American Association of Neuropathologists 93RD ANNUAL MEETING

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS, INC.

June 8 - 11, 2017

Hyatt Regency Orange County
Garden Grove, California

This activity is provided by the American Association of Neuropathologists.

TABLE OF CONTENTS

The American Association of Neuropathologists, Inc.
5575 S. Sycamore St., Suite 235
Littleton, Colorado 80120
Phone: 720-372-0888; Fax: 303-568-0406
Email: aanp@aoeconsulting.com

Dear Colleagues:

It is with great pleasure that I welcome you to the 93rd Annual Meeting of the American Association of Neuropathologists in Orange County, CA.

For over 90 years, the AANP has been working to create new, innovative educational content in the field of neuropathology through scientific research and dissemination of that content through publications, lectures and courses. This year's conference will connect you to key leaders as well as provide the latest insights into cutting-edge science and groundbreaking research.

While the learning you'll receive at the 93rd Annual Meeting is unmatched, you'll have abundant opportunity to forge new relationships and strengthen existing ties with neuropathology colleagues from all over the globe. An exhibitor space with representatives ready and eager to discuss their capabilities and opportunities is just another great highlight to the conference.

What can you expect over the next few days? This year's annual meeting includes a one-day Special Course dedicated to *"Genetics, Genomics, and Epigenomics in Clinical Neuropathology Practice"* and *"Understanding Autism,"* two days of scientific platform and poster sessions presenting original research, an evening Diagnostic Slide Session, and a half-day Presidential Symposium focusing on *"Recent Advances and Reviews in the Field of Neuropathology."*

As my year as President approaches its end, I look forward to coming together to share knowledge and insights, promote discovery and advance the field of neuropathology. Thank you for your energy and commitment towards shaping a bright future. I hope the 93rd Annual Meeting is a rewarding and memorable experience for each of you.

Sincerely,

Arie Perry, MD
President
The American Association of Neuropathologists


Save the Date!
94TH ANNUAL MEETING
June 7-10, 2018 / Hyatt Regency Louisville
95TH ANNUAL MEETING
June 6-9, 2019 / Grand Hyatt Atlanta
All meetings to be held the second weekend of June
American Association of Neuropathologists

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

AANP OFFICE

5575 S. Sycamore St., Suite 235, Littleton, CO 80120
Phone: 720-372-0888; Fax: 303-568-0406
Email: aanp@aoeconsulting.com

OFFICERS

Arie Perry, MD, *University of California San Francisco*, President
Cheryl A. Palmer, MD, *University of Utah,* Vice President
Elizabeth Cochran, MD, *Medical College of Wisconsin*, President-Elect
Daniel J. Brat, MD, PhD, *Emory University School of Medicine*, Vice President-Elect
Kathy L. Newell, MD, *University of Kansas Medical Center*, Vice President of Professional Affairs
Karen M. Weidenheim, MD, *Montefiore Medical Center*, Secretary-Treasurer
R. Ross Reichard, MD, *Mayo Clinic*, Assistant Secretary-Treasurer

OTHER EXECUTIVE COUNCIL MEMBERS

Eileen H. Bigio, MD, *Feinberg School of Medicine, Northwestern University*
Rebecca Folkerth, MD, *New York City Office of the Chief Medical Examiner*
Michael N. Hart, MD, *University of Wisconsin School of Medicine*
Anthony T. Yachnis, MD, *University of Florida College of Medicine*
Thomas J. Montine, MD, PhD, *Stanford University School of Medicine*
Suzanne Z. Powell, MD*, Houston Methodist Hospital*

ARCHIVIST

Michael N. Hart, MD, *University of Wisconsin School of Medicine*

OFFICIAL JOURNAL

Journal of Neuropathology and Experimental Neurology
Michael N. Hart, MD, Editor-in-Chief
Eileen S. Healy, Managing Editor
E-mail: jnen@pathology.wisc.edu
Home page: http://www.jneuropath.com

DIAGNOSTIC SLIDE SESSION

Caterina Giannini, MD, PhD, *Moderator*
Rebecca Folkerth, MD, *Manager*

COUNCILORS TO THE INTERNATIONAL SOCIETY OF NEUROPATHOLOGY

Alexander Judkins, MD, *Children's Hospital Los Angeles*
George Perry, PhD, *The University of Texas at San Antonio*
Arnulf H. Koeppen, MD, *Albany Stratton Veterans Affairs Medical Center*
E. Tessa Hedley-Whyte, MD, *Massachusetts General Hospital; Harvard Medical School*
Adekunle Adesina, MD, PhD, *Texas Children's Hospital*

AANP COMMITTEES

Awards Committee

Fausto Rodriquez, MD (Chair)
Jennifer Baccon, MD, PhD
Ewa Borys, MD
Cynthia Hawkins, MD, PhD
Cheng-Ying Ho, MD, PhD, FRCPC
Kyle Hurth, MD, PhD
Jamie Jacobsohn, MD
Julia Kofler, MD
Mirna Lechpammer, MD, PhD
Norman L. Lehman, MD, PhD
James W. Mandell, MD, PhD
Maria Martinez-Lage Alvarez, MD
Rupal Mehta, MD
Carrie Ann Mohila, MD
David Nauen, MD, PhD
Hilary Highlfield Nickols, MD, PhD
Richard Perrin, MD, PhD
Edward Plowey, MD, PhD
Matija Snuderl, MD
Joshua Sonnen, MD

Education Committee

R. Ross Reichard, MD (Chair)
Jennifer Baccon, MD, PhD
Brent Harris, MD
Jesse Kresak, MD
Edward Lee, MD
Quinwen Mao, MD, PhD
Maria Martinez-Lage Alvarez, MD
Anat Stemmer-Rachamimov, MD
Jane Uyehara-Lock, MD
Cindy Welsh, MD
Charles L. White III, MD
Gabrielle A. Yeaney, MD
Rachel Vaubel, MD, PhD*

Constitution Committee

Amyn Rojiani, MD, PhD (Chair)
Steven Carroll, MD, PhD
Andreana Rivera, MD
Alexander Z. Feldman, MD*

Membership Committee

Peter Pytel, MD (Chair)
Sarah E. Bach, MD
Leslie Hamilton, MD
Melike Pekmezci, MD
Jose Velazquez Vega, MD*
Roberta Seidman, MD
Bret Mobley, MD

Nominating Committee

Suzanne Z. Powell, MD (Chair)
Thomas J. Montine, MD, PhD
Anthony T. Yachnis, MD
Charles L. White III, MD

Professional Affairs Committee

Kathy L. Newell, MD (Chair)
Eileen H. Bigio, MD
Dan J. Brat, MD, PhD
William H. Yong, MD
Mariarita Santi-Vicini, MD, PhD
Steven Dubner, MD
Douglas Anthony, MD, PhD
Bradley Miller, MD, PhD
Leomar Y. Ballester, MD, PhD*

