

Chair: Steven L. Lewis, M.D., *Rush University Medical Center*
Co-Chair: Tracey A. Cho, M.D., *Massachusetts General Hospital, Harvard University*

Neurologic education is a global enterprise and effective neurologic education is needed for optimal prevention, diagnosis and management of neurologic disease worldwide. The 2015 Education Special Interest Group session will discuss the role of U.S. neurology residents in training local providers and sustaining neurology care in low income countries; the development of a certificate program in global and humanitarian neurology for neurology residents; the role of U.S. neurology residents and students as embedded medical educators internationally; ongoing regional educational initiatives for neurology trainees from low income countries; and the role of bidirectional partnerships in global neurology education and research training. Brief presentations by experts involved in these initiatives will be followed by ample time for a lively discussion.

LEADERS IN THE FIELD PRESENTATIONS:
3:30 – 3:45 PM
Introduction

3:45 – 4:00 PM
Role of U.S. Neurology Residents in Training Local Providers and Sustaining Neurology Care in Low Income Countries
Aaron Berkowitz, M.D., *Brigham and Women's Hospital, Harvard University*

4:00 – 4:15 PM
Development of a Certificate Program in Global Humanitarian Neurology for Neurology Residents
Tracey Cho, M.D., *Massachusetts General Hospital, Harvard University*

4:15 – 4:30 PM
U.S. Neurology Residents and Students as Embedded Medical Educators
Ralph Jozefowicz, M.D., *University of Rochester*

4:30 – 4:45 PM
Regional Educational Initiatives for Neurology Trainees from Low Income Countries
Steven Lewis, M.D., *Rush University Medical Center*

4:45 – 5:00 PM
Role of Bidirectional Partnerships in Global Neurology Education and Research Training
Ana-Claire Meyer, M.D., M.S.H.S., *Yale School of Medicine*

5:00 – 5:30 PM
Panel-Audience Discussion

PROGRAM

HEADACHE AND PAIN*

Clark, 4th Floor

Chair: Andrew H. Ahn, M.D., Ph.D., *Eli Lilly and Company*
Co-Chair: Peter Goadsby, *Kings College, London, United Kingdom*

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 session 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.

LEADER IN THE FIELD PRESENTATION

3:30 – 3:50 PM
Premonitory Symptoms and Triggers of Migraine - Overlapping Phenomenology
Peter J. Goadsby, M.D. Ph.D., *King's College, London, United Kingdom*

3:50 – 4:00 PM
Q&A

DATA BLITZ PRESENTATION

4:00 – 4:10 PM
Cranial Autonomic Symptoms in Migraine
Yong-Won Shin, M.D., *Seoul National University Hospital*

DATA BLITZ PRESENTATION

4:10 – 4:20 PM
Headache in the ED
Keith Dombrowski, M.D., *Duke University*

4:20 – 4:30 PM
Q&A

LEADER IN THE FIELD PRESENTATION

4:30 – 4:50 PM
Brain Signatures of Perceptual Quality
Andrew H. Ahn, M.D., Ph.D., *Eli Lilly and Company*

4:50 – 5:00 PM
Q&A

DATA BLITZ PRESENTATION

5:00 – 5:10 PM
DBS and Stimulation Parameters for SCI Pain
Corneliu Luca, M.D., Ph.D., *University of Miami*

5:10 – 5:20 PM
Q & A / Adjourn

HEALTH SERVICES RESEARCH

Michigan/Michigan State, 6th Floor

Chair: Kevin Kerber, M.D., *The University of Michigan Health System*
Co-Chair: Lesli Skolarus, M.D., M.S., *The University of Michigan Health System*

The topic of this session is research priorities in health services research. A panel of health services research experts will each separately present their vision for research priorities in health services research in neurology. Attendees will then have the opportunity to ask questions of this distinguished panel.

LEADERS IN THE FIELD PRESENTATIONS

3:30 – 3:50 PM
Research Priorities in Neurology-Related Health Services Research
Barbara Vickrey, M.D., M.P.H., Mount Sinai Health System

3:50 – 4:10 PM
Research Priorities in Neurology-Related Health Services Research
S. Claiborne Johnston, M.D., Ph.D., *The University of Texas*

4:10 – 4:30 PM
Research Priorities in Neurology-Related Health Services Research
Robert Holloway, M.D. M.P.H., *University of Rochester*

4:30 – 4:45 PM
Panel Discussion

DATA BLITZ PRESENTATIONS

4:45 – 4:50 PM
National Characteristics and Predictors of Neurologic 30-day Readmissions
Elan Guterman, M.D., *University of California, San Francisco*

4:50 – 4:55 PM
Q & A

4:55 – 5:05 PM
Increasing Stroke Knowledge in Korean Seniors: A Culturally-Tailored Education Intervention
Sarah Song, M.D., M.P.H., *Rush University Medical Center*

5:05 – 5:10 PM
Q & A

5:10 – 5:20 PM
Neuroimaging Overuse is More Common in Medicare Compared to the VA
James Burke, M.D., M.S., *University of Michigan*

5:20 – 5:30 PM
Q & A / Adjourn

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

INTERVENTIONAL NEUROLOGY

Indiana/Iowa, 6th Floor

Chair: Rishi Gupta, M.D., *Wellstar Kennestone Hospital*
Co-Chair: Muhammad Hussain, M.D., *Cleveland Clinic*

Many neurologists are unaware of the advances in acute stroke care and the future horizons regarding the impact on the systems of care. The intent for this session, which is targeted to practicing neurologists who care for patients suffering from ischemic stroke in the Emergency Room, is to bring forth the latest data in the field along with the anticipated future directions for neurologists to prepare their institutions and communities.

LEADERS IN THE FIELD PRESENTATIONS
3:30 – 3:50 PM
Mechanical Thrombectomy is the Standard of Care for Large Vessel Occlusions Causing Ischemic Stroke
Sam Zaidat, M.D., *Medical College of Wisconsin*

3:50 – 4:10 PM
Reducing Picture to Puncture Times Improves Clinical Outcomes With Mechanical Thrombectomy
Bijoy Menon, M.D., D.M., *University of Calgary*

4:10 – 4:30 PM
Reviving Neuroprotection in Stroke: The Rebirth of Hypothermia in the Setting of Mechanical Thrombectomy
Rishi Gupta, M.D., M.B.A., *Wellstar Kennestone Hospital*

4:30 – 4:50 PM
Stroke Systems of Care: Time for a Redesign?
Muhammad Hussain, M.D., *Cleveland Clinic Foundation*

4:50 – 5:10 PM
Expanding the Time Window for Mechanical Thrombectomy in Ischemic Stroke
Tudor Jovin, M.D., *University of Pittsburgh Medical Center*

5:10 – 5:30 PM
Panel Discussion

NEUROMUSCULAR DISEASE

Los Angeles/Miami/Scottsdale, 5th Floor

Chair: Catherine Lomen-Hoerth, M.D., Ph.D., *University of California, San Francisco*
Co-Chair: Eric Sorenson, M.D., *Mayo Clinic of Rochester*

This session will cover the latest in research, and therapeutics in key areas of neuromuscular diseases. We are planning "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 – 3:45 PM
Advances in ALS
Teepu Siddique, M.D., *Northwestern University*

DATA BLITZ PRESENTATION
3:45 – 3:55 PM
Reactivation of Lysosomal Ca2+ Efflux Rescues Abnormal Lysosomal Storage in CMT4J
Jun Li, M.D., *Vanderbilt University*

LEADER IN THE FIELD PRESENTATION
3:55 – 4:10 PM
Neuropathies and Autoantibodies
Alan Pestronk, M.D., *Washington University in St. Louis*

DATA BLITZ PRESENTATION
4:10 – 4:20 PM
High-Fat Diet-Induced Murine Metabolic Neuropathy
Lucy Hinder, Ph.D., *University of Michigan*

4:20 – 4:30 PM
Q&A

LEADER IN THE FIELD PRESENTATION
4:30 – 4:45 PM
Adult Onset Myopathies
Margherita Milone, M.D., Ph.D., *Mayo Clinic, Rochester*

DATA BLITZ PRESENTATION
4:45 – 4:55 PM
Results of a Double-Blind Placebo-Controlled Study of 3,4-Diaminopyridine (DAP) in Lambert-Eaton Myasthenic Syndrome (LEMS)
Vern Juel, M.D., *Duke University Medical Center*

LEADER IN THE FIELD PRESENTATION
4:55 – 5:10 PM
Advances in Congenital Myopathies
Duygu Selcen, M.D., *Mayo Clinic, Rochester*

DATA BLITZ PRESENTATION
5:10 – 5:20 PM
Biomarker Development for C9ORF72 Antisense Oligonucleotide Therapy Using iPS Neurons
Lindsey Hayes, M.D., Ph.D., *Johns Hopkins University*

5:20 – 5:30 PM
Q&A

SLEEP DISORDERS AND CIRCADIAN RHYTHM

Purdue/Wisconsin, 6th Floor

Chair: Phyllis Zee, M.D., Ph.D., *Northwestern University*
Co-Chair: Louis Ptacek, M.D., *University of California, San Francisco*

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 session is to promote broader understanding of sleep and circadian phenotypes and the implications these have on health of the nervous system.

PROGRAM

LEADER IN THE FIELD PRESENTATION
3:30 – 3:45 PM
Molecular Pathways Linking Circadian Clocks and Huntington's Disease
Ravi Allada, M.D., Ph.D., *Northwestern University*

DATA BLITZ PRESENTATION
3:45 – 3:55 PM
Slow Wave Sleep and Memory Enhancement in Older Adults Using Auditory Stimulation During Sleep
Penelope Papalambros, M.D., *Northwestern University*

LEADER IN THE FIELD PRESENTATION
3:55 – 4:10 PM
Role of Circadian Clocks in Neuroinflammation and Neurodegeneration
Erik Musiek, M.D., Ph.D., *Washington University in St. Louis*

DATA BLITZ PRESENTATION
4:10 – 4:20 PM
Prevelance and Incidence of Narcolepsy in a Longitudinal Study of Family Members of Narcoleptic Individuals
Maurice Ohayon, M.D., D.Sc., Ph.D., *Stanford University*

4:20 – 4:30 PM
Q&A

LEADER IN THE FIELD PRESENTATION
4:30 – 4:45 PM
Sleep-EEG Markers in Traumatic Brain Injury
Miranda Lim, M.D., Ph.D., *Oregon Health and Science University*

DATA BLITZ PRESENTATION
4:45 – 4:55 PM
Influence of Nighttime Lights on the Sleep of the General Population
Maurice Ohayon, M.D., DSc, Ph.D., *Stanford University*

LEADER IN THE FIELD PRESENTATION
4:55 – 5:10 PM
Genetics and Immunology of Narcolepsy and Kleine Levin Syndrome
Emmanuel Mignot, M.D., Ph.D., *Stanford University*

DATA BLITZ PRESENTATION
5:10 – 5:20 PM
Subclinical Cerebral Microvascular Ischemic Disease in Patients with Restless Legs Syndrome
Arthur Walters, M.D., *Vanderbilt University Medical Center*

5:20 – 5:30 PM
Q&A

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

Grand Ballroom, 7th Floor

POSTER CATEGORIES

Autoimmune Neurology	Poster #M101 – M133WIP
Dementia and Aging	Poster #M201 – M229WIP
Education	Poster #M301 – M307WIP
Headache and Pain	Poster #M401 – M410
Health Services Research	Poster #M501 – M510
Interventional Neurology	Poster #M601 – M609
Neuromuscular Disease	Poster #M701 – M761WIP
Sleep Disorders and Circadian Rhythm	Poster #M801 – M810WIP

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

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

Chicago Ballroom, 5th Floor

All attendees invited as we celebrate 140 years of neurologic academic excellence!

TUESDAY, SEPTEMBER 29

6:30 AM – 2:00 PM REGISTRATION

5th Floor Registration

7:00 – 9:00 AM CONTINENTAL BREAKFAST

Chicago Ballroom Foyer, 5th Floor

7:30 – 9:00 AM FACULTY DEVELOPMENT COURSES

EARLY CAREER LEVEL FACULTY DEVELOPMENT COURSE III

Grant Writing and Getting Funded
Clark, 4th Floor

Chairs: David Greer, M.D., M.A., F.C.C.M., F.A.N.A., F.N.C.S., *Yale University*
Joachim M. Baehring, M.D., D.Sc., *Yale University*

Faculty: Walter Koroshetz, M.D., *National Institute of Neurological Disorders and Stroke, National Institutes of Health*

Two senior speakers will provide an NIH and extramural perspective on funding opportunities and grant writing strategies. During an interactive session attendants will have the opportunity to discuss actual grant writing issues with the speakers.

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

MID/SENIOR CAREER LEVEL FACULTY DEVELOPMENT COURSE III

Becoming a Department Chair: Pitfalls, Challenges and Rewards
Avenue Ballroom, 4th Floor

Chairs: Amy Pruitt, M.D., *University of Pennsylvania*
Roy Hamilton, M.D., M.S., *University of Pennsylvania*

Faculty: Frances Jensen, M.D., *University of Pennslyvania*
David Standaert, M.D., Ph.D., *University of Alabama at Birmingham*
Dimitri Krainc, M.D., Ph.D., *Northwestern University Feinberg School of Medicine*

Academic neurology is in a constant state of change with decreasing NIH funding and further constraints on clinical revenue funds to support the academic mission. Becoming a department chair is not without its own pitfalls, challenges and rewards. Chairs are responsible for the growth and development not only of the academic missions and cross funding from the clinical enterprise, but also of the faculty within the department. This session will discuss the creative solutions applied to achieve success in the role of department chair.

AUPN CHAIR DEVELOPMENT COURSE III

Chairing a Department: If You Can't Laugh, You're Done
Addison, 4th Floor

Chair: David Fink, M.D., *University of Michigan*

Faculty: Nina Schor, M.D., Ph.D., *University of Rochester School of Medicine*

Being the Chair of a clinical department is an increasingly complex task. Chairs juggle oversight of and responsibility for clinical service; research; education and career development at many levels; local, regional and national politics; fundraising; ethical and legal matters; community relations and advocacy; and finances. This presentation will explore the tools and mechanisms that underlie successful leadership. Be sure to bring your questions and experiences for discussion!

9:00 – 9:15 AM COFFEE BREAK

Chicago Ballroom Foyer, 5th Floor

9:15 – 11:15 AM PRESIDENTIAL SYMPOSIUM: EVOLVING CONCEPTS IN PRION BIOLOGY: HOW MANY NEURODEGENERATIVE DISEASES ARE CAUSED BY PRIONS?

Chicago Ballroom A–E, 5th Floor

Chairs: Robert H. Brown, Jr., D.Phil., M.D., *University of Massachusetts Medical School*
Henry Paulson, M.D., Ph.D., *University of Michigan Health System*

Recent studies have documented that misfolding of proteins is a central component of many neurodegenerative disorders. Almost a dozen proteins including Abeta, alpha-synuclein, SOD1 and some RNA binding proteins exhibit self-assembly into aggregates that can propagate additional misfolding. These abnormal conformers not only multiply within neurons but also spread through neuronal networks. Thus, the behavior of these proteins closely resembles PrP prions that cause Creutzfeldt-Jakob disease (CJD). Starting with the history of the prion concept, this program will provide a critical review of the emerging theory that prions are responsible for a growing array of CNS degenerative disorders.

Learning Objectives: Having completed this symposium, participants will be able to:

1. Identify recent progress in understanding conventional prion diseases, including potential therapeutic strategies
2. List the principle clinical and pathological features of the major categories of neurodegenerative disorders, with an emphasis on those that are relevant to the prion hypothesis
3. Describe the breadth and diversity of genetic pathologies in these diseases
4. Distinguish between the pros and cons using data underlying the prion hypothesis in neurodegeneration

9:15 – 9:20 AM
Distinguished Neurology Teacher Award
Joseph Berger, M.D., *University of Pennsylvania*

9:20 – 9:45 AM
Prion Diseases: The Classical View
John Collinge, C.B.E., M.D., F.R.C.P., F.R.S., FMedSci., *UCL Institute of Neurology, National Hospital for Neurology and Neurosurgery*

9:50 – 10:15 AM
Prion-like Propagation of SOD1 Misfolding in Amyotrophic Lateral Sclerosis
Neil Cashman, M.D., *Brain Research Center, University of British Columbia*

10:20 – 10:45 AM
How Prion Biology is Informing our Understanding of Common Neurodegenerative Diseases
Marc Diamond, M.D., *University of Texas Southwestern Medical Center*

10:50 – 11:15 AM
RNA Granule Dynamics in ALS and Related Diseases
J. Paul Taylor, M.D., Ph.D., *St. Jude Children's Research Hospital, Memphis*

11:30 AM – 1:00 PM LUNCH

Chicago Ballroom Foyer, 5th Floor

Boxed lunches are available to be taken into Interactive Lunch Workshops

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

A CLINICIAN'S APPROACH TO CHRONIC TRAUMATIC ENCEPHALOPATHY

Denver/Houston/Kansas City, 5th Floor

Moderator: Nicole Reams, M.D., *University of Michigan*
Co-Moderator: Elisabeth Marsh, M.D., *Johns Hopkins University*

Faculty: Jeffrey Kutcher, M.D., *University of Michigan*

CTE remains a pathologic diagnosis at this time. While research advances our understanding on this entity, the clinician needs a framework for diagnosis and clinical management of these athletes. This workshop will serve to review our current knowledge of CTE, introduce the concept of Traumatic Encephalopathy Syndrome (TES) and how it differs from CTE, and propose a clinical approach to athletes that present with persistent neurocognitive complaints.

PROGRAM

EPILEPSY GENETICS: DISCOVERIES THROUGH TEAM SCIENCE
Los Angeles/Miami/Scottsdale, 5th Floor

Moderator: Alicia Goldman, M.D., Ph.D., M.S., *Baylor College of Medicine*
Co-Moderator: Sydney Cash, M.D., Ph.D., *Massachusetts General Hospital*

Faculty: Annapurna Poduri, M.D., M.P.H., *Harvard Medical School*
Edward Cooper, M.D., Ph.D., *Baylor College of Medicine*
Alicia Goldman, M.D., Ph.D., M.S., *Baylor College of Medicine*

In this interactive workshop, epilepsy is used as a model highlighting benefits and limitations of three conceptually different "team science" approaches in unraveling disease biology. The discussion will be centered on the power of discoveries through large patient cohorts, the clinical relevance of studying disease endophenotypes, and the value of detailed phenotypic ontology in population research and in individualized molecular medicine.

MEET THE EDITORS*
Michigan/Michigan State, 6th Floor

Faculty: Jack Kessler, M.D., *Annals of Clinical and Translational Neurology*
Scott Pomeroy, M.D., Ph.D., *Annals of Neurology*
Roger N. Rosenberg, M.D., *JAMA Neurology*
Heather Wood, *Nature Reviews Neurology*

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

SEX DISPARITIES IN NEUROLOGIC RESEARCH
Northwestern/Ohio State, 6th Floor

Moderator: Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins University*
Co-Moderator: Seemant Chaturvedi, M.D., F.A.A.N., F.A.H.A., F.A.N.A., *University of Miami*

Faculty: Louise McCullough, M.D., Ph.D., *University of Connecticut Health Center*
Seemant Chaturvedi, M.D., F.A.A.N., F.A.H.A., F.A.N.A., *University of Miami*

There has been increased concern about the lack of subject diversity in both preclinical and clinical studies. Specifically, female animals are frequently not used in basic science studies and women are underrepresented in many types of clinical research. Lack of representation of female animals in preclinical studies may lead to delayed recognition of pharmacologic adverse reactions that are more likely to occur in women. Inadequate representation of women in clinical trials can lead to uncertainties regarding treatment benefit or harms in women.

This session will address some of the recent recommendations from the NIH and FDA regarding greater representation of females in preclinical and clinical studies. In the clinical realm, there will be specific mention of stroke, epilepsy, and oncology studies.

USE OF RETINAL IMAGING IN THE DIAGNOSIS AND MANAGEMENT OF ALZHEIMER'S AND OTHER NEUROLOGICAL DISEASES
Purdue/Wisconsin, 6th Floor

Moderator: Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins University*
Co-Moderator: Ivan Bodis-Wollner, M.D., D.Sc., *State University of New York*

Faculty: Aikaterini Markopoulou, M.D., Ph.D., *Northshore University HealthSystem*
Pradeep Ramulu, M.D., Ph.D., M.H.S., *Johns Hopkins University*
Peter Calabresi, M.D., *Johns Hopkins University*

Retinal imaging techniques have been applied to neurodegenerative as well as demyelinating diseases. These techniques, including optical coherence tomography (OCT) allow the visualization and quantification of changes in specific retinal layers and neurons beyond nerve fiber layer defects. The results of many studies raise the hope to use these inexpensive, widely available and technically simple techniques in neurodegenerative and demyelinating diseases. OCT has the potential to be used as a diagnostic marker, therapeutic markers, and as a biomarker of risk of disease. The interactive workshop shall address use of this technique in research as well as clinical practice, with a focus on Alzheimer's Disease, Parkinson's Disease, and Multiple Sclerosis.

11:45 AM – 1:00 PM ANA LIAISONS LUNCH*
Clark, 4th Floor

This luncheon is for all ANA neurology department liaisons and will be led by ANA President, Dr. Robert H. Brown Jr.

11:45 AM – 1:00 PM AUPN'S NETWORKING LUNCH FOR SMALL ACADEMIC DEPARTMENTS OF NEUROLOGY*
Addison, 4th Floor

Moderator: Gretchen E. Tietjen, M.D., *University of Toledo*

While all neurology departments share some common attributes, there are challenges unique to smaller academic departments, including handling teaching and clinical service responsibilities, while protecting time for research and faculty development. This lunch, sponsored by the AUPN and hosted by Gretchen E. Tietjen, M.D., Chair of Neurology at the University of Toledo since 1998, provides an opportunity for chairs of smaller departments to meet, discuss issues and share strategies.

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

1:15 – 3:15 PM SYMPOSIUM: CONTROVERSIES IN STROKE THERAPEUTICS

Chicago Ballroom A–E, 5th Floor

Chair: Rebecca Gottesman, M.D., Ph.D., *Johns Hopkins University*
Moderator: Lee Schwamm, M.D., *Massachusetts General Hospital, Harvard University*

This session will address two controversies in stroke management in a debate format. Experts in the field will discuss two stroke management issues: use of endovascular therapy in acute stroke and antiplatelet therapy in secondary stroke prevention. Specifically, each debate will ask the question, "is two better than one?" For acute stroke, results from recent clinical trials will be reviewed and the remaining controversy of whether dual reperfusion therapy (IV tPA followed by endovascular therapy) is better than endovascular therapy alone for patients with vascular occlusions will be debated. For secondary stroke prevention, existing clinical trial data will be reviewed to debate the use of dual antiplatelet therapy versus antiplatelet monotherapy in secondary stroke prevention.

1:15 – 1:20 PM
DEBATE 1: The first debate will address dual versus monotherapy, for antiplatelet use in stroke prevention.
Lee Schwamm, M.D., *Massachusetts General Hospital, Harvard University*

1:20 – 1:35 PM
Is Two Better Than One For Secondary Stroke Prevention? Yes
Marc Chimowitz, M.D., *Medical University of South Carolina*

1:35 – 1:50 PM
Use of Dual vs Mono Antiplatelet Therapy for Stroke Prevention: Con
Graeme Hankey, M.B.B.S., M.D., *University of Western Australia*

1:50 – 2:15 PM
Audience Questions/Debate

2:15 – 2:20 PM
DEBATE 2: The second debate will cover endovascular therapy in acute stroke, with consideration of dual reperfusion strategies (IV tPA followed by endovascular therapy) versus endovascular monotherapy.
Lee Schwamm, M.D., *Massachusetts General Hospital, Harvard University*

2:20 – 2:35 PM
Presentation from the Soriano Awardee
Two is Better than One: Combined Intravenous Fibrinolysis and Endovascular Thrombectomy for Acute Ischemic Stroke
Jeffrey Saver, M.D., *University of California, Los Angeles*

2:35 – 2:50 PM
Time is Brain: Skip the tPA and Head Straight for the Cath Lab!
Colin Derdeyn, M.D., *Washington University School of Medicine*

2:50 – 3:15 PM
Audience Questions/Debate

3:15 PM MEETING ADJOURNMENT

SATURDAY SPEAKER ABSTRACTS

SYMPOSIUM: CIRCUITS AND CIRCUIT DISORDERS: APPROACHES TO NEUROMODULATION

Circuits, Circuit Disorders, and Neurologic Signs and Symptoms

Mahlon DeLong, M.D., *Emory University*

The introduction of high-frequency deep brain stimulation (DBS) over two decades ago, first for tremor and then for Parkinson's disease, has led to a renaissance in functional stereotaxic surgery for movement disorders, as well as a wide variety of other neurologic and psychiatric disorders. When combined with neuroimaging, neurophysiology and other investigative approaches, DBS has also proven to be a powerful and novel investigative tool for understanding network function and dysfunction. Exploration of the basal ganglia thalamocortical circuits in animal models and humans undergoing surgery for movement disorders has provided considerable insight into the nature of these disturbances. It is apparent that the signs and symptoms of these disorders result from signature underlying abnormalities in specific networks, which can be modulated by a variety of approaches, including ablation, electrical stimulation, and pharmacologic approaches. Furthermore, DBS is not disease-specific, but rather circuit-specific, since the same target may be used to treat a variety of movement disorders. I will focus largely on disorders resulting from disturbances within the "motor" circuit, including parkinsonism, chorea, and dystonia, discussing the significant progress made and questions remaining in order to better understand the mechanism of action of DBS and to realize its full potential.

Circuit Mechanisms of Dystonia: Insights from Combined Cortical and Basal Ganglia Recordings in Humans Undergoing Deep Brain Stimulator Implantation

Phillip A. Starr M.D., *University of California San Francisco*

Basal ganglia deep brain stimulation (DBS) can be effective for alleviating primary dystonia, but the circuit-level mechanisms that produce dystonia remain mysterious. Using the technique of combined cortical and basal ganglia recording in humans undergoing DBS lead implantation, we have elucidated electrophysiological characteristics of primary dystonia and compared them with Parkinson's disease patients.

We studied electrocorticography (ECoG) potentials over the arm area of the sensorimotor cortex using a temporary six-electrode subdural strip in patients who underwent DBS surgery simultaneous with STN or GPI local field potential (LFP) recording. Data were analyzed to extract signal power in multiple frequency bands, and phase amplitude coupling (PAC) between beta and broadband gamma, both at rest and during simple movement tasks, and during DBS.

We studied 22 primary dystonia and 15 age-matched, akinetic-rigid PD patients. All patients had resting state alpha-beta peaks in the ECoG power spectra for primary motor cortex (M1), and most had similar peaks in the basal ganglia, which did not differ in peak amplitude between groups. PD and primary generalized dystonia patients had higher resting state broadband power over M1, and a high level of beta-gamma phase-amplitude coupling, compared with craniocervical dystonia. Generalized dystonia with arm involvement showed impaired movement related beta desynchronization over M1.

Generalized dystonia and PD may have physiologic overlap with respect to motor cortex synchronization and resting state activity. Potential mechanisms of DBS, including decoupling of population spiking from the motor beta rhythm, will be discussed.