Program Committee

Sriram Venneti, MD, PhD (Chair)
Giselle Y. Lopez, MD, PhD*
Thomas Beach, MD
Ivana Delalle, MD
Kimmo J. Hatanpaa, MD, PhD
Charles Specht, MD
Miguel Guzman, MD
Michael Lawlor, MD
Adekunle Adesina MD, PhD
Han Lee, MD
Anne Hiniker, MD, PhD
Richard Perrin, MD, PhD
Julie Kofler, MD
Nancy C. Kois, MD, FCAP
Michelle Madden Felicella, MD
Brent Orr, MD, PhD

Website Committee (Ad Hoc of Professional Affairs)

Douglas Anthony, MD, PhD (Chair)
John Crary, MD, PhD
Luis F. Gonzalez-Cuyar, MD
Jingxin Qiu, MD, PhD
Henry G. Brown, MD, PhD
Sonika Dahiya, MD
Kar-Ming Fung, MD, PhD
Charles L. White, III, MD
Murat Gokden, MD
Edward Lee, MD
Brian Moore, MD
Hilary Nickols, MD, PhD
J. Stephen Nix, MD
Michael Punsoni, MD*

*Affiliate Member

CME INFORMATION

TARGET AUDIENCE

The educational design of the AANP's Annual Meeting addresses the needs of physicians and scientists in the field of neuropathology who are involved in the diagnosis and/or treatment of patients with neurological disorders.

STATEMENT OF NEED

The purpose of this activity shall be to advance medical and scientific knowledge, understanding, and competence in the practice of neuropathology. The practice of neuropathology is understood to include diagnosis of diseases of the nervous system, scientific investigation into their causes, and teaching of neuropathology principles to colleagues and trainees.

LEARNING OBJECTIVES

Upon completion of this activity, participants should be able to:

I. Cite 2-3 examples of new information regarding the underlying etiologies and mechanisms of neurologic diseases.

II. Discuss research findings related to the genetics, genomics and general mechanisms related to CNS neoplasia, infectious diseases, muscle disorders, and developmental pathology, including autism.

III. Identify new methodologic and diagnostic knowledge that can improve the daily clinical practice and teaching of neuropathology.

DISCLAIMER

Participants have an implied responsibility to use the newly acquired information to enhance patient outcomes and their own professional development. The information presented in this activity is not meant to serve as a guideline for patient management. Any procedures, medications, or other courses of diagnosis or treatment discussed in this activity should not be used by clinicians without evaluation of patient conditions and possible contraindications on dangers in use, review of any applicable manufacturer's product information, and comparison with recommendations of other authorities.

PHYSICIAN ACCREDITATION STATEMENT

The American Association of Neuropathologists is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

PHYSICIAN CREDIT DESIGNATION

The American Association of Neuropathologists designates this live educational activity for a maximum of 25.0 *AMA PRA Category 1 Credits™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

CME INFORMATION (Continued)

CME CREDIT

Instructions to Receive Credit

In order to receive credit for this activity, the participant must complete the CME evaluations and credit applications for sessions attended, which are made available through the AANP Meeting App or by using the following link http://eventmobi.com/aanp2017**.**

The chart below outlines which sessions are offered for CME credit and the maximum number of credit hours a physician can earn for each educational activity being certified for *AMA PRA Category 1 Credit™* at this year's Annual Meeting.

Activity	CME Credit Hours
Special Course	6.75
Scientific Sessions	8
Korey Lecture	1
DeArmond Lecture	1
Parisi Lecture	1
Moore Lecture	1
What Every Neuropathologist Needs to Know	1
Diagnostic Slide Session	3
Presidential Symposium	2.25
Total	**25**

CONTACT INFORMATION

For any questions regarding the accreditation of this meeting, please contact AANP's CME Coordinator, Sarah Schott, via e-mail at: sschott@aoeconsulting.com, or via phone at: 303-557-0859 x84.

DISCLOSURE INFORMATION

Disclosure of Commercial Support:
This activity is supported by an educational, in-kind donation of microscopes, provided by Nikon Instruments, Inc.

Disclosure of Unlabeled Use:
This educational activity may contain discussion of published and/or investigational uses of agents that are not indicated by the FDA. The American Association of Neuropathologists does not recommend the use of any agent outside of the labeled indications.

The opinions expressed in this educational activity are those of the faculty and do not necessarily represent the views of any organization associated with this activity. Please refer to the official prescribing information for each product for discussion of approved indications, contraindications and warnings.

Disclosure of Conflict of Interest:
The American Association of Neuropathologists requires instructors, planners, managers and other individuals who are in a position to control the content of this activity to disclose any real or apparent conflict of interest they may have as related to the content of this activity. All identified conflicts of interest are thoroughly vetted by AANP for fair balance, scientific objectivity of studies mentioned in the materials or used as the basis for content, and appropriateness of patient care recommendations.

Planners and Managers

*The following planners and managers have **nothing to disclose**:*

Adekunle **Adesina**, Jennifer **Baccon**, Thomas **Beach**, Eileen **Bigio**, Dan **Brat**, , Elizabeth **Cochran**, Jennifer **Cotter**, Ivana **Delalle**, Charles **Eberhart**, David **Ellison**, Rebecca **Folkerth**, Caterina **Giannini**, Miguel **Guzman**, Brian **Harding**, Brent **Harris**, Michael **Hart**, Kimmo **Hatanpaa,** Cynthia **Hawkins**, Anne **Hiniker**, , Julia **Kofler**, Nancy **Kois**, Jesse **Kresak**, Edward **Lee**, Han **Lee**, Giselle **Lopez**, Michelle **Madden Felicella**, Qinwen **Mao**, Maria **Martinez**-**Lage**, Ashley **Marostica**, Thomas **Montine**, Kathy **Newell**, Brent **Orr**, Cheryl **Palmer**, Melike **Pekmezci**, Richard **Perrin**, Arie **Perry**, Edward **Plowey**, Suzanne **Powell**, Veena **Rajaram**, R. Ross **Reichard**, Fausto **Rodriguez**, Mariarita **Santi**-**Vicini**, David **Solomon**, Charles **Specht**, Anat **Stemmer-Rachamimov**, Jane **Uyehara-Lock**, Rachael **Vaubel**, Sriram **Venneti**, Karen **Weidenheim**, Cindy **Welsh**, Charles **White**, Anthony **Yachnis**, Gabrielle **Yeaney**

The following **planners/managers** reported the following financial relationships or relationships to products or devices they or their spouse/ partner have with commercial interests related to the content of this CME Activity.