Deep Brain Stimulation for Tourette Syndrome and Related Disorders

Jonathan W. Mink, M.D., Ph.D., *University of Rochester*

Tourette Syndrome (TS) is a neurobiological disorder defined by the presence of sudden, stereotyped, repetitive movements and sounds called tics. It is a relatively common condition affecting up to 1% of children and continuing into adulthood on many affected individuals. Standard treatments provide moderate benefit overall and include behavior therapy and pharmacotherapy alone or in combination. A small percentage of individuals with TS have severe, medically-refractory tics that persist into adulthood and cause substantial disability. For those individuals, DBS may provide meaningful benefit. Recent studies have demonstrated that DBS in either intralaminar thalamic regions or globus pallidus pars interna can be beneficial. However, many questions remain. These questions arise from uncertainties about the underlying neurocircuitry of TS, the paroxysmal nature of tics, and the clinical course of the disorder, which tends to improve (rather than worsen) over time.

A closely related disorder, obsessive compulsive disorder (OCD), has also been the subject of recent investigation. The relevant neural circuitry for OCD likely overlaps with the relevant circuitry for TS, though there are important differences.

This presentation will focus on the results of recent studies of DBS in TS and OCD, current controversies, and emerging directions.

Therapeutic Modulation of Cingulate-Cortical Pathways in Major Depression

Helen S. Mayberg, M.D., *Emory University*

Deep Brain Stimulation is an emerging experimental treatment strategy for patients with intractable major depression. As testing of DBS in this context has expanded and matured, neuroimaging continues to play a crucial role, with recent work now focused on refinement and optimization of the procedure using multimodal methods combined with real-time behavioral and electrophysiological metrics. The evolving use of selective animal models and the strategic integration of neuroengineering innovations will further provide complementary perspectives necessary for the cohesive understanding of the pathophysiology of neuropsychiatric disorders and DBS mechanisms of actions with implications beyond depression.

Chemogenetics: A Robust Translational Platform for Cell-type Specific Neuromodulation

Bryan L. Roth, M.D., Ph.D., *University of North Carolina*

In this talk I will briefly summarize a chemogenetic platform invented by my lab known as Designer Receptors Exclusively Activated by Designer Drugs (DREADDs) which affords robust and reliable remote control of neuronal activity in a cell-type specific and non-invasive manner. I will then highlight applications of this technology as a therapeutic approach for a variety of neurological disorders. Finally, I will present new information showing how chemogenetic technologies facilitate the sequential multi-modal control of neurons in freely moving animals.

SUNDAY SPEAKER ABSTRACTS

SYMPOSIUM: CAUSES AND TRIGGERS OF MULTIPLE SCLEROSIS

Genetic Variation Leading to Risk of MS

David A. Hafler, M.D., *Yale School of Medicine and The Broad Institute of MIT*

Since the development of the clinical description of MS by Charcot over 160 years ago, there has been a gradual understanding of the underlying etiology of the disease, based first on microscopy revealing inflammatory cells accompanying losses of myelin. As new technologies have been applied, an emerging disease model has evolved driven by modern genetics, epigenetics, and observations in the EAE model. First, MS is inheritable, strongly clustering with other autoimmune diseases; genome-wide association studies have identified variation in predominantly immune genetic loci. Recent techniques have allowed a more precise identification of nucleotide changes and the mechanisms that cause heritable differences among individuals. MS is dictated by genetic variants that are predominantly in the open chromatid of T cells and B cells associated in particular with the NFkB, EBF1, and MEF2A transcription factor binding sites. Environmental stimuli interacting with these genetic factors appear to trigger the activation of myelin-reactive T cells. CD4 cell libraries generated from patients and control subjects from naïve, CCR6 neg memory, or $CCR6^{+}$ memory populations isolated *ex vivo* from blood and interrogated for reactivity to myelin self-antigens revealed that autoreactive T cells in patients with MS predominantly resided in CCR6 positive memory T cell compartment, secreting the pro-inflammatory cytokines IL-17, GM-CSF, and IFNϒ, but less IL-10 as compared to controls. Moreover, myelin-reactive T cells from MS patients exhibited a pathogenic gene expression signature similar to the encephalitogenic T cells in EAE. However, MS is not simply due to "bad genes", but instead is the bad outcome of too many variants in non-coding regions leading to a lower threshold of T cell activation in response to multiple environmental factors, including lower vitamin D, smoking, higher body mass index, exposure to EBV, and perhaps higher salt intake. In this regard, increased salt in the diet dramatically boosts the induction of Th17 cells mediated by SGK1. Th17 cells generated under high-salt display a pathogenic phenotype characterized by the up-regulation of the pro-inflammatory cytokines IL-17, GM-CSF, TNFα and IL-2. In summary, myelin-reactive T cells have a lower threshold of activation in MS that is genetically determined. Environmental stimuli interacting with these genetic factors appear to trigger the activation of myelin reactive T cells secreting inflammatory cytokines mediating the disease.

References:

1. Hafler DA, Compston A, Sawcer S, et al. Risk alleles for multiple sclerosis identified by a genomewide study. *N Engl J Med.* 2007;357(9):851.
2. Kleinewietfeld M, Manzel A, Titze J, et al. Sodium chloride drives autoimmune disease by the induction of pathogenic Th17 cells. *Nature.* 2013;496(7446):518.
3. Farh K, Marson A, Zhu J, et al. Genetic and epigenetic fine mapping of causal variants in autoimmune disease. *Nature.* 2015;518(7539):337.
4. Cao Y, Goods BA, Raddassi K,et al. Distinct inflammatory profiles of myelin-reactive T cells from patients with Multiple Sclerosis. *Science Translational Medicine.* 2015;7:287.
5. Housley, WJ, Grutzendler J, Cotsapas C, Hafler DA. Genetic variants associated with autoimmunity drive NFκB signaling and responses to inflammatory stimuli. *Science Translational Medicine.* 2015;7:291.

Environmental Factors Leading to the Risk of MS

Alberto Ascherio, M.D., Dr.P.H., *Harvard School of Public Health*

While genetic susceptibility explains the clustering of MS cases within families, the changes in MS risk that occur with migration can only be explained by changes in the environment. The strongest known risk factor is infection with the Epstein-Barr virus (EBV). MS is extremely rare in individuals who are not infected with EBV, but it has been shown in a longitudinal study that their MS risk increases sharply following EBV infection. As compared with uninfected individuals, the hazard of developing MS is at least 10-fold higher among individuals infected with EBV in childhood and over 20-fold higher among individuals infected in adolescence or later in life. Further, the antibody titer to the EBV nuclear antigen 1 is a strong marker of MS risk. Although the mechanisms underlying this association remain unclear, this data provides strong evidence of a causal relation between EBV infection and MS risk. Some aspects of the epidemiology of MS, however, are not explained by EBV, suggesting that either there are different EBV strains, with different propensity to cause MS, or other factors are also involved. Among the latter, one of the most prominent is vitamin D. An increased risk of MS in individuals with vitamin D insufficiency, originally proposed to explain the strong latitude gradient in MS prevalence, is now convincingly supported by several rigorous investigations. Three longitudinal studies have been reported. In the first, based on assessment of vitamin D intake from diet and supplements, risk of MS was found to be 40% lower among women who reported regular intake of vitamin D supplements. In the second study, conducted among young adults in the U.S. Army and Navy, vitamin D status was assessed by averaging multiple season-adjusted measures of 25(OH) vitamin D. During an average of five years of follow-up, MS risk among healthy young adults with high serum levels of 25(OH) vitamin D (> 100 nmol/L) was about 60% lower than in individuals of the same age and sex with low serum 25(OH)D levels. The third study confirmed prospectively an inverse association between serum 25(OH) D levels and MS risk among Swedish women. Combined, these results support the existence of a causal protective effect of vitamin D on MS risk. Further, there is strong evidence that vitamin D insufficiency is a risk factor for conversion from CIS to MS and for MS progression. Ideally, the benefit of vitamin D supplementation for MS prevention should be supported by large randomized trials, but the ethical and logistic obstacles for preventive trials are formidable. Other preventable risk factors for MS include cigarette smoking and childhood obesity.

Environmental Factors Interacting with Genes

Lisa F. Barcellos, Ph.D., M.P.H., *University of California at Berkeley*

Strong genetic and epidemiologic evidence supports the contribution of both genetic and environmental factors to multiple sclerosis (MS) susceptibility. Variation within major histocompatibility complex (MHC) region genes on chromosome 6p21 confers the greatest risk of MS, primarily within the human leukocyte antigen (HLA)-DRB1 locus (and specifically, the HLA-DRB1*15:01 allele); however additional MHC contributions have also recently been identified, underscoring the complexity of this gene region in MS. Through International MS Genetics Consortium efforts, genome-wide association (GWAS) and replication studies have identified >190 non-MHC MS susceptibility loci with modest effects (most odds ratios [ORs] <1.3); however, a considerable portion of the heritable component of MS remains unknown. Missing heritability (and potential sources of), has been the subject of much debate. Consideration of specific environmental factors important to development of MS may be necessary to further identify genetic contributions. Exposure to tobacco smoke, low serum levels of vitamin D, Epstein-Barr virus infection and childhood/adolescent obesity (high body mass index or BMI) have all been strongly implicated as MS risk factors. Standard approaches to identify evidence for interaction between genetic and environmental factors ('GxE') require large well-

characterized data sets; common study designs for GxE investigations have strengths and a few limitations. To date, GxE interactions influencing MS risk have been revealed, including specific HLA alleles with high BMI and tobacco smoke exposure (both active and passive). Evidence for interaction between arylamine N-acetyltransferase (NAT1) variation and tobacco smoke exposure has also been observed; specifically, a several-fold increased MS risk in smokers who are homozygous for NAT1 variants. NAT1 catalyzes transfer of an acetyl group from acetyl-CoA to various arylamine and hydrazine substrates, and is involved in metabolism of drugs and other xenobiotics, including constituents of tobacco smoke. Genetic variants can also be utilized as exposures in an observational study; specifically, 'Mendelian randomization', or instrumental variable (IV) analysis is a unique approach, for which measured variation in genes of known or putative biological function are used to estimate the causal relationship between exposure and disease risk. The genetic basis of both obesity and low vitamin D status, for example, has been demonstrated through recent GWAS. We have constructed IVs and have characterized through modeling, the relationships between obesity, low vitamin D serum levels, and MS susceptibility in large MS case-control studies. Further, we have used regression-based mediation analysis to examine direct and indirect effects of genes relating to obesity and low vitamin D serum levels on MS susceptibility. We find new evidence for a causal association between MS and these two important environmental exposures, even after accounting for other established risk factors.

References:

1. Patsopoulos NA, Barcellos LF, Hintzen RQ, et al. Fine-mapping the genetic association of the major histocompatibility complex in multiple sclerosis: HLA and nonHLA effects. *PLoS Genetics*. 2013;9:e1003926.
2. International Multiple Sclerosis Genetics Consortium, Wellcome Trust Case Control Consortium, Sawcer SJ, et al. Genetic risk and a primary role for cell-mediated immune mechanisms in multiple sclerosis. *Nature*. 2011;476:214-9.
3. International Multiple Sclerosis Genetics Consortium, Beecham AH, et al. Analysis of immune-related loci identifies 48 new susceptibility variants for multiple sclerosis. *Nature Genetics*. 2013;45:1353-60.
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No Quiet Surrender: Molecular Guardians in MS Brain

Larry Steinman, M.D., *Stanford University*

The brain under immunological attack does not surrender quietly. Investigation of brain lesions in multiple sclerosis (MS) reveals a coordinated molecular response involving various proteins and small molecules, ranging from heat shock proteins to small lipids, neurotransmitters, and even gases, which provide protection and foster repair. Reduction of inflammation serves as a necessary prerequisite for effective recovery and regeneration. Remarkably, many lesion-resident molecules activate pathways leading to both suppression of inflammation and promotion of repair mechanisms. These guardian molecules and their corresponding physiologic pathways could potentially be exploited to silence inflammation and repair the injured and degenerating brain and spinal cord in both relapsing-remitting and progressive forms of MS and may be beneficial in other neurologic and psychiatric conditions.

References:

1. Steinman L. No quiet surrender: molecular guardians in multiple sclerosis brain. *Journal of Clinical Investigation*. 2015;125:1371-1378.
2. Steinman L. Why are prions and amyloid structures immune suppressive and other intriguing questions facing neuroimmunologists in the future. *Rev Neurol (Paris)*. 2014;170: 602-607. doi: 10.1016/j.neurol.2014.07.011.
3. Steinman L. Immunology of Relapse and Remission in Multiple Sclerosis. *Annu Rev Immunol.* 2014;32:257-81. doi: 10.1146/annurevimmunol-032713-120227. Epub 2014 Jan 15.
4. Steinman L. The road not taken: antigen-specific therapy and neuroinflammatory disease. *JAMA Neurology*. 2013;1:1-2. doi: 10.1001/jamaneurol.2013.3553.

SYMPOSIUM: DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR SYMPOSIUM

Presentation of Derek Denny-Brown: Young Neurological Award in Basic Science

The Genetics of Epilepsy: A Complex Architecture

Annapurna Poduri, M.D., M.P.H. *Harvard Medical School*

Epilepsy affects 0.5-1% of the population, and about one-third of individuals with epilepsy have medically refractory epilepsy. Among this group are children with severe early onset epileptic encephalopathies and epilepsy caused by epileptic brain malformations. We know from studies of twins and families with epilepsy that epilepsy can be genetically mediated, but there are a number of different ways that genes can be involved in the causation of epilepsy. In this era of genomic medicine, we now have an expanding array of tools with which to study the complex and varied genetics of epilepsy; this has allowed us to shed light on a number of mechanisms of genetic epilepsy, including the continued role of ion channels but also a role for synaptic proteins, mitochondrial proteins, and signaling proteins, to name a few.

Mutations—either single nucleotide (base) changes in the DNA or copy number variations (deletions or duplications, missing or extra regions of DNA)—can arise spontaneously any time a cell divides. While some mutations are inherited, other mutations appear newly, or "*de novo*," in individuals with epilepsy. I will review some *de novo* causes of epilepsy recently identified through collaborative efforts across many institutions. One subgroup of genetic epilepsy involves epileptogenic brain malformations. A major challenge in the field of neurogenetics of epileptic brain malformations is that, unlike some genetic syndromes in which many organ systems are affected, many children with developmental brain malformations appear to have exclusive or preferential involvement of the brain. This is the case for focal cortical dysplasia, a common cause of refractory epilepsy, as well as most cases of hemimegalencephaly, a highly epileptogenic malformation involving one hemisphere of the brain. The apparently exclusive involvement of the brain in a given patient may reflect a mutation in a gene that is expressed only in the developing brain, or a mutation arising in an embryonic precursor to neuronal-glial progenitors causing a "brain-only" phenotype. Such a "somatic" mutation would not be inherited, but instead would have occurred early in the development of an individual, such that only a subset of cells in the individual would carry the mutation. However, if the mutation occurred later, then it might not be detectable in the individual's leukocytes, but could be detectable in the brain.

References:

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2. Poduri A, Evrony GD, Cai X, Walsh CA. Somatic mutation, genomic variation, and neurological disease (review). *Science.* 2013;341(6141):1237758. PMID: 23828942. PMCID: PMC3909954.
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Presentation of Derek Denny-Brown: Young Neurological Award in Basic Science
Tau, Hyperexcitability, and Alzheimer's Disease

Erik Roberson, M.D., Ph.D., *University of Alabama at Birmingham*

A major hurdle to treating Alzheimer's disease is the need for a better understanding of tau's role in the disease. We set out to investigate the role of tau in a commonly used AD mouse model, crossing with tau knockout mice if tau reduction had any effect. We identified an unexpected and critical requirement for tau in Aβ-induced dysfunction.[1] Mice lacking tau, and even those with partial tau reduction, were protected against Aβ, even though Aβ levels were not changed. The robust effect emphasized the potential benefits of targeting tau in AD, contributing to a surge of interest in tau.

Efforts to understand how tau reduction is protective in AD led to another surprising discovery, that tau plays a critical role regulating neuronal excitability. Tau reduction made AD mouse models resistant to seizures[1] and prevented the resulting compensatory hippocampal remodeling. Tau reduction also reduced interictal epileptiform spiking induced by Aβ.[2] By demonstrating the protective effects of reducing excitability in AD mouse models, these findings have emphasized AD-related hyperexcitability as a therapeutic target.

Our quest to understand the mechanisms underlying tau's role in AD-related hyperexcitability drew our attention to the neuronal dendrites, which are particularly affected in AD. Working with Dax Hoffman's lab to directly patch-clamp the dendrites in a mouse model of AD, we showed that Aβ-induced hyperexcitability begins with an increase in dendritic excitability,[3] apparently due to loss of Kv4.2, a potassium channel selectively expressed in the dendrites where it dampens dendritic excitability and plays a critical role in learning and memory. The loss of Kv4.2 in AD mice depends on tau.[3]

These studies demonstrated a novel function for tau that had not been appreciated in the decades since its discovery in the mid 1970s. As a microtubule-associated protein, prior work on tau function had focused mostly on the cytoskeleton and its effects on microtubule-based transport. Importantly, we found that tau reduction protected not only AD mouse models from seizures, but also nontransgenic mice, indicating a normal role for endogenous tau in regulating neuronal excitability and seizure susceptibility. Tau reduction reduces susceptibility to seizures induced by a variety of causes and reduces neuronal excitability in brain slices.[1,2] Thus, a critical function of tau in the adult brain is regulating neuronal excitability, suggesting that tau reduction–based therapeutic approaches could have even broader indications than AD and could also include forms of epilepsy.

My lab remains committed to translating the therapeutic potential of this discovery. We have focused on blocking what appears to be a key function of tau: its interaction with Fyn tyrosine kinase. Increasing Fyn levels is detrimental in AD models and we showed that tau reduction prevents Fyn-dependent deficits.[2] Therefore, we began a program to develop tau–Fyn interaction inhibitors. We screened 100,000 compounds for their ability to block the tau–Fyn interaction, identifying several potent hits with attractive chemical properties for further development[4] and are currently in the hit-to-lead phase of this drug discovery project.

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Presentation of Derek Denny-Brown: Young Neurological Award in Clinical Science
Advanced Imaging of Lesion Repair in Multiple Sclerosis

Daniel Reich, M.D., Ph.D., *National Institute of Health, National Institute of Neurological Disorders*

How, and how well, do acute white matter lesions heal in multiple sclerosis? Can tissue repair be characterized in vivo? And how might we test emerging treatments to promote such repair in early phase clinical trials? After more than a century of research into the pathology of MS, and 35 years since MRI was first applied in the disease, the answers to these questions still elude us. Yet emerging data from epidemiological studies seem to confirm our intuition, as neurologists, that tissue destruction within lesions may be highly relevant to the long-term accumulation of disability that occurs in progressive MS. At the same time, treatments that are most effective in reducing the chance of new lesion formation can also be dangerously immunosuppressive. Fortunately, there is now convincing experimental evidence that extensive endogenous repair, including remyelination, can occur soon after a lesion first appears, raising the possibility that therapeutic promotion of such repair might have both short-term and long-lasting benefits. In this talk, I will present data from our group's studies in the radiology and pathology of active MS[1,2] and primate experimental autoimmune encephalomyelitis[3] that together provide a framework for the spatiotemporal evolution of new white matter lesions. I will discuss how the blood-brain barrier is altered in distinct ways at different stages of lesion formation, and in particular how these alterations are reflected in magnetic susceptibility changes detectable using ultra-high-field (7 tesla) MRI. I will further show how such changes can be used to monitor and predict the extent of lesion repair, even over periods of several months. The ability to image these processes leads naturally to a set of efficient trial designs for short-term, proof-of-concept clinical trials to assess lesion repair,[4] potentially opening the way for the development of add-on agents that may limit the amount of tissue damage that occurs within new white matter lesions.

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Presentation from the Grass Awardee
Longevity Factor Klotho Enhances Cognition and Confers Resilience Against Neurodegenerative Pathologies

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

Aging is the primary risk factor for cognitive decline, an emerging threat to rapidly aging societies worldwide. "Anti-aging" approaches may represent new therapeutic pathways to counter the devastating cognitive erosion caused by age-related neurodegenerative diseases. Klotho is an aging regulator that, when overexpressed, extends lifespan in worms and mice. Elevated klotho in humans, in part caused by a genetic variant termed KL-VS, also promotes longevity. We investigated whether klotho counters brain aging and disease with genetic, cognitive, and experimental approaches in humans and mice. We found that in human populations, elevated klotho or the genetic variant that increases its levels, associated with (1) better cognitive function[1,2] and structural reserve[2], (2) resilience against age-related changes in biomarkers of neurodegenerative disease, and (3) decreased effects of chronic stress[3]. We next tested if and how klotho causes enhanced cognition and disease resilience. We examined whether global overexpression of klotho in aging and transgenic mice affects synaptic, network, and cognitive functions using behavioral, biochemical, pharmacologic, and electrophysiological approaches. We found that elevating klotho in mice enhanced normal cognition in young, middle-aged, and old mice in multiple tests of learning and memory[1]. It also enhanced synaptic plasticity and enriched the NMDA receptor subunit GluN2B in postsynaptic densities. Blockade of GluN2B decreased the GluN2B component of synaptic NMDA receptor activity and abolished klotho–mediated enhancement of learning and memory[1]. Furthermore, elevating klotho in mouse models of Alzheimer's disease (AD)[4] and Parkinson's disease (PD) counteracted deficits without altering levels of key pathogenic proteins such as Aβ, tau, or α-synuclein. Therapeutic strategies that increase the level or activity of longevity factor klotho may improve cognition and confer resilience against brain aging and neurodegenerative diseases, such as AD and PD.

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Presentation from the Wolfe Research Prize Awardee
Dietary Reversal of Neuropathy in a Murine Model of Prediabetes

Lucy Hinder, Ph.D., *University of Michigan*

Prediabetes patients with obesity, dyslipidemia, hypertension, and impaired glucose tolerance (IGT) develop the same macro- and microvascular complications as diabetic patients.[1] In type 2 diabetes, tight glycemic control hardly affects peripheral neuropathy development and progression; therefore, metabolic factors in addition to hyperglycemia likely contribute to neuropathy.[2] Moreover, dietary modification in prediabetic patients with neuropathy improves weight, IGT, cholesterol, nerve conduction velocities, and cutaneous innervation.[3] To experimentally investigate prediabetic peripheral nerve injury mechanisms, we examined longitudinal prediabetes and neuropathy development in six mouse strains/genotypes (BKS-*wt*, BKS-*db*/+, C57BL/6J(BL6)-*wt*, BL6-*db*/+, BTBR-*wt*, BTBR-*ob*/+) fed a 54% high-fat diet (HFD; from lard). All HFD-fed mice developed large fiber neuropathy and IGT. Changes appeared early and consistently in BL6-*wt* mice. Terminally, BL6-*wt* mice displayed large fiber dysfunction, distal small fiber degeneration, obesity, hyperinsulinemia, dyslipidemia, and oxidized low density lipoproteins (oxLDL). Dietary reversal, whereby BL6-*wt* mice fed HFD from 4-20 weeks were switched to standard chow for 4 weeks, completely normalized large fiber function, promoted weight loss, improved glucose tolerance, and restored LDL-cholesterol and oxLDL by 50% compared to HFD control mice. Thus, this dietary reversal model provides the basis for mechanistic studies investigating hyperglycemia-independent peripheral nerve damage to ultimately develop adjuncts to existing diabetic neuropathy therapies.

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MONDAY SPEAKER ABSTRACTS

SYMPOSIUM: THE LIFE AND DEATH OF AXONS IN NEUROLOGICAL DISEASE

Autophagy and Mitophagy in Neuronal Homeostasis and Neurodegeneration

Erika Holzbaur, Ph.D., *University of Pennsylvania*

Neurons are large, highly polarized, and post-mitotic cells that require active homeostatic mechanisms to maintain cellular health over the life of an organism. One mechanism critical to the maintenance of neuronal homeostasis is the degradative pathway known as autophagy, which clears both protein aggregates and aged or dysfunctional organelles. To investigate the role of autophagy in neurons, we examined the dynamics of the pathway using live cell microscopy of primary mammalian neurons. We identified a robust, constitutive process of autophagosome formation at the distal end of the axon, occurring via an ordered pathway with stereotypical kinetics. Concomitant with autophagosome formation, we observed the engulfment of cargos including aggregated proteins and mitochondrial fragments. Once formed, these autophagosomes are robustly transported to the cell soma; this transport is accompanied by a vectorial process of compartment maturation and acidification, leading to efficient cargo degradation. Consistent with a key role for active axonal transport, we find that inhibition of autophagosome motility leads to a block in both acidification and cargo degradation. Following up on the observation of mitochondrial engulfment, we identified the ubiquitin-binding protein optineurin as a receptor for damaged mitochondria. In HeLa cells, we found that optineurin is required downstream of PINK1 and Parkin, two Parkinson's disease genes previously implicated in mitophagy. Optineurin mediates the formation of LC3-positive autophagosomes around depolarized mitochondria, leading to the sequestration and degradation of these organelles. An ALS-linked mutation in optineurin that blocks ubiquitin binding inhibits the degradation of depolarized mitochondria in HeLa cells, suggesting that defects in mitophagy may contribute to the pathogenesis of neurodegenerative disease. In current work, we are investigating the role of optineurin in primary neurons; we note that optineurin is recruited to Parkin-positive mitochondria in the soma; in contrast, optineurin recruitment is rarely observed for mitochondria along neurites. These observations suggest that there is a compartmentalized pathway for removal of damaged mitochondria via optineurin and Parkin-dependent mitophagy; this selective pathway is primarily active in the soma, as compared to the robust formation of constitutive autophagosomes observed at the axon tip. Together, these results highlight the role of proteins in the autophagy pathway including PINK1, Parkin, and optineurin in maintaining a healthy neuron, and provide insights into the mechanisms by which mutations in these proteins may result in neurodegenerative diseases including Parkinson's and ALS. *Supported by NIH R37 NS060698.*

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Molecular Mechanism of Axon Degeneration After Injury and Relevance to Disease

Michael Coleman, Ph.D., *Babraham Institute, United Kingdom*

Axons and synapses are lost early in a wide range of neurodegenerative disorders, including peripheral neuropathy, Alzheimer's disease, multiple sclerosis, Huntington's disease, Parkinson's disease, amyotrophic lateral sclerosis, glaucoma, hereditary spastic paraplegia, and traumatic brain injury. Axonal transport is impaired in many of these conditions and declines substantially during normal ageing.

The Slow Wallerian degeneration protein (Wld^S) is an aberrant protein that preserves axons when they are injured or when axonal transport is impaired. It arose in mice but when ectopically expressed in transgenic rats, flies and zebrafish, or in transfected human neurons, it exerts the same protective effect.

Structure-function studies of Wld^S show that its intrinsic NAD synthesizing (Nmnat) activity is critical for axon survival. Of the three normal mammalian Nmnat isoforms, only Nmnat2 has been confirmed to be in axons. Nmnat2 is essential for axon survival and has a short half-life, so axons require its constant replenishment by axonal transport. When delivery of Nmnat2 is prevented by nerve injury, or by knockdown or knockout of Nmnat2, axons degenerate or fail to grow. Wld^S can rescue them by replacing the Nmnat enzyme activity and does so for a prolonged period as it has a much longer half-life.