Michael Lawlor	**Grant/Research Support:** Audentes Therapeutics, Solid GT, Demeter Therapeutics **Honoraria:** Audentes Therapeutics

DISCLOSURE INFORMATION (Continued)

Faculty

*The following faculty have **nothing to disclose:***

Malak **Abedalthagafi**, Jason **Adams**, Homa **Adle-Biassette**, Sanda **Alexandrescu**, Heather **Ames**, Matthew **Anderson**, Rajnish **Bharadwaj**, Melissa **Blessing**, Sandra **Camelo-Piragua**, David **Capper**, Manuel **Casanova**, Liam **Chen**, Jason **Chiang**, Leila **Chimelli**, Rati **Chkheidze**, Woon **Chow**, Patrick **Cimino**, Nicholas **Coley**, Jennifer **Cotter**, Laura **Cracco**, Travis **Danielsen**, Marc **Del Bigio**, Ivana **Delalle**, John **DeWitt**, Dennis **Dickson**, Meghan **Driscoll**, Charles **Eberhart**, David **Ellison**, Lyndsey **Emery**, Jennifer **Eschbacher**, Arline **Faustin**, Rebecca **Folkerth**, Andrew **Gao**, Bernardino **Ghetti**, Ahmed **Gilani**, Pallavi **Gopal**, Andrew **Guajardo**, Natalya **Hakim**, Cynthia **Hawkins**, Marco **Hefti**, Anne **Hiniker**, Cheng-Ying **Ho**, Eric **Huang**, Anita **Huttner**, Karra **Jones**, Patrick **Kiernan**, Joshua **Klonoski**, Edward **Lee**, Jiancong **Liang**, M. Beatriz **Lopes**, David **Louis**, Seth **Love**, Hsiang-Chih **Lu**, Qinwen **Mao**, Marta **Margeta**, Eliezer **Masliah**, Evan **Matshes**, William **McDonald**, Ann **McKee**, Brian **Moore**, Steven **Moore**, Daniel **Mordes**, Robert **Mrak**, Peter **Nelson**, Ho Keung **Ng**, James **Nix**, Cheryl **Palmer**, Richard **Perrin**, Arie **Perry**, David **Pisapia**, Edward **Plowey**, Yuan **Rong**, Matthew **Rose**, Namita **Sinha**, Priya **Skaria**, David **Solomon**,, Di **Tian**, Kathryn **Urankar**, David **Van Essen**, Sriram **Venneti**, Angela **Viaene**, Erik **Williams**, Drew **Williamson**, Matthew **Wood**, Paul **Yell**

The following **faculty** reported the following financial relationships or relationships to products or devices they or their spouse/ partner have with commercial interests related to the content of this CME Activity.

Michael Lawlor	**Grant/Research Support:** Audentes Therapeutics, Solid GT, Demeter Therapeutics **Honoraria**: Audentes Therapeutics
Mirna Lechpammer	**Employee (spouse):** Pfizer, Inc. **Stock Shareholder:** Jazz Pharmaceuticals, Tesaro
Sean Pittock	**Consultant/Independent Contractor:** Alexion Pharmaceutical, MedImmune LLC, and Chugai Pharma **Grant/Research Support:** Alexion Pharmaceutical
Shakti Ramkissoon	**Employee:** Foundation Medicine, Inc. **Stock Shareholder:** Foundation Medicine, Inc.
Steve Miller	**Consultant:** Luminex Corp.
Matthew State	**Consultant:** BlackThorn Therapeutics, ArRETT, Pfizer **Stock Shareholder:** BlackThorn Therapeutics, ArRETT

ANNUAL MEETING SELF-ASSESSMENT MODULES

Shortly after the 93rd Annual Meeting the following online self-assessment modules (SAMs) will launch. The SAMs will be available at www.neuropath-education.org.

SAM Title	SAM Credit Hour(s)
2017 Presidential Symposium	2.25
2017 Special Course	6.75
2017 Diagnostic Slide Session	3.00
2017 DeArmond Lecture: Structure, Function, Connectivity, Development and Evolution of Human Cerebral Cortex	1.00
2017 Korey Lecture: Disease Modifying Therapeutical Approaches for Synucleinopathies of the Aging Population	1.00
2017 Parisi Lecture: Autoimmune Gliopathies: A Journey of Discovery	1.00
2017 Moore Lecture: An Update of the WHO Classifications of Tumors of the Pituitary Gland, 4th Edition	1.00

Important information regarding Annual Meeting SAMs:

- You will need to use your old website log-in via Dayspring to gain access to these SAMs.
- Each SAM costs $25.00 unless you previously purchased the SAMs bundle
- To participate in each SAM, you must have attended the live session held at the 2017 AANP Annual Meeting in Orange County, CA.
- To purchase the SAMs bundle visit this link: www.neuropath.org/sams-bundle. Please note there is a one to two week delay in unlimited access being set-up on your Dayspring account.

GENERAL INFORMATION

LOCATION

Hyatt Regency Orange County
11999 Harbor Boulevard
Garden Gove, CA 92840

All meeting sessions will be held at the Hyatt Regency Orange County.

All platform presentations and general sessions (Special Course, Korey Lecture, DeArmond Lecture, Parisi Lecture, Moore Lecture, Business Meetings, Diagnostic Slide Session, and Presidential Symposium) will be held in **Grand Ballroom ABCD & Grand Ballroom EFG** of the hotel on the main floor.

All poster sessions will be held in **Garden 2 & 3, N. Tower** on the main floor.

REGISTRATION DESK

Grand Rotunda	
Wednesday, June 7	4:00 pm – 8:00 pm
Thursday, June 8	7:00 am – 5:00 pm
Friday, June 9	7:00 am – 5:00 pm
Saturday, June 10	7:00 am – 5:00 pm
Sunday, June 11	7:00 am – 12:00 pm

PRE-REGISTRATION PICK-UP

Attendees pre-registered and pre-paid for the meeting will have their name badge, program booklet, and the June 2017 issue of the *Journal of Neuropathology and Experimental Neurology (JNEN), inclusive of Annual Meeting* abstracts, ready for pick-up at the AANP Registration Desk, located in the **Grand Rotunda**, directly across from the hotel front desk area. On-site registration and additional tickets for the Annual Reception will be available at the registration desk. Registration receipts are available upon request.

NAME BADGE REQUIREMENT

Your name badge is required for admittance to any function of the Association, including the Special Course, all Friday, Saturday and Sunday sessions, and the Thursday evening reception.

MICROSCOPE VIEWING ROOM

Multi-headed microscopes will be available in **Salon III – N. Tower** on the second floor of the hotel.

Location: Salon III – N. Tower (2nd Floor)	
Thursday, June 8	7:00 am – 7:30 pm
Friday, June 9	7:00 am – 7:30 pm
Saturday, June 10	7:00 am – 7:30 pm
Sunday, June 11	7:00 am – 10:30 am

GENERAL INFORMATION (Continued)

SPECIAL MEETINGS BY INVITATION

Day/Date	Meeting	Time/Location
Wednesday, June 7	**NP Program Directors Meeting**	4:30 pm – 6:30 pm **Pacific Room – N. Tower**, 2nd Floor
	Education Committee Meeting	6:30 pm – 9:30 pm **Harbor Room – N. Tower**, 2nd Floor
Thursday, June 8	**Awards Committee Meeting #1**	5:30 pm – 6:00 pm **Pacific Room – N. Tower**, 2nd Floor
	Executive Council Meeting	7:00 pm – 10:00 pm **Salon I – N. Tower**, 2nd Floor
Friday, June 9	**Trainee Luncheon*** ***Open to all Trainees and Travel Award Winners**	11:45 am – 2:00 pm **Salon VII-VIII – N. Tower**, 2nd Floor
	Website Committee Meeting	12:30 pm – 1:30 pm **Salon II – N. Tower**, 2nd Floor
	Awards Committee Meeting #2	5:30 pm – 6:30 pm **Harbor Room – N. Tower,** 2nd Floor
	Professional Affairs Committee Meeting	5:30 pm – 7:00 pm **Salon IV – N. Tower**, Main Floor
Saturday, June 10	***JNEN* Editorial Board Meeting**	7:00 am – 8:00 am **Harbor Room – N. Tower**, 2nd Floor
	Awards Committee Meeting #3	6:00 pm – 8:00 pm **Salon VI – N. Tower**, 2nd Floor
	Presidential Reception	6:00 pm – 8:00 pm **Malibu Suite** – **N. Tower**, 2nd Floor
Sunday, June 11	**DSS Founders Breakfast**	7:00 am – 8:00 am **Salon IV – N. Tower**, 2nd Floor

ANNUAL RECEPTION

The annual reception will be held outside from 5:30 pm to 7:30 pm, Thursday, June 8 at the **Pool – N. Tower** on the main floor of the Hyatt Regency. Registrants and guests of the AANP are welcome to attend. Additional tickets are $20 each for guests of AANP attendees, and may be purchased at the time of registration, or at the door.

Pool – N. Tower, Main Floor (Outside)	
Thursday, June 8, 2017	5:30 pm – 7:30 pm

TRAINEE LUNCHEON

Trainees and Travel Award winners are invited to attend the 2017 Trainee Luncheon on Friday, June 9 in **Salon VII-VIII – N. Tower** (2nd Floor), hosted by Dr. Suzanne Powell. Lunch will be provided, followed by dessert. The agenda is posted below.

2017 Trainee Luncheon Agenda

I. 11:45 am – 12:15 pm: Welcome & Lunch
II. 12:15 pm – 12:20 pm: JNEN Overview: Dr. Ray Sobel
III. 12:15pm – 1:00 pm: Panel Discussion: Expectations of the Job Market
IV. 1:00 pm – 1:15 pm: Travel Awards Recognition
V. 1:15 pm – 1:20 pm: Book Raffle Results
VI. 1:20 pm – 1:45 pm: Dessert and Mingle with Executive Council

EXHIBITORS & SPONSORS

Thank you to our 2017 exhibitors and sponsors! Please visit the exhibit booths in the Grand Ballroom Foyer.

Location: Grand Ballroom Foyer	
Thursday, June 8	7:00 am – 5:30 pm
Friday, June 9	7:00 am – 5:30 pm
Saturday, June 10	7:00 am – 5:30 pm

EXHIBITORS

OXFORD JOURNALS OXFORD UNIVERSITY PRESS	Oxford University Press is the proud publisher of the *Journal of Neuropathology & Experimental Neurology* (*JNEN*). OUP also publishes some of the most renowned and respected medicine books and journals in the world. Visit our stand for promotional items, free copies of *JNEN* and to ask any questions regarding the journal. **Note:** Copies of the June Edition of the *JNEN* have been provided by Oxford University Press.	Booth #1
Booth #2	Leica Biosystems is a global leader in workflow solutions and automation. As the only company to own the workflow from biopsy to diagnosis, we are uniquely positioned to break down the barriers between each of these steps. Our mission of "Advancing Cancer Diagnostics, Improving Lives" is at the heart of our corporate culture. Our easy-to-use and consistently reliable offerings help improve workflow efficiency and diagnostic confidence. The company is represented in over 100 countries. It has manufacturing facilities in 9 countries, sales and service organizations in 19 countries, and an international network of dealers. The company is headquartered in Nussloch, Germany. Visit LeicaBiosystems.com for more information.	Leica BIOSYSTEMS
ELSEVIER	Elsevier is a world-leading provider of information solutions that enhance the performance of science, health, and technology professionals, empowering them to make better decisions, and deliver better care.	Booth #3
Booth #4	Nikon Instruments Inc. is a world leader in the development and manufacture of optical and digital imaging technology for biomedical and clinical applications. Now in its 100th year, Nikon provides imaging systems that offer optimal versatility, performance and productivity. Cutting-edge instruments include microscopes, digital imaging products and software.	Nikon
SUDC FOUNDATION	The SUDC Foundation's mission is to eliminate the tragedy of sudden unexpected and unexplained death in childhood. A centralized resource supporting grieving families worldwide, it develops support and advocacy programs specific to their needs. It works with families, professionals and the general public to share information, advance medical research, and advocate for issues relative to SUDC.	Booth #5
Booth #6	Menarini Silicon Biosystems has developed the DEPArray™ NxT, an instrument to sort, manipulate, and collect individual rare cells or groups of cells from heterogeneous samples. Using an electronic chip-based microfluidic cartridge and fluorescent image-based analysis, the DEPArray isolates 100% pure tumor cells from FFPE samples or single circulating tumor cells.	MENARINI silicon biosystems

EXHIBITORS & SPONSORS

MAYO CLINIC Mayo Medical Laboratories	Mayo Medical Laboratories (MML) is a global reference laboratory operating within Mayo Clinic's Department of Laboratory Medicine and Pathology. Guided by expert pathologists, laboratory scientists, neurologists, and genetic counselors, MML offers cost-effective patient care-driven testing approaches for hundreds of neurological conditions.	Booth #7
Booth #8	Wolters Kluwer Health is a global provider of information, business intelligence and point-of-care solutions for the healthcare industry. Brands include Lippincott Williams & Wilkins, a leading international publisher of medical books, journals, and electronic media. Please visit our display to browse our comprehensive product line and visit us at our website www.wolterskluwerhealth.com.	Wolters Kluwer
autism BRAIN NET	Although there is substantial evidence from neuroimaging studies that the brain of a child with autism is undergoing abnormal development, little is known about the underlying cellular, molecular and genetic mechanisms that lead to the onset of autistic symptoms. **The only way to answer questions related to the fundamental genetic and neuropathological aspects of autism spectrum disorder is to study brain tissue from individuals with autism spectrum disorder.** The Autism BrainNet is a collaboration of 4 different research organizations to increase the number of brains available to researchers for study. Studies of postmortem brain tissue will lead the way to better prevention and treatment of autism spectrum and related neurodevelopmental disorders. To learn more go to www.autismbrainnet.org.	Booth #9
Booth #10	NACC maintains standardized longitudinal research data from NIA-funded Alzheimer's Disease Centers. Since 2005 there have been over 35,000 subjects enrolled, of which over 4,400 have NP data available. NACC provides an unparalleled research resource. **NACC data are freely available to all researchers**. National Alzheimer's Coordinating Center (NACC) www.alz.washington.edu; naccmail@uw.edu.	NACC National Alzheimer's Coordinating Center
Roche	The Roche Ventana portfolio embodies a proven history of innovation and delivery of leading-edge automated instruments and reagent systems for slide-based tissue diagnosis of cancer and infectious disease. Our comprehensive solution includes digital pathology, companion diagnostics, high-value assays, automated slide staining and state-of-the-art workflow solutions. The portfolio empowers your lab to deliver enhanced medical value by helping you improve laboratory workflow efficiency, ensure patient and user safety, and increase diagnostic accuracy.	Booth #11