Events both upstream and downstream of Nmnat2 are emerging in a wider pathway regulating axon survival. We find that Nmnat2 turnover is regulated by its palmitoylation-dependent targeting to axonal transport vesicles, followed by ubiquitination. Prevention of this vesicle targeting unexpectedly enhances the protective capacity of Nmnat2 to a level higher even than that of Wld^S, suggesting a cytosolic site of action. Downstream of Nmnat2, we identify a rise in the metabolic intermediate NMN as a key event. Inhibition of Nampt, the enzyme catalyzing NMN synthesis, preserves injured axons. Sarm1, a Toll-like receptor adapter required for Wallerian degeneration, appears to act in the same pathway at a downstream site. Sarm1 is necessary for axon degeneration after Nmnat2 knockdown but its deletion neither stabilizes Nmnat2 nor prevents the rise in NMN.

Thus, Nampt inhibition pharmacologically mimics the protective effect of Wld^S and Sarm1 represents another site for intervention on the same pathway. As further details of this pathway emerge, new targets should become evident for the prevention of axon loss in "dying back" disorders.

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Myelinating Glia and the Metabolic Support of Axon Function

Klaus-Armin Nave, Ph.D., *Max Planck Institute for Experimental Medicine*

Oligodendrocytes are only known for making myelin, which speeds up axonal impuls propagation and is essential for motor-sensory functions. We made the unexpected observation that oligodendrocytes are also required for the very survival of axons in white matter tracts. The two main functions of oligodendrocytes in (i) myelination and (ii) axonal support can be genetically uncoupled in mouse mutants. For example, MBP-deficient or cholesterol synthesis-deficient oligodendrocytes fail to myelinate, but CNS axons remain fully functional. In contrast, PLP- or CNP-deficient oligodendrocytes myelinate well, but then lead to signs of axonal transport defects, causing Wallerian degeneration and premature death. The underlying pathomechanisms have remained obscure, but it is not the absence of myelin which causes axons to die. We recently identified a novel aspect of neuron-glia interactions, the oligodendroglial support of axonal energy metabolism, which provides glucose-derived lactate as a fuel for axonal mitochondria. This supportive function of glycolytic oligodendrocytes is even more important for myelinated tracts because the insulating myelin sheath shields the axonal compartment from metabolites of the extracellular milieu. However, the generation of lactate must be carefully matched to the underlying axonal energy needs in order to prevent lactic acidosis. Spiking axons release glutamate, which opens the intriguing possibility that myelinating oligodendrocytes use NMDA receptors to detect glutamate release as a proxy for average spiking activity and axonal energy needs. Indeed, activation of oligodendroglial NMDA receptors causes a rapid redistribution of glucose transporters to the oligodendroglial cell surface, leading to enhanced glucose import and lactate release. In mice, the conditional inactivation of oligodendroglial NMDA receptors expression does not interfere with myelination, but reduces functional GLUT1 expression in oligodendrocytes and therefore perturbs axon function under metabolic stress. We suggest a model in which activity-dependent glutamate signalling within myelinated tracts enhances and maintains the glucose metabolism of oligodendrocytes to match the energy demands of fast spiking axons. Loss of metabolic support by injured oligodendrocytes is likely a contributing factor for axonal degeneration in various demyelinating diseases.

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Developing Therapies Aimed at Preventing Distal Axonal Degeneration

Ahmet Höke, M.D., Ph.D., *Johns Hopkins University*

There have been many failed drug development efforts aimed at modifying the disease course in peripheral neuropathies. Although there are multiple causes of such failures, some unique to the specific diseases, several common themes emerge from a careful review of the literature. A unique feature of many peripheral neuropathies is relative lack of neuronal death but presence of distal axonal degeneration and recognition that the molecular mechanisms that underlie this distal axonal degeneration may be different than the mechanisms that lead to neuronal death in the cellular and animal models used in preclinical studies. One such example is the changes that occur in mitochondria and mitochondrial DNA in long axons that make them more vulnerable to oxidative damage and less able handle to energy needs of their microenvironment compared to mitochondria within the cell body or proximal axons. Such mitochondrial changes may underlie "length-dependency" of axonal peripheral neuropathies.

For many years, the primary strategy for the early stages of drug development have relied on pharmacological screening based on molecular targets, but a different strategy, phenotypic drug screening, is gaining momentum in the field and is perfectly suited for drug development for prevention of distal axon degeneration that may share common molecular mechanisms across different diseases. Although the exact initiating events that lead to Wallerian degeneration and distal axonal degeneration seen in many peripheral neuropathies are likely to be different, they are likely to share common key players and observations from the *Wlds* mouse and recent forward genetic screens in flies that yielded *Sarm* gene teach us that manipulation of these pathways can, at least partially, abrogate some forms of peripheral neuropathy. Furthermore, drug-screening strategies based on phenotypic screening, coupled with validation using animal models that utilize outcome measures that are more relevant to the human disease, are more likely to result in successful clinical candidates.

Nevertheless, these newer strategies have not been put to test yet as none of them have advanced to Phase III studies. It is important for the field to choose the clinical target appropriately and match the drug and it's mechanism of action to the pathogenesis of distal axonal degeneration seen in peripheral neuropathy or the stage of neuropathy. For example, it does not make any sense to try a drug in chronic diabetic peripheral neuropathy (despite its huge potential market) if its primary mechanism of action is neuroprotection or axonoprotection, at least as a first clinical target. It may be more appropriate to use chemotherapy induced peripheral neuropathy (CIPN) as a clinical target with its huge unmet clinical need and perfect scenario to test neuroprotective strategies. CIPN offers the field an opportunity to test many compounds relatively quickly as the design of disease modifying clinical trials is much easier and cheaper than "traditional" peripheral neuropathy targets such as diabetic or HIV-associated peripheral neuropathies.

SYMPOSIUM: HARNESSING PLASTICITY: TECHNOLOGIES FOR MOTOR AND COGNITIVE NEUROREHABILITATION

Noninvasive Brain Stimulation for Stroke Recovery

Heidi Schambra, M.D., *Columbia University*

As stroke prevalence increases in the next 30 years, the number of patients with stroke-related disability is expected to soar. The major push in neurorehabilitation is to identify neuromodulatory approaches that maximize recovery. In the weeks following stroke, it is believed that a critical period of neuroplasticity exists that can be engaged by targeted training therapies (Corbett, Jeffers et al. 2015). However, given ever-diminishing lengths of stay in acute rehabilitation, there is also substantial interest in potentiating therapies with adjuvants — interventions that alone do not induce plasticity, but amplify activity-dependent plasticity driven by training.

One promising adjunctive approach is noninvasive brain stimulation. Two of the most common methods are repetitive transcranial magnetic stimulation (rTMS) and transcranial electrical stimulation (tES) (Liew, Santarnecchi et al. 2014). rTMS involves the application of repeated magnetic pulses, whereas tES involves the application of an electrical current. Both are applied painlessly to the head for a period of time, producing prolonged changes in cortical excitability. Although both methods modulate cortical excitability, they are likely to have different mechanism of actions. Importantly, both must be given in conjunction with physical or cognitive training to yield behavioral improvement.

The application of rTMS is largely based on the interhemispheric competition model, which posits that the nonlesioned hemisphere excessively inhibits the lesioned hemisphere following stroke (Di Pino, Pellegrino et al. 2014). To restore interhemispheric inhibitory balance, rTMS is used to diminish excitability in the nonlesioned hemisphere or to increase excitability in the lesioned hemisphere. Once this balance has been briefly "reset," the system can be engaged with training for behavioral consolidation. We will discuss work using inhibitory rTMS for the amelioration of neglect and paresis.

The most common tES approach is transcranial direct current stimulation (tDCS), which uses a low-intensity constant current passed from an anodal to a cathodal electrode. Although cortical excitability is differentially modulated based on electrode polarity, it is now generally assumed that the electrical current diffusely washes over and through the brain. The action of tDCS may arise through "functional targeting"— the modulation of only networks that are simultaneously active during stimulation (Bikson and Rahman 2013). We will discuss work using tDCS in humans with and without motor stroke.

Each method has its advantages and disadvantages. While rTMS provides greater stimulation focality and is given before therapy, the device is costly, usually immobile, and requires an experienced operator. tDCS is inexpensive, portable, and easy to administer, but must be given during therapy and lacks the stimulation focality desired for some applications.

To date, the clinical effects seen with noninvasive brain stimulation are modest but promising. The field of noninvasive brain stimulation is still relatively young, and work is currently underway to identify stimulation parameters that optimize behavioral gains. With their excellent safety profiles and tolerability, rTMS and tDCS may well become part of routine clinical treatment in recovering stroke patients.

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Functional Electrical Stimulation for Reanimation of Paralyzed Limbs

P. Hunter Peckham, Ph.D., *Case Western University*

Toward Clinically-viable Brain-machine Interfaces

Krishna Shenoy, Ph.D., *Stanford University*

Millions of people suffer from motor disabilities and injuries, resulting in the loss of arm movements and independence. A relatively new medical technology termed brain-computer interfaces (BMIs) aims to help restore this lost function by converting movement intentions from neurons in the brain into control signals for guiding prosthetic arms and computer cursors. BMI research spans engineering, neuroscience, and translational medicine. In this talk I will discuss some recent advances in all of three domains, and describe how this is leading to higher-performance BMIs in tetraplegic participants diagnosed with Lou Gehrig's disease (ALS) enrolled in our FDA pilot clinical trial. Future BMIs, which are a core technology for "reading information from" and "writing information to" the brain, will likely be applied to a wide range of neurological and psychiatric impairments and help ever increasing numbers of people.

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Modulating Plasticity to Repair the Injured Brain

Randolph Nudo, Ph.D., *University of Kansas*

Neuroprosthetic devices generally can be categorized as open-loop neuromodulation systems, that directly or indirectly excite neural tissue, or brain-computer interfaces, that derive control signals from the brain to operate external devices. Increasingly, neuroscientists, computer scientists and engineers are beginning to envision and develop closed-loop systems that stimulate neuronal populations contingent upon a particular neuronal signal derived from another population of neurons. Both modalities are now common in laboratory settings and may soon move into clinical practice to treat neurological conditions that include epilepsy, Parkinson's disease, stroke, traumatic brain injury and spinal cord injury. Thus, it is now critical to understand how such systems interact with the neural circuitry and how neural communication may be altered in an adaptive way.

Whether open or closed-loop, neuromodulation systems are thought to induce plasticity either by altering general excitability or by facilitating or inhibiting specific pathways. While neuroplasticity is often inferred when the modulatory treatment results in altered function, direct assessment of neural changes, especially in animal models, provides insight into underlying neural mechanisms of action, and may guide

optimization of treatment protocols. Such biomarkers of neuroplasticity can be examined at many levels of analysis, including altered gene regulation, expression of neurotrophic, neurogenic, and angiogenic factors, synaptogenesis, dendritic sprouting and axonal arborization. Neurophysiological changes have also been documented, ranging from altered neuronal spike discharge and synaptic facilitation, to altered functional motor and sensory representations. Many plasticity phenomena observed at the molecular, cellular and network levels of analysis in animal models have direct parallels in human studies using transcranial magnetic stimulation -based mapping, functional magnetic resonance imaging, and diffusion tensor imaging. The conundrum of designing optimal approaches to neural repair and recovery will require a comprehensive integration of the complementary bits of information derived from these approaches.

Our current research program focuses on the potential ability for closed-loop systems to regulate synaptic potentiation in long-distance pathways in the nervous system, particularly cortico-cortical pathways between different functional areas. Because the demonstration of long-term potentiation and long-term depression in animal preparations has utilized stimulation timing protocols that are not typically feasible using non-invasive techniques, our current pre-clinical model employs recording microelectrodes implanted within the cerebral cortex, and microdevices that discriminate individual action potentials, process discriminated spikes from multiple input channels, and then electrically stimulate remote brain regions using implanted microelectrodes. Recent studies have demonstrated the ability of this closed-loop system to modulate synaptic potentiation between the two areas and promote functional recovery after brain injury. Despite the challenges of invasive procedures using implantable technology, such closed-loop systems have the potential to provide new treatment avenues, including pathway-specific targeting, for treating neurological conditions.

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TUESDAY SPEAKER ABSTRACTS

SYMPOSIUM: PRESIDENTIAL SYMPOSIUM: 1. EVOLVING CONCEPTS IN PRION BIOLOGY: HOW MANY NEURODEGENERATIVE DISEASES ARE CAUSED BY PRIONS?