SPONSORS

THERAPATH NEUROPATHOLOGY	Therapath Neuropathology was founded in 2004. We are a specialized group of board certified neuropathologists that perform professional and technical services for health care specialists in hospitals, private offices, and the pharmaceutical industry. Diagnostic services include epidermal nerve fiber density testing, sweat gland nerve fiber density testing, muscle biopsy analysis, and peripheral nerve biopsy analysis.	Meeting Registration Bag Sponsor

ALEX PLANK IS A FILMMAKER, ACTOR, FOUNDER OF WRONGPLANET.NET, AND A SUPERHERO. HE WAS DIAGNOSED WITH ASPERGER'S SYNDROME AT AGE 9.

IT TAKES BRAINS TO SOLVE AUTISM

Alex Plank never saw himself as super. More like "awesome" really. He feels that being on the autism spectrum is a gift, and he wants to share that gift with scientists so they can understand what makes his brain unique.

Many think Alex and others just like him are super heroes. Why?

They've all been united by one brave and heroic act… pledging to donate their brain tissue to science when they are, sadly, no longer with us. It's difficult to think about, but the reality is that brain tissue is urgently needed for the scientific research that will help thousands of people with autism.

So join Alex to help ensure a brighter future for all. Because it takes brains to solve autism.

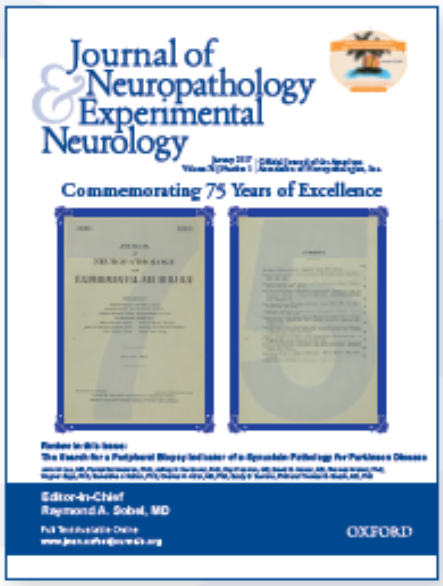
Visit **TakesBrains.org** to learn more.

it takes BRAINS …to solve autism

This initiative is guided by:

AUTISM SCIENCE FOUNDATION · AUTISM SPEAKS · SFARI Simons Foundation Autism Research Initiative · UC DAVIS MIND Institute · UT Southwestern Medical Center · Beth Israel Deaconess Medical Center · Icahn School of Medicine at Mount Sinai

Journal of Neuropathology & Experimental Neurology



Journal of Neuropathology & Experimental Neurology is the official journal of the American Association of Neuropathologists, Inc. (AANP). The journal publishes peer-reviewed studies on neuropathology and experimental neuroscience, book reviews, letters, and Association news, covering a broad spectrum of fields in basic neuroscience with an emphasis on human neurological diseases. It is written by and for neuropathologists, neurologists, neurosurgeons, pathologists, psychiatrists, and basic neuroscientists from around the world.

Why Publish in *JNEN*?

- No page charges for active AANP members
- Reduced Open Access fees for AANP members
- Rapid first decision and online publication
- Special features that highlight AANP members e.g. autobiographies
- Research articles are used as sources for AANP's self-assessment modules (SAMS)
- Free color

Celebrate *JNEN's* 75th anniversary of publication with a special collection curated by a number of senior AANP members.

Freely available here: bit.ly/JNEN_75

American Association of Neuropathologists

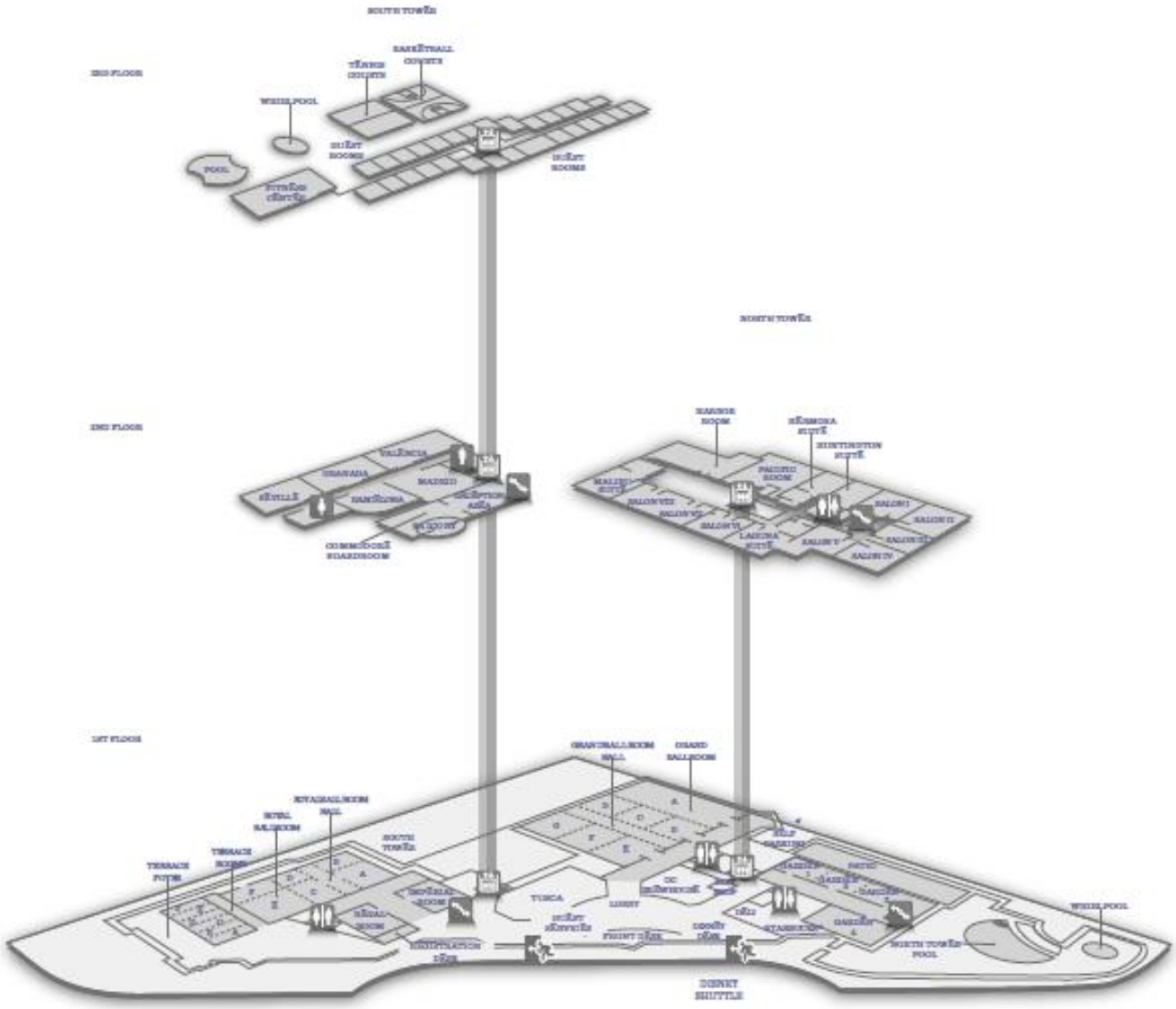
OXFORD UNIVERSITY PRESS

THE HYATT REGENCY FLOOR PLANS

HYATT REGENCY ORANGE COUNTY
11999 Harbor Boulevard
Garden Grove, CA 92840
USA

T +1 714 750 1234
F +1 714 740 0465
orangecounty.hyatt.com

3-D FLOOR PLAN



2017 MEETING AT A GLANCE

2017 SPECIAL COURSE

Genetics, Genomics, and Epigenomics in Clinical Neuropathology Practice and Understanding Autism

Director: Cheryl A. Palmer, MD

Thursday, June 8, 2017	
Time:	**Grand Ballroom ABCD**
7:00 am – 8:00 am	**CONTINENTAL BREAKFAST-** Grand Ballroom Foyer
8:00 am – 8:15 am	*Welcome and CME Pre-Test*
8:15 am – 9:00 am	*DNA Methylation-Based Classification of CNS Tumors* David Capper, MD Charité Universitätsmedizin Berlin, Berlin, Germany
9:00 am – 9:45 am	*Next Generation Sequencing: An Ancillary Diagnostic Tool for CNS Tumors* David A. Solomon, MD, PhD University of California San Francisco, San Francisco, CA
9:45 am – 10:30 am	*The 2016 CNS WHO Classification: One Year Later* David N. Louis, MD Massachusetts General Hospital, Boston, MA
10:30 am – 11:00 am	**REFRESHMENT BREAK -** Grand Ballroom Foyer
11:00 am – 11:45 am	*Metagenomic Sequencing for Diagnosis of Central Nervous System Infections* Steve Miller, MD, PhD University of California San Francisco, San Francisco, CA
11:45 am – 12:30 pm	*Neuromuscular Disease: Using Biopsy Pathology to Predict or Confirm Genetic Diagnoses* Steven A. Moore, MD, PhD University of Iowa, Iowa City, IA
12:30 pm – 1:30 pm	**LUNCH ON OWN**
1:30 pm – 2:15 pm	*Neuropathology of Autism* Manuel F. Casanova, MD University of South Carolina School of Medicine, Greenville, SC
2:15 pm – 3:00 pm	*Recent Progress in the Genomics of Autism Spectrum Disorders* Matthew W. State, MD, PhD University of California San Francisco, San Francisco, CA
3:00 pm – 3:30 pm	**REFRESHMENT BREAK -** Grand Ballroom Foyer
3:30 pm – 4:15 pm	*Cellular and Neural Networks in Autism* Matthew P. Anderson, MD, PhD Beth Israel Deaconess Medical Center, Boston, MA
4:15 pm – 5:00 pm	*Young Neuron Migration in the Human Brain: Insights into Neurodevelopmental Diseases* Eric J. Huang, MD, PhD University of California San Francisco, San Francisco, CA
5:00 pm – 5:10 pm	*Closing Remarks and CME Post-Test*
5:30 pm – 7:30 pm	**ANNUAL RECEPTION – Moved from Friday to Thursday Night!** *All Attendees Welcome* **–** Pool, N. Tower

2017 MEETING AT A GLANCE

2017 ABSTRACTS AND NAMED LECTURES, DAY 1

Director: Arie Perry, MD

<table>
<tr><th colspan="3">Friday, June 9, 2017</th></tr>
<tr><td>7:00 am – 8:00 am</td><td colspan="2">CONTINENTAL BREAKFAST
Grand Ballroom Foyer</td><td>Garden 2 & 3, N. Tower</td></tr>
<tr><td></td><td>Grand Ballroom ABCD</td><td>Grand Ballroom EFG</td><td rowspan="12">Posters #33-109
Friday, June 9
8:00 am – 5:00 pm</td></tr>
<tr><td>8:00 am – 10:00 am</td><td>PLATFORM 1
Neurodegenerative: Alzheimer</td><td>PLATFORM 2
Tumors: Adult</td></tr>
<tr><td>10:00 am – 10:30 am</td><td colspan="2">REFRESHMENT BREAK
Grand Ballroom Foyer</td></tr>
<tr><td></td><td colspan="2">Grand Ballroom ABCD</td></tr>
<tr><td>10:30 am – 11:30 am</td><td colspan="2">PARISI LECTURE
Autoimmune Gliopathies: A Journey of Discovery

Sean J. Pittock, MD
Mayo Clinic, Rochester, MN</td></tr>
<tr><td>11:30 am – 11:45 am</td><td colspan="2">AWARD FOR MERITORIOUS CONTRIBUTIONS TO NEUROPATHOLOGY
Honoring Ronald C. Kim, MD
Presented by Joseph E. Parisi, MD</td></tr>
<tr><td>11:45 am – 12:45 pm</td><td colspan="2">BUSINESS MEETING I – All Members Welcome</td></tr>
<tr><td>12:45 pm – 2:00 pm</td><td colspan="2">LUNCH ON OWN</td></tr>
<tr><td></td><td>Grand Ballroom ABCD</td><td>Grand Ballroom EFG</td></tr>
<tr><td>2:00 pm – 4:00 pm</td><td>PLATFORM 3
Muscle, Nerve and Eye Pathology</td><td>PLATFORM 4
Tumors: Pediatric</td></tr>
<tr><td>4:00 pm – 4:45 pm</td><td colspan="2">POSTER VIEWING & REFRESHMENT BREAK
Garden 2 & 3, N. Tower; Grand Ballroom Foyer</td></tr>
<tr><td></td><td colspan="2">Grand Ballroom ABCD</td></tr>
<tr><td>4:45 pm – 5:45 pm</td><td colspan="2">DEARMOND LECTURE
Structure, Function, Connectivity, Development and Evolution of Human Cerebral Cortex