Prion Diseases: The Classical View

John Collinge, C.B.E., M.D., F.R.C.P., F.R.S., F.R.C.P., FMedSci, *UCL Institute of Neurology, National Hospital for Neurology and Neurosurgery*

Prion-like Propagation of SOD1 Misfolding in Amyotrophic Lateral Sclerosis

Neil Cashman, M.D., *University of British Columbia*

Approximately 10% of ALS cases are familial, with ~20% of these due to mutations in the gene encoding Cu/Zn superoxide dismutase (SOD1), a ubiquitous free-radical defense enzyme. A consequence of SOD1 mutation and/or oxidation is a propensity of the protein to misfold and aggregate. We sought to molecularly dissect the effects of intracellular obligate misfolded SOD1 mutant proteins on natively structured wild-type SOD1 (1). Expression of the enzymatically inactive, natural familial ALS SOD1 mutations G127X and G85R in human cell lines induced misfolding of wild-type natively-structured SOD1, as indicated by: 1) acquisition of immunoreactivity with SOD1 misfolding-specific monoclonal antibodies; 2) markedly enhanced protease sensitivity suggestive of structural loosening; and 3) non native disulfide-linked oligomer and multimer formation. Expression of G127X and G85R in mouse cell lines did not induce misfolding of murine wild-type SOD1, and a species restriction element for human wild-type SOD1 conversion was mapped to a region of sequence divergence in loop II and beta-strand 3 of the SOD1 beta-barrel (residues 24-36), then further refined surprisingly to a single tryptophan residue at codon 32 in human SOD1 (1). We now find that small molecules interacting with Trp32 can inhibit SOD1 propagated misfolding. Cytosolic mislocalization of FUS and TDP43, two proteins implicated in familial and sporadic ALS, was associated with human wild-type SOD1 misfolding (2). Culture medium from cells transiently transfected with wild-type or mutant SOD1 (3), or TDP43/FUS mutations, induced misfolding of endogenous SOD1 when incubated with naive cell cultures, and this process was stably propagated in serial passage. Nonspecific uptake of misfolded SOD1 from the supernatant was excluded by siRNA knockdown of SOD1 in the fresh recipient cells, indicating a requirement for endogenously expressed SOD1 as a substrate. The agent responsible for induction of misfolding was determined to be a relatively massive particle pelleted by ultracentrifugation, consistent with transmission by exosomes or protein aggregates (3). Transmission of SOD1 misfolding in vitro was abrogated by extracellular pan- and misfolding-specific SOD1 antibodies (3). On quantitative immunoprecipitation, misfolded SOD1 was found to constitute ~4% of total SOD1 in spinal cord from SOD1- and C9ORF72-FALS, as well as sporadic ALS (3). G37R Tg mice treated with misfolding-specific SOD1 antibodies displayed prolonged survival of ~11 days ($p < 0.001$). In conclusion, SOD1 misfolding can propagate within and between cells, and can be triggered by pathologies induced by other ALS-implicated genes. ALS joins company with Alzheimer's, Parkinson's, and other neurodegenerative diseases as a "prion-like" disorder, opening new therapeutic avenues for this dreadful disorder (4).

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How Prion Biology is Informing our Understanding of Common Neurodegenerative Diseases

Marc Diamond, M.D., *University of Texas Southwestern Medical Center*

Evidence suggests that neurodegenerative diseases linked to protein amyloids could be caused by trans-cellular propagation of protein aggregates.[1] This is the mechanism by which prions propagate pathology. According to this model, protein assemblies escape one cell, enter a vulnerable cell, and create new pathology by acting as a template, or "seed," for further aggregation. We have tested whether seed prevalence and conformation can predict human disease phenotypes. We developed a FRET-based biosensor cell assay to quantify seeding activity in biological samples.[2] We have used this to measure seeding activity, and test the role of proteopathic seeds in the development of pathology. We have identified tau prion seeds in human and mouse brain far in advance of standard histopathology, and in disorders as diverse as Alzheimer's disease and chronic traumatic encephalopathy. Prion "strains" replicate faithfully over decades in animal hosts, and underlie the tremendous clinical and neuropathological diversity of prionopathies. These strains are comprised of distinct amyloid structures. Like prionopathies, tauopathies are clinically and neuropathologically heterogeneous, and feature intraneuronal accumulation of tau amyloid assemblies that are rich in beta sheet structure. We have described essential strain characteristics of tau prions, in which unique conformations propagate indefinitely in cells and in animals, and create unique patterns of neuropathology, and have linked human tauopathies to distinct groups of tau prion strains.[3] We find that after intracerebral inoculation, strain conformation defines the structural and biochemical properties of the induced aggregates, neuronal pathology, and patterns of spread. Our results suggest a very proximal role for proteopathic seeds in the pathogenesis of tauopathies, with unique strains determining patterns of pathology and clinical phenotype. We hypothesize this could ultimately enable syndromic classification of tauopathies based entirely on aggregate structure, and could have important therapeutic implications.

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RNA Granule Dynamics in ALS and Related Diseases

J. Paul Taylor, M.D., Ph.D., *St. Jude Children's Research Hospital*

Stress granules are membrane-less organelles composed of RNA-binding proteins (RBPs) and RNA. Functional impairment of stress granules has been implicated in amyotrophic lateral sclerosis, frontotemporal dementia and multisystem proteinopathy - diseases that are characterized by cytosolic fibrillar inclusions of RBPs that comprise stress granules (e.g. TDP-43, FUS, and hnRNPA1). The molecular basis for stress granule assembly is poorly understood and the role of stress granules in disease is unknown. We discovered that the certain key RBPs undergo reversible liquid-liquid phase separation (LLPS) into protein-rich droplets mediated by a prion-like low complexity sequence domain (LCD). Notably, these prion-like LCDs are a frequent site of disease-causing mutations. Importantly, while not required for phase separation, we found that fibrillization of LCD-containing RBPs occurs within the condensed liquid phase of RBP droplets. Thus, LLPS mediated by the prion-like LCD is the biophysical basis for the assembly of stress granules and accounts for their liquid properties. Moreover, these insights reveal the mechanistic link between persistent stress granules and fibrillar protein pathology in disease.

SYMPOSIUM: CONTROVERSIES IN STROKE THERAPEUTICS

Is Two Better Than One For Secondary Stroke Prevention? Yes

Marc Chimowitz, M.D., *Medical University of South Carolina*

Studies have shown that dual antiplatelet therapy compared with single antiplatelet therapy does not lower the risk of recurrent ischemic stroke but does increase the risk of major hemorrhage in patients with heterogeneous causes of ischemic stroke[1] and in patients with lacunar stroke.[2] In these studies, patients were enrolled up to three[1] or six[2] months after stroke onset. A more recent trial has shown that dual antiplatelet therapy is more effective than antiplatelet mono-therapy for lowering the risk of recurrent stroke at 90 days if patients are started on dual antiplatelet therapy within 24 hours of stroke onset.[3] Additionally, studies have suggested that dual antiplatelet therapy for a limited period of time (90 days) may have particular efficacy in preventing recurrent stroke in patients with atherosclerotic intracranial large artery stenosis who have had a TIA or stroke within the previous 24 hours - 30 days[3,4] As such, there are accumulating data to support the use of dual antiplatelet therapy for stroke prevention depending on the underlying cause of stroke and when the patient was last symptomatic.

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Use of Dual vs Mono Antiplatelet Therapy for Stroke Prevention: Con

Graeme Hankey, M.B.B.S., M.D., *University of Western Australia*

Primary Prevention of First-ever Stroke

Long-term single antiplatelet therapy with aspirin has not proven effective in the primary prevention of first-ever stroke in 6 randomized controlled trials (RR 0.95, 0.85-1.06).[1] Long-term dual antiplatelet therapy with clopidogrel plus aspirin has not proven more effective than aspirin monotherapy in one RCT involving patients with vascular risk factors; dual antiplatelet therapy was associated with an increase in all-cause mortality compared with aspirin monotherapy (5.4% vs. 3.8%, P=0.04).[2]

Secondary Prevention of Recurrent Stroke

Acute single antiplatelet therapy with aspirin is effective in reducing the risk of any early recurrent stroke by 12% (OR 0.88, 0.80-0.97) compared with no antiplatelet therapy. Acute dual antiplatelet therapy is more effective than monotherapy in reducing the risk of any early recurrent stroke when given to high risk patients with TIA or mild ischemic stroke (RR 0.69, 0.60 to 0.80).[3] Most of the benefit is realized within the first few days of treatment. The effect of acute dual antiplatelet therapy in patients at low risk of recurrent stroke, and with disabling ischemic stroke, is uncertain. Long-term single antiplatelet therapy with aspirin is effective in reducing the risk of any early recurrent stroke by 17% (RR 0.83, 0.72-0.96) compared with no antiplatelet therapy, and clopidogrel monotherapy is marginally more effective than aspirin monotherapy in reducing major vascular events (RR 0.91, 0.84 to 0.99).

Long-term (>1 year) dual antiplatelet therapy is not more effective than monotherapy with aspirin (RR 0.89, 0.78-1.01; I^2=57%) or clopidogrel (RR 1.01, 0.93 to 1.08; I^2=0%) in preventing recurrent stroke, and has a higher rate of intracranial haemorrhage than clopidogrel monotherapy (RR 1.46, 1.17 to 1.82; I^2=0%).[4] Single antiplatelet therapy is as effective as dual antiplatelet therapy for preventing first-ever and recurrent stroke, except when used in the first few days to weeks after ischaemic stroke or TIA due to atherothromboembolism in patients at high risk of early recurrent stroke and low risk of haemorrhagic transformation of the brain infarct (i.e., TIA or mild ischemic stroke); for these patients dual antiplatelet therapy appears more effective.

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Presentation from the Soriano Awardee

Two is Better than One: Combined Intravenous Fibrinolysis and Endovascular Thrombectomy for Acute Ischemic Stroke

Jeffrey Saver, M.D., *University of California, Los Angeles*

Combined intravenous fibrinolysis (IVF) and endovascular thrombectomy (ET) may confidently be predicted to be a superior therapy over endovascular thrombectomy alone for acute ischemic stroke (AIS).

For combined IVF+ET to be advantageous over ET alone, the added, initial intravenous fibrinolysis would need to confer moderate to substantial additional benefits and avoid causing moderate to substantial increased harm. IVF before ET clearly meets both of these criteria.

Clinical studies indicate that IVF before ET improves AIS patient outcomes,[1] and numerous mechanisms may be expected to mediate this benefit. There is a long interval between the time when IV tPA can be started and the first deployment of a thrombectomy device, due to consent elicitation for the procedure, patient transport to the neurocath lab, patient transfer onto the cath table, interventional team mobilization, shaving the groin, groin puncture, sheath placement, performance of a diagnostic angiogram, and navigation of the device to the clot. During this 45-200 minute interval,[2] IV tPA dissolves the clot before device deployment can begin, in 10-40% of patients. Indeed, in an important minority of patients, the thrombectomy device will turn out to be unable to reach the target occlusion, due to hostile vessel anatomy or patient cardiorespiratory instability. Since inability to navigate to the target occlusion will not be discovered until after the IV tPA window has closed, rescue IVF is not an option. Unless these patients receive IV tPA before planned ET, they will miss receiving any reperfusion treatment whatsoever. In addition, even when it does not achieve complete clot digestion, IV tPA can chemically degrade the clot, increasing subsequent ET mechanical reperfusion rates and reducing number of passes and time needed for ET reperfusion.[1,3] Finally, ET often causes embolization of fragments of the target thrombus to distal medium and small arteries unreachable by a mechanical device. Already on-board IV tPA is very effective in "cleaning up" these residual smaller thrombi.

With regard to safety of combined therapy, multiple randomized trials and large cohort studies have demonstrated that IV tPA does not increase the rate or severity of symptomatic intracranial hemorrhage ET.[4]

Of course, randomized trials need to be performed to confirm our field's strong expectation that combined IVF+ET is superior to ET alone. But, when they are completed, assuredly they will show, in the words of one of our leading global intellectuals (Taylor Swift), "…with all that's said and done / Two is better than one, two is better than one."

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Time is Brain: Skip the tPA and Head Straight for the Cath Lab!

Colin Derdeyn, M.D., *Washington University School of Medicine*

Time to reperfusion is the most important factor in recovery after an acute ischemic stroke: every minute counts.[1] Intravenous tPA has little or no impact on recanalization for a large majority of patients with large vessel occlusions.[2,3] In addition, it may add risk for symptomatic intracranial hemorrhage or groin access complication after endovascular therapy. Going straight to definitive revascularization as rapidly as possible has great potential for improving patient outcomes. The advent of newer mechanical thrombectomy devices over the past few years has dramatically improved time to recanalization and recanalization rates. These two factors are the primary drivers for the recent positive trials of endovascular stroke intervention. The equivalent outcomes in the two treatment groups in the SYNTHESIS Expension study comparing IV tPA to endovascular treatment alone (the majority intra-arterial tPA only), despite an additional hour to start endovascular treatment, suggests a repeat study with modern devices would show a benefit for the endovascular arm.[4] The major challenge for making this work is speed – door-to-clot. Process-related barriers clearly can be overcome. Door-to-balloon times of 20 minutes or less are obtainable for percutaneous coronary intervention for acute myocardial infarction. Door-to-clot times should not be much longer. Imaging triage is necessary, but this can be done faster. Several different methods could be evaluated: a non-contrast head CT may be sufficient for some patients. New angiographic imaging technology that allows non contrasted head CT scans, CT angiography and perfusion is in development.

In summary, eliminating IV tPA and getting to the clot as soon as possible is not crazy talk. The magnitude of benefit with more effective and efficient revascularization achieved with modern mechanical devices makes investigation of these different process-improvement approaches imperative.

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2015 AWARD INFORMATION

RAYMOND D. ADAMS LECTURESHIP

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 as the Massachusetts General Hospital. An ANA member at the Annual Meeting presents the lectureship.

David Hafler, M.D.,
Yale New Haven Hospital

Dr. Hafler is the William S. and Lois Stiles Edgerly Professor and Chairman Department of Neurology, Yale School of Medicine and is the Neurologist-in-Chief of the Yale-New Haven Hospital. He graduated magna cum laude in 1974 from Emory University with combined B.S. and M.Sc. degrees in biochemistry, and the University of Miami School of Medicine in 1978. He then completed his internship in internal medicine at Johns Hopkins followed by a neurology residency at Cornell Medical Center-New York Hospital in New York. Dr. Hafler received training in immunology at the Rockefeller University then at Harvard where he joined the faculty in 1984. He was one of the Executive Directors of the Program in Immunology at Harvard Medical School and was on the faculty of the Harvard-MIT Health Science and Technology program where he was actively involved in the training of graduate students and post-doctoral fellows. Dr. Hafler has been elected to membership in the American Society of Clinical Investigation, The American Neurological Association, the Alpha Omega Society, and was a Harvey Weaver Scholar of the National Multiple Sclerosis Society. He is currently a member of the editorial boards for *Journal of Clinical Investigation* and the *Journal of Experimental Medicine,* and is co-founder of the Federation of Clinical Immunology Societies. Dr. Hafler is a clinical scientist with a research interest in understanding the mechanism of autoimmunity with a particular interest in inflammatory central nervous system diseases, with over 300 publications in the field of autoimmunity and immunology. He received the 1st National Multiple Sclerosis five year Collaborative Center Award for tackling the MS genetic effort. Hafler leads the NIH Autoimmunity Prevention Center Grant at Yale, and was a Jacob Javits Merit Award Recipient from the NIH. His laboratory focuses on the understanding of human autoimmune diseases with the theme that investigation of naturally occurring human diseases give insight into the basic processes of T cell regulation, in addition to providing fundamental understanding and development of new therapies for human diseases. The laboratory has defined immunodominant epitopes of autoantigens, and has developed new technologies to measure both functionality and frequency of autoreactive T cells. More recently, Dr. Hafler has focused on broadly characterizing the molecular pathogenesis of the disease, both at the DNA, mRNA, and proteomic level. Dr. Hafler is a founding member of the International MS Genetic Consortium, a group recently formed to define the genetic causes of MS including scientists from University of Cambridge and University of California, San Francisco.