David C. Van Essen, PhD
Washington University in St. Louis, St. Louis, MO</td></tr>
</table>

2017 MEETING AT A GLANCE

2017 ABSTRACTS AND NAMED LECTURES, DAY 2

Director: Arie Perry, MD

<table>
<tr><th colspan="3">Saturday, June 10, 2017</th></tr>
<tr><td>7:00 am – 8:00 am</td><td colspan="2">CONTINENTAL BREAKFAST
Grand Ballroom Foyer</td><td>Garden 2 & 3, N. Tower</td></tr>
<tr><td></td><td>Grand Ballroom ABCD</td><td>Grand Ballroom EFG</td><td rowspan="16">Posters #142-219
Saturday, June 10
8:00 am – 5:00 pm</td></tr>
<tr><td>8:00 am – 10:00 am</td><td>PLATFORM 5
Neurodegenerative: FTD/Lewy body/ Parkinson/Vascular/Prion</td><td>PLATFORM 6
Developmental/ Pediatric</td></tr>
<tr><td>10:00 am – 10:30 am</td><td colspan="2">REFRESHMENT BREAK
Grand Ballroom Foyer</td></tr>
<tr><td></td><td colspan="2">Grand Ballroom ABCD</td></tr>
<tr><td>10:30 am – 11:00 am</td><td colspan="2">What Every Neuropathologist Needs to Know:
The Updated WHO Classification and Pediatric Neuro-Oncology
David W. Ellison, MD, PhD
St. Jude Children's Research Hospital, Memphis, TN</td></tr>
<tr><td>11:00 am – 11:15 am</td><td colspan="2">AWARD FOR MERITORIOUS CONTRIBUTIONS TO NEUROPATHOLOGY
Honoring Harry V. Vinters, MD
Presented by William H. Yong, MD</td></tr>
<tr><td>11:15 am – 12:15 pm</td><td colspan="2">BUSINESS MEETING II – All Members Welcome</td></tr>
<tr><td>12:15 pm – 1:30 pm</td><td colspan="2">LUNCH ON OWN</td></tr>
<tr><td></td><td>Grand Ballroom ABCD</td><td>Grand Ballroom EFG</td></tr>
<tr><td>1:30 pm – 3:30 pm</td><td>PLATFORM 7
CTE/Pediatric Forensic/ Trauma</td><td>PLATFORM 8
Tumor Diagnostic Tools and Social Media</td></tr>
<tr><td>3:30 pm – 4:15 pm</td><td colspan="2">POSTER VIEWING & REFRESHMENT BREAK
Garden 2 & 3, N. Tower; Grand Ballroom Foyer</td></tr>
<tr><td></td><td colspan="2">Grand Ballroom ABCD</td></tr>
<tr><td>4:15 pm – 4:45 pm</td><td colspan="2">What Every Neuropathologist Needs to Know:
Basic Electron Microscopy of Muscle
Robert E. Mrak, MD, PhD
University of Toledo College of Medicine, Toledo, OH</td></tr>
<tr><td>4:45 pm – 5:45 pm</td><td colspan="2">KOREY LECTURE
Disease Modifying Therapeutical Approaches for Synucleinopathies of the Aging Population
Eliezer Masliah, MD
National Institute on Aging, Bethesda, MD</td></tr>
<tr><td></td><td colspan="2">Grand Ballroom ABCD</td></tr>
<tr><td>8:00 pm – 11:00 pm</td><td colspan="2">DIAGNOSTIC SLIDE SESSION
10 Cases Moderated by Caterina Giannini, MD, PhD and Rebecca D. Folkerth, MD</td></tr>
</table>

2017 MEETING AT A GLANCE

2017 PRESIDENTIAL SYMPOSIUM

Recent Advances and Reviews in the Field of Neuropathology

Director: Arie Perry, MD

Sunday, June 11, 2017	
Time:	**Grand Ballroom ABCD**
7:00 am – 8:00 am	**CONTINENTAL BREAKFAST -** Grand Ballroom Foyer
8:00 am – 8:05 am	*Welcome and CME Pre-Test* Arie Perry, MD University of California San Francisco, San Francisco, CA
8:05 am – 9:00 am	**MOORE LECTURE** *An Update of the WHO Classifications of Tumors of the Pituitary Gland, 4th Edition* M. Beatriz S. Lopes, MD, PhD University of Virginia School of Medicine, Charlottesville, VA
9:00 am – 9:45 am	*Zika Virus Encephalomyelopathy* Leila Chimelli, MD, PhD State Institute of Brain and Federal University of Rio de Janeiro, Rio de Janeiro, Brazil
9:45 am – 10:15 am	**AANP AWARDS PRESENTATIONS**
10:15 am – 10:30 am	**REFRESHMENT BREAK -** Grand Ballroom Foyer
10:30 am – 11:15 am	*Perinatal Neuropathology – Current Concepts* Marc R. Del Bigio, MD, PhD, FRCPC University of Manitoba, Winnipeg, MB, Canada
11:15 am – 12:00 pm	*Innovative Approaches to Neuropathology Teaching - Panel Discussion: Music, Social Media and Online Resources* Arie Perry, MD University of California San Francisco, San Francisco, CA Brian Moore, MD, MEd University of Colorado School of Medicine, Aurora, CO Cheryl A. Palmer, MD University of Utah, Salt Lake City, UT
12:00 pm – 12:15 pm	**INSTALLATION OF NEW OFFICERS AND ADJOURNMENT**

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Special Course

Thursday, June 8, 2017

Learning Objectives:

1. *Describe new genomic information in the classification of tumors of the central nervous system.*
2. *Summarize new developments in genomic testing.*
3. *Explain exome and genome sequencing strategies.*
4. *Cite advances in understanding autism spectrum disorder.*

SPECIAL COURSE

DNA Methylation-Based Classification of CNS Tumors

Time: 8:15 am – 9:00 am

David Capper, MD, *Professor for Molecular Neuropathology, Department of Neuropathology, Charité Universitätsmedizin Berlin*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Determine if a given case fulfills the material requirements for methylation analysis.
2. Delineate the basic output data of the methylation classifier.
3. Summarize the additional output information of a methylation analysis and identify cases for which this additional information is of value.