F.E. BENNETT MEMORIAL LECTURESHIP

Foster Elting Bennett, M.D., established a lectureship in memory of his son, which has been given to outstanding researchers and educators in neurology since 1979.

Erika Holzbaur, Ph.D.,
University of Pennsylvania

Erika Holzbaur received her B.S. in chemistry and history from the College of William and Mary, and her Ph.D. in biochemistry from the Pennsylvania State University. Her Ph.D. research focused on the mechanochemistry of dynein, and her postdoctoral work at the Worcester Foundation investigated the regulation of dynein by dynactin. Holzbaur joined the faculty at the University of Pennsylvania in 1992, and is now Professor of Physiology in the Perelman School of Medicine. The Holzbaur lab studies molecular motors, axonal transport, autophagy, and neurodegeneration. The lab is funded by National Institutes of Health, and was recently awarded a Javits Award from the National Institute of Neurological Disorders and Stroke.

DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR AWARD

The Derek Denny-Brown Young Neurological Scholar Award, one of the ANA's highest and most prestigious honors, 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.

This award was originally given to a newly elected member of the Association who had achieved significant stature in neurological research and who planned to continue making major contributions to the field of neurology. In 2011, this award was divided into two parts: basic science and clinical science. In 2014 a taskforce was established to broaden the criteria of this award to be more inclusive of the association's growing membership base.

Under this new criteria, the Membership, Honorary and Awards Committee can designate up to a total of three (3) awards in the following three areas:

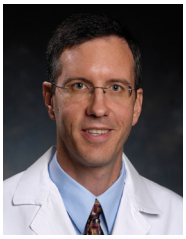
- Physician Scientist – Basic
- Physician Scientist – Clinical
- Neuroscientist – relevant to disease

Specifically, the award is now designed to recognize significant contributions by ANA members working in neurology and neuroscience in their first 10 years at the assistant/associate faculty (equivalent) level.

DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR AWARDS IN BASIC SCIENCE

Annapurna Poduri, M.D., M.P.H.
Harvard Medical School

Annapurna Poduri, M.D., M.P.H., is a physician-scientist with a focus on epilepsy genetics. Her goals include contributing to the genetic landscape of epilepsy, creating models of human epilepsy, and developing novel treatments. She serves on the faculty of Boston Children's Hospital Department of Neurology and directs the Epilepsy Genetics Program. She discovered somatic mutations as a cause of the malformation hemimegalencephaly, she has reported inherited forms of early onset epilepsy, and her team is modeling epilepsy genes in the zebrafish system. She is active in "team science" through the Epilepsy Phenome/Genome Project, Epi4K, and the Epilepsy Precision Medicine consortia.



Eric Roberson, M.D., Ph.D.
University of Alabama at Birmingham

Eric Roberson, M.D., Ph.D., earned his A.B. with highest honors from Princeton University and his M.D. and Ph.D. in neuroscience from Baylor College of Medicine, studying molecular mechanisms of learning and memory. He completed a residency in neurology at the University of California San Francisco, serving as Chief Resident in 2002-2003. He then trained in behavioral neurology with Dr. Bruce Miller at UCSF and resumed basic research in the laboratory of Dr. Lennart Mucke at the Gladstone Institute of Neurological Disease, initiating his current studies of neurodegenerative disease using mouse models. He joined the faculty at UAB in 2008.

DEREK DENNY-BROWN YOUNG NEUROLOGICAL SCHOLAR AWARD IN CLINICAL SCIENCE

Daniel Reich, M.D., Ph.D.,
National Institute of Neurological Disorders and Stroke

Daniel Reich, M.D., Ph.D., a neurologist and neuroradiologist – directs the Translational Neuroradiology Unit in the National Institute of Neurological Disorders and Stroke, part of National Institutes of Health. In his clinical practice, he cares for patients with multiple sclerosis and other neurological diseases, and he also leads several clinical studies focusing on multiple sclerosis. Research in his lab focuses on

CONTINUED

the use of advanced MRI techniques to understand the sources of disability in multiple sclerosis and on ways of adapting those approaches for research trials and patient care. He is particularly interested in harnessing noninvasive imaging modalities to dissect biological mechanisms of tissue damage.

DISTINGUISHED NEUROLOGY TEACHER AWARD

The Distinguished Neurology Teach Award was established in order to recognize outstanding accomplishments in teaching neurology students. The purpose is to encourage efforts to recognize and reward contributions by gifted and talented teachers in neurology.

Joseph Berger, M.D., F.A.C.P., F.A.A.N., F.A.N.A.,
University of Pennsylvania

Joseph R. Berger, M.D., F.A.C.P., F.A.A.N., F.A.N.A., is Professor of Neurology and Chief of the Multiple Sclerosis Division of the Department of Neurology at the Perelman School of Medicine at the University of Pennsylvania in Philadelphia. Dr. Berger was a summa cum laude graduate of the Pennsylvania State University – Jefferson Medical College 5 Year Accelerated Medical Program and is a member of national medical honor society, Alpha Omega Alpha. He completed his residency in internal medicine at Georgetown University Hospital and his neurology residency at University of Miami School of Medicine and is board certified in both internal medicine and neurology. In 1981, he joined the faculty of the University of Miami School of Medicine serving in both the Departments of Neurology and Internal Medicine. At that institution, he held the Whigham-Berger Endowed Chair for the study of the neurological complications of HIV/AIDS, the first endowed chair for the study of the neurological complications of HIV/AIDS. From 1995 through 2013, Dr. Berger was the Chairman of the Department of Neurology at the University of Kentucky where he was the Ruth L. Works Professor of Neurology and director of the UK Multiple Sclerosis Clinic. He is a fellow of the American College of Physicians, American Academy of Neurology and the American Neurological Association. His research interests include progressive multifocal leukoencephalopathy, the neurological complications of HIV/AIDS, multiple sclerosis, and other inflammatory disorders of the brain. He has published more than 230 refereed papers, more than 100 chapters, and has co-edited three textbooks (Berger JR and Levy RM: AIDS and the Nervous System, 2nd edition, Raven Press, New York, 1996; Nath A, Berger JR: Clinical Neurovirology, Marcel Dekker, New York, 2003; Portegies P, Berger JR: HIV/AIDS and the Nervous System, Elsevier, Amsterdam, 2007). Dr. Berger co-founded and chaired the first international conference on the neurological complications of HIV, the Neuroscience of HIV meeting. He also established the Commonwealth Neurological Society for neurologists in the state of Kentucky. Dr. Berger has a longstanding interest in international health and was one of the founding members of People-to-People, an organization for HIV/AIDS care and education in East Africa.

THE GRASS FOUNDATION – ANA AWARD IN NEUROSCIENCE

The Grass Foundation – ANA Award in Neuroscience was established in 2007 to honor outstanding young investigators doing research in basic or clinical neuroscience.

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

Dena B. Dubal M.D., Ph.D., is an Assistant Professor of Neurology at the University of California, San Francisco and holds the David A. Coulter Endowed Chair in Aging and Neurodegenerative Disease. She received her M.D. and Ph.D. degrees from the University of Kentucky College of Medicine and completed a medical internship and neurology residency at UCSF, where she also served as chief resident. Following a basic research and clinical fellowship in aging and dementias, she directs a laboratory with a research focus on longevity and brain resilience – and has discovered that "aging suppressors" such as the hormone klotho can boost brain function and counter neurologic diseases. Her work integrates genetic and molecular investigation of human populations with a range of mouse model systems to dissect causative pathways of resilience against neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease.

Dr. Dubal sees patients at the San Francisco General Hospital at UCSF. Among her recent honors, Dr. Dubal received the NIA/American Federation for Aging (AFAR) Paul Beeson Career Development Award for Research in Aging, the Glenn Award for Research in Biologic Mechanisms of Aging, and the AFAR Junior Faculty Award for Aging Research.

SORIANO LECTURESHIP

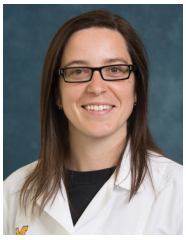
The first Soriano lecture was given in 1987 which marked the 40th year of consecutive attendance at ANA meetings by Victor Soriano and his wife. The couple chose to sponsor a lectureship to be given at the ANA, so that in future years the Sorianos would always be linked to all of you, through a brilliant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

Jeffrey Saver, M.D.,
UCLA Comprehensive Stroke Center

Dr. Saver is Director of the Comprehensive Stroke Center and Senior Associate Vice-Chair of Neurology at UCLA. He trained at Harvard Medical School, the Harvard-Longwood Neurology Training Program (neurology), the University of Iowa (neurobehavior), and Brown (vascular neurology). Author of over 470 research articles, two books, and 35 book chapters, Dr. Saver's research interests are in acute stroke treatment, stroke prevention, neuroimaging, clinical trial design, and neurocognitive consequences of stroke. He served as PI of the NIH-NINDS FAST-MAG trial and Chair of the American Heart Association Stroke Council. He is currently associate editor at *JAMA, the Journal of the American Medical Association*, and consulting editor at the journal, *Stroke*.

WOLFE NEUROPATHY RESEARCH PRIZE

The Wolfe Neuropathy Research prize was established in 2009 by Winston Wolfe and the ANA. The award was designed to honor an outstanding investigator who has identified a new cause or treatment of axonal peripheral neuropathy.



Lucy Hinder, Ph.D.,
University of Michigan

Lucy Hinder received her Ph.D. from the University of Aberdeen, Scotland, UK in 2007. Her graduate research focused on the neural control of altered skin blood flow and behavioral responses in experimental diabetes; assessing the efficacy of antioxidant, vasodilator and growth factor therapeutic strategies on functional and quantitative indices of diabetic neuropathy. Dr. Hinder joined Eva Feldman's lab in 2009 to pursue her postdoctoral work on the contribution of dyslipidemia to the development of diabetic neuropathy. As part of an NIH-funded Challenge Grant, Dr. Hinder explored the biological relevance of lipid metabolism gene expression signatures in diabetic peripheral nerves. Dr. Hinder is the recipient of the JDRF Angelika Bierhaus Postdoctoral Fellowship in Diabetic Complications, and is currently exploring diabetes-induced metabolic reprogramming within peripheral nerves, and the associations between oxidized lipids, inflammation and peripheral nerve injury.

2015 AWARD INFORMATION

TRAVEL AWARDS

Each year the ANA selects the top abstracts submitted by fellows or residents 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, September 27; poster numbers listed with an M will be presented on Monday, September 28.

Corresponding poster categories can be viewed on pages 17 and 24 of this program book.

Peter Abdelmalik, M.D., Ph.D., *Thomas Jefferson University Hospitals*
#S601 Propofol Exposure as a Risk Factor for the Development of Critical Care Neuromuscular Weakness in Patients with Sepsis and Acute Respiratory Failure

Daniel Abenroth, M.D., *University of Utah*
#M101 Lambert-Eaton Myasthenic Syndrome: Epidemiology and Therapeutic Response in the National Veterans Affairs (VA) Population

Samuel Ahn, B.A., *Albert Einstein College of Medicine*
#S302 Gamma Event Functional Connectivity (GEFC) and Graph Theory Measures in Mesial Temporal Lobe Epilepsy

Farwa Ali, M.B.B.S., *Mayo Clinic*
#S404 Camptocormia: The Mayo Clinic Experience

Charenya Anandan, M.D., *University of Toledo*
#M704 Autonomic Dysfunction (AD) Prevalence in Hospitalized Guillian-Barre Syndrome (GBS) Patients: Analysis of the Nationwide Inpatient Sample (NIS)

Helena Ariño, Hospital Clinic, *University of Barcelona*
#M103 GAD Autoimmunity: Syndromes, Comorbidities, and Coexisting Antibodies in 121 Patients

Thaís Armangue, M.D., *Institut d[apos]Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS) - Hospital Clínic*
#M104 Adult-Onset Opsoclonus-Myoclonus Syndrome (OMS): 136 Patients

Kunakorn Atchaneeyasakul, M.D., *University of Miami Miller School of Medicine*
#S202 Optimal Technique for Canine Mesenchymal Stem Cells Labeling with Novel SPIO, MIRB[trade]: for MRI Detection of Transplanted Stem Cells in Canine Stroke Model

Paula Barreras, M.D., *Johns Hopkins University School of Medicine*
#S203 Vascular Myelopathies: Clinical and Neuroimaging Profiles

Meredith Bock, B.A., *University of California San Francisco*
#M709 Cognitive-Behavioral Changes in Amyotrophic Lateral Sclerosis: Natural History and Impact on Patients and Caregivers

Raja Boddepalli, M.D., *Cleveland Clinic Florida*
#S433 Gait Disorders Etiology: Classification and Epidemiology

Hugo Botha, M.B.Ch.B., *Mayo Clinic*
#S101 Category Specific Imaging Correlates in Neurodegenerative Anomia

Stephen Briggs, *Albert Einstein College of Medicine*
#S303 Interleukin-1[beta] in the Pathogenesis of Infantile Spasms in Rats

Audrey Brumback M.D., Ph.D., *University of California, San Francisco*
#S102 Convergent Excitability Defects in Prefrontal Corticothalamic Pyramidal Neurons Link Genes to Behavior in Mouse Models of Autism

Jung-Ick Byun, M.D., *Seoul National University Hospital*
#M105 Efficacy and Safety of Rituximab in Autoimmune Encephalitis

Emily Coderre, Ph.D., *Johns Hopkins University School of Medicine*
#S103 Event-Related Potentials as Implicit Measures of Vocabulary in Individuals with Autism

Parker Cunningham, B.A., *Vanderbilt University*
#M503 Teleneurology Consultations for Acute Neurologic Emergencies

Sushrut Dharmadhikari, M.D., *University of Miami Miller School of Medicine*
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Alessandro Didonna, Ph.D., *University of California San Francisco*
#S504 A Multiple Sclerosis Disease-Risk Variant in EVI5 Links Susceptibility to the S1P Pathway

Chandrakanth Reddy Edamakanti, Ph.D., *Feinberg School of Medicine-Northwestern University*
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Kate Essad, M.D., *Dartmouth-Hitchcock Medical Center*
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Eoin Flanagan, M.B.B.Ch., *Mayo Clinic*
#M108 Basal Ganglia T1-Hyperintensity in LGI1-Autoantibody-Associated Faciobrachial Dystonic Seizures

Tarun Girotra, M.D., *Henry Ford Health System*
#S422 Amantadine Responsive Isolated Dropped Head Syndrome

Maxwell Greene, M.D., *University of Pennsylvania*
#M111 Delta/Notch-Like Epidermal Growth Factor-Related Receptor (DNER) is Not a Notch Ligand

Yong Guo, M.D., Ph.D., *Mayo Clinic*
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Elan Guterman, M.D., University of California, San Francisco
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Mallory Hacker, Ph.D., *Vanderbilt University*
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Yael Hacohen, M.R.C.P.C.H., *University of Oxford*
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Jeffrey Hakim, *Mayo Clinic*
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Lauren Heusinkveld, *Vanderbilt University*
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Saif Huda, M.R.C.P., *University of Oxford*
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Cecilia Kelly, M.D., *Emory University School of Medicine*
#M728 Divergent Behavioral and Cognitive Trajectories in ALS

Eun Hye Kim, B.A., B.S., *Ohio State University College of Medicine*
#S107 Impaired Emotional Empathy

Sidney Le, B.A., *University of California, San Francisco*
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David Lerner, M.D., *Duke University Hospital*
#S223 The National Institutes of Health Stroke Severity Score for Stroke Localization

Nicolas Madigan, M.B.B.Ch., Ph.D., *Mayo Clinic*
#M736 GDNF-Secreting Schwann Cells in Multichannel OPF+ Hydrogel Scaffolds Promote Ascending Axonal Regeneration, Remyelination and Partial Locomotor Recovery Following Complete Spinal Cord Transection in Rats

Michael Marshall, *University of Illinois at Chicago College of Medicine*
#M737 AAV9 Gene Therapy Synergizes with Hematopoietic Replacement to Prevent Major Neurological Defects in a Mouse Model of Krabbe Disease

Jennifer Martinez-Thompson, M.D., *Mayo Clinic*
#M738 Ganglioside Composite Autoantibody Testing Aids in Identification of Immunotherapy Responsive Multifocal Motor Neuropathy (MMN)

Courtney McIlduff, M.D., *Beth Israel Deaconess Medical Center/Harvard Medical School*
#M739 Electrical Impedance Myography of the Tongue at 200 kHz Provides Improved Discrimination of Healthy Subjects from Neuromuscular Disease Patients

Sarah Millan, *Vanderbilt University Medical Center*
#S438 Investigating the Influence of BDNF rs6265 on BMI in Early Stage Parkinson's Disease

Shahnaz Miri, M.D., *SUNY Downstate Medical Center*
#S439 Visual Function Testing and Retinal Imaging in Parkinson[apos]s Disease Diagnosis

Neda Najimi, *University of Maryland, Baltimore*
#M742 Mitochondrial Transcription Factor a Regulation of Mitochondrial Degeneration in Experimental Diabetic Neuropathy

Aaron Noles, *Wayne State University School of Medicine*
#S228 Race and Gender Influence Medical Therapy Among Patients Undergoing Carotid Revascularization

Hidenori Ogata, M.D., *Neurological Institute, Graduate School of Medical Sciences, Kyushu University*
#M123 IgG4 Neurofascin 155 Antibody-Positive Hypertrophic Demyelinating Polyneuropathy

Achint Agamsharan Patel, M.D., *Icahn School of Medicine at Mount Sinai*
#S232 Obesity Paradox in Patients Hospitalized for Acute Ischemic Stroke: An Analysis of Nationwide Inpatient Sample Data

Urvish Patel, M.D., *Icahn School of Medicine at Mount Sinai*
#M607 Endovascular Mechanical Thrombectomy Outcomes Among Obese and Smokers Hospitalized with Acute Ischemic Stroke

Qi Qin, *Harbin Medical University*
#S443 Effect of LRRK2 Inhibitors on Dopamine Release in the Striatum and Dopamine Neuron Firing in the SNc

Nicole Reams, M.D., *University of Michigan*
#M218 Proposed Guidelines for the Clinical Diagnosis of Chronic Traumatic Encephalopathy (Traumatic Encephalopathy Syndrome)

Faith Robertson, B.S., *Harvard University*
#S239 The Impact of Early Intervention on Outcomes After Decompressive Craniectomy for Stroke: A Nationwide Analysis

Mark Rubin, M.D., *Mayo Clinic*
#S312 Seizure Localization with IntraCranial hEmodynamics (SLICE)

Kavelin Rumalla, *University of Kansas Medical Center*
#S615 Surgical Treatment of Traumatic Subdural Hematoma: Weekday Versus Weekend Admission

Amy Rumora, Ph.D., *University of Michigan*
#M750 Hyperlipidemia Alters Mitochondrial Trafficking in Sensory Neurons

Ali Saber Tehrani, M.D., *University of Illinois College of Medicine*
#S243 A Radiographic Target Sign for Abnormal Vertebral Artery Flow in Stroke Patients with Acute Vestibular Syndrome

Joel Salinas M.D., M.B.A., *Massachusetts General Hospital, Harvard Medical School*
#S110 Social Support, BDNF, and the Risk for Stroke and Dementia: The Framingham Heart Study

2015 AWARD INFORMATION CONTINUED

TRAVEL AWARDS *(continued)*

Benjamin Sanchez, Ph.D., *Beth Israel Deaconess Medical Center*
#M753 Electrical Impedance Techniques Detect the Effects of Myostatin Inhibition in Wild-Type Mice

Andrea Schneider, M.D., Ph.D., *Johns Hopkins University*
#M220 Diabetes, Pre-Diabetes and Brain Volumes: Atherosclerosis Risk in Communities Neurocognitive Study (ARIC-NCS)

Jason Sico, M.D., *Yale University School of Medicine*
#S247 International Variability in Preventive Care in a Stroke Prevention Clinical Trial

David Soleimani-Meigooni, M.D., *Washington University School of Medicine*
#M126 JC Virus Granule Cell Neuronopathy in the Setting of Chronic Lymphopenia Treated with Recombinant Interleukin-7

Cesar Soria, B.A., Biochemist, *University of Chicago*
#S450 Developing a Novel miRNA-Mediated Therapeutic Approach for SCA6

Nicholas Sterling, M.S., *Pennsylvania State University-Milton S. Hershey Medical Center*
#S452 Higher Plasma LDL-Cholesterol Is Associated with Preserved Executive Function in Parkinson's Disease

Maxim Turchan, B.A., *Vanderbilt University School of Medicine*
#S455 Discrepancies in Informed Consent Rates for Patients and Proxies

Jie Wen, Ph.D., *Washington University*
#S511 In Vivo Quantitative Evaluation of Cortical Gray Matter Damage in Multiple Sclerosis

Janice Wong, M.D., *Brigham and Women's and Massachusetts General Hospital*
#M510 Modeling Traumatic Brain Injury in Africa in 2050

Sukhvir Wright, M.B.B.S., *Birmingham Children's Hospital*
#M127 N-methyl-D-aspartate Receptor (NMDA) Antibody Encephalitis Mimicking Autistic Regression in Infants

Nicholas Zalewski, M.D., *Mayo Clinic*
#M128 Neuronal Calcium Channel Autoantibodies in a Paraneoplastic Serological Context

NATURE REVIEWS NEUROLOGY'S POSTER PRIZE

Crystal Dixon, M.D., University of South Florida
#S516 WIP Rapid Elimination Procedure of Teriflunomide with Colestipol Hydrochloride

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Professor P. Satish Chandra
Director/Vice-Chancellor
National Institute of Mental Health and Neuro Sciences (NIMHANS)

Participating in the Interactive Lunch Workshop titled "Non-Lesional Drug-Resistant Focal Epilepsy: Controversies in Diagnostic Evaluation and Surgical Management"

Professor Satish V. Khadilkar
Head of Dept. of Neurology
Grant Medical College and Sir J.J. Group of Hospitals

Participating in the Interactive Lunch Workshop titled "Current Ethical Dilemmas in Neurology"

Professor Arbinda Mukherjee
President, *Indian Academy of Neurology*
Professor, *Vivekanand Institute, Kolkata*

Participating in the Interactive Lunch Workshop titled, "Neurology Goes Global - International Issues in Diagnostics & Therapeutics"

MONDAY SEPTEMBER 28 11:45 AM–1:00 PM

Professor UK Misra
Professor & Head, Department of Neurology
Sanjay Gandhi Post Graduate Institute of Medical Sciences

Participating in the Interactive Lunch Workshop titled "Emerging CNS Infection"

Professor Kameshwar Prasad
Prof. & Head, Department of Neurology; Director,
Clinical Epidemiology Unit
All India Institute of Medical Sciences

Participating in the Interactive Lunch Workshop titled,
"Challenges and Innovation in Prehospital Stroke Care"

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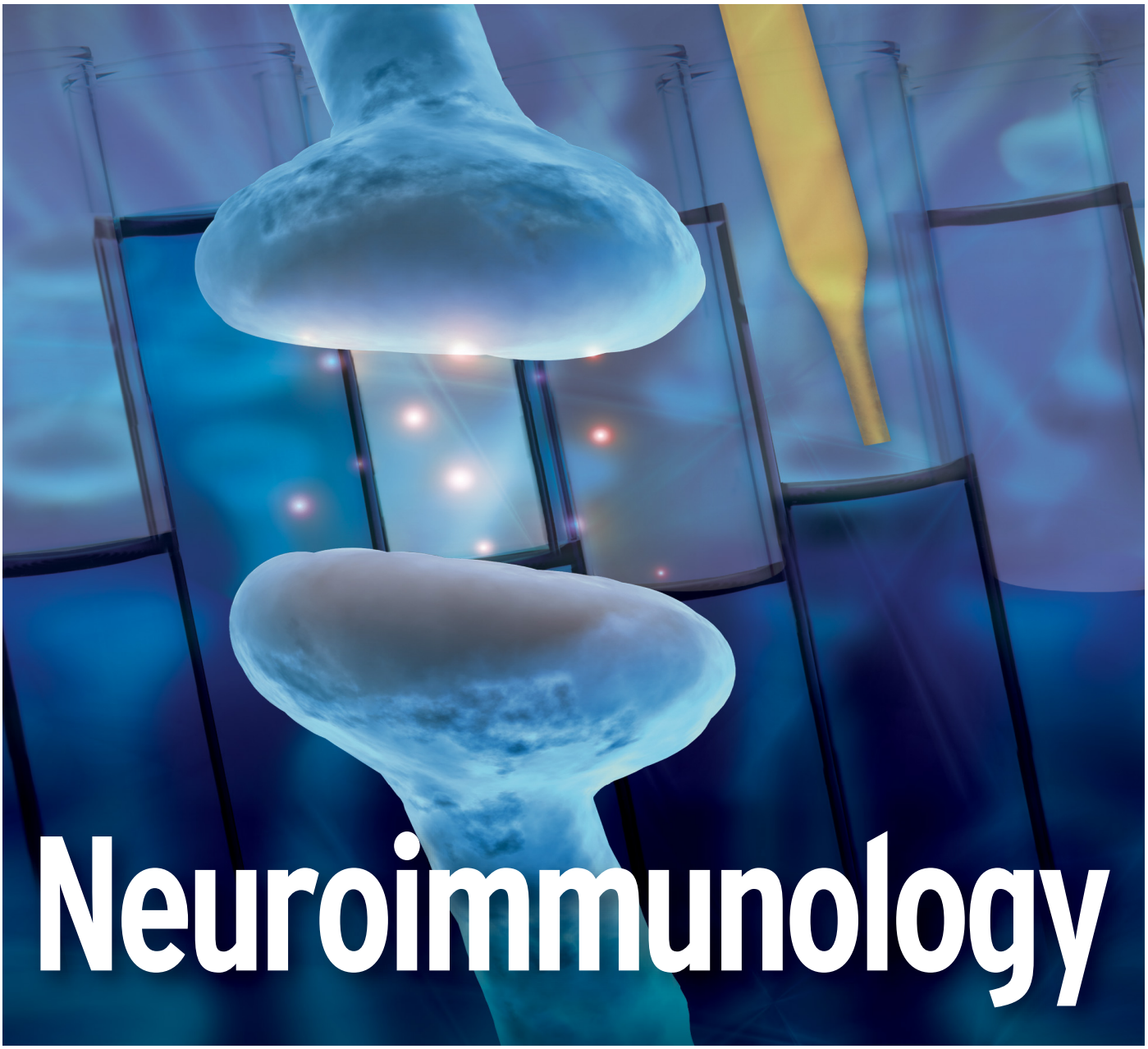

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