II. Abstract & Relevant References

Modern neuropathology is challenged by an increasing number of clinically-relevant central nervous system (CNS) tumor subgroups that in addition to histopathological analysis currently require assessment of a multitude of molecular markers for classification. Inter-observer variability leads to tumor misclassification, which can have severe consequences for affected patients. We compiled a cohort of genome-wide DNA methylation profiles of 2,682 tumors from 82 histologically and/or molecularly distinct CNS tumor classes across all ages and histologies that served as reference for a Random Forest-based diagnostic classifier. This classifier was used to prospectively investigate 1,104 CNS tumor samples in order to determine its clinical utility. In addition to classification, the array data is used to generate copy number profiles and MGMT promoter methylation status. Reproducibility for different technical platforms and between clinical centers was assessed. The classifier was able to reliably assign CNS tumor samples to a given diagnostic category with a misclassification rate of less than 2%. The system functioned robustly across laboratories and regardless of the method of generating DNA methylation profiles. Prospective application to clinical samples resulted in a reclassification of 12% of tumors. The reference cohort and Random Forest-based classifier are available online as a valuable community tool for improving precision in brain tumor diagnostics.

References:

1. Hovestadt V, Remke M, Kool M, et al. Robust molecular subgrouping and copy-number profiling of medulloblastoma from small amounts of archival tumour material using high-density DNA methylation arrays. Acta neuropathologica 2013;125:913-6.
2. Sturm D, Orr BA, Toprak UH, et al. New Brain Tumor Entities Emerge from Molecular Classification of CNS-PNETs. Cell 2016;164:1060-72
3. Sturm D, Witt H, Hovestadt V, et al. Hotspot mutations in H3F3A and IDH1 define distinct epigenetic and biological subgroups of glioblastoma. Cancer cell 2012;22:425-37.
4. Pajtler KW, Witt H, Sill M, et al. Molecular Classification of Ependymal Tumors across All CNS Compartments, Histopathological Grades, and Age Groups. Cancer cell 2015;27:728-43.
5. Bady P, Delorenzi M, Hegi ME. Sensitivity Analysis of the MGMT-STP27 Model and Impact of Genetic and Epigenetic Context to Predict the MGMT Methylation Status in Gliomas and Other Tumors. J Mol Diagn 2016;18:350-61.

III. Faculty Biography

Dr. med David Capper has been recently appointed Professor for Molecular Neuropathology at the Charité Universitätsmedizin Berlin and joined the Berlin German Cancer consortium (DKTK) Faculty. Previously, he worked as senior physician of Neuropathology at the University Hospital Heidelberg and was member of the Clinical Cooperation Unit Neuropathology at the German Cancer Research Center (DKFZ). His research focuses on the pathology and genetics of tumors of the central nervous system with a special focus on the development of specific diagnostic markers ("mutation-specific monoclonal antibodies") and classification of brain tumors by genome-wide analysis of DNA methylation patterns. In addition, he headed the central pathology for several national and international studies (especially INFORM register study MNP2.0 study).

SPECIAL COURSE

Next Generation Sequencing: An Ancillary Diagnostic Tool for CNS Tumors

Time: 9:00 am – 9:45 am

David A. Solomon, MD, PhD, *Assistant Professor, Division of Neuropathology and Clinical Cancer Genomics Laboratory, University of California San Francisco, San Francisco, CA*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Describe the basic principles of next-generation DNA sequencing.
2. Outline the role of NGS as an ancillary diagnostic tool in surgical neuropathology.
3. Recognize the benefits and limitations of amplicon-based versus capture-based NGS, as well as tumor-only versus paired tumor-normal NGS analysis.
4. Interpret the results of an NGS report and use the findings to formulate a molecularly-integrated pathologic diagnosis for CNS tumor specimens.

II. Abstract & Relevant References

Next-generation sequencing (NGS) refers to any number of recently developed technologies that can perform rapid sequencing analysis of individual DNA molecules, often on a massive scale. These technologies now provide the capability of sequencing the whole genome of an individual human, the entire coding exome of a tumor, or the entire transcriptome of specific tissues at an affordable cost and in a manner of hours to days. The advent of NGS technology has the potential to profoundly impact diagnostic neuropathology and direct personalized treatment for neuro-oncology patients. NGS of genomic DNA isolated from tumor tissue can be performed to assay for the complete spectrum of genetic alterations known to occur in brain tumors including single nucleotide variants, small insertions/deletions, amplifications, deletions, gene fusions, and other structural variants. Methods have also been developed to assess for genome-wide chromosomal copy number alterations using NGS data. NGS platforms are currently being implemented at medical centers around the world for clinical use and have already been shown to help clarify the pathologic diagnosis, detect tumor-predisposing germline alterations, and identify potentially targetable genetic alterations in neuro-oncology patients. In this lecture, I will review the principles of NGS, discuss the different methodologies being employed, and highlight its capabilities and limitations as an ancillary diagnostic tool for CNS tumor specimens.

References:

1. Kline CN, Joseph NM, Grenert JP, van Ziffle J, Talevich E, Onodera C, Aboian M, Cha S, Raleigh DR, Braunstein S, Torkildson J, Samuel D, Bloomer M, Campomanes AG, Banerjee A, Butowski N, Raffel C, Tihan T, Bollen AW, Phillips JJ, Korn WM, Yeh I, Bastian BC, Gupta N, Mueller S, Perry A, Nicolaides T, Solomon DA. Targeted next-generation sequencing of pediatric neuro-oncology patients improves diagnosis, identifies pathogenic germline mutations, and directs targeted therapy. Neuro Oncol. 2016 Nov 14 [Epub ahead of print]
2. Ramkissoon SH, Bandopadhayay P, Hwang J, Ramkissoon LA, Greenwald NF, Schumacher SE, O'Rourke R, Pinches N, Ho P, Malkin H, Sinai C, Filbin M, Plant A, Bi WL, Chang MS, Yang E, Wright KD, Manley PE, Ducar M, Alexandrescu S, Lidov H, Delalle I, Goumnerova LC, Church AJ, Janeway KA, Harris MH, MacConaill LE, Folkerth RD, Lindeman NI, Stiles CD, Kieran MW, Ligon AH, Santagata S, Dubuc AM, Chi SN, Beroukhim R, Ligon KL. Clinical targeted exome-based sequencing in combination with genome-wide copy number profiling: precision medicine analysis of 203 pediatric brain tumors. Neuro Oncol. 2017 Jan 19 [Epub ahead of print]

3. Sahm F, Schrimpf D, Jones DT, Meyer J, Kratz A, Reuss D, Capper D, Koelsche C, Korshunov A, Wiestler B, Buchhalter I, Milde T, Selt F, Sturm D, Kool M, Hummel M, Bewerunge-Hudler M, Mawrin C, Schüller U, Jungk C, Wick A, Witt O, Platten M, Herold-Mende C, Unterberg A, Pfister SM, Wick W, von Deimling A. Next-generation sequencing in routine brain tumor diagnostics enables an integrated diagnosis and identifies actionable targets. Acta Neuropathol. 2016; 131: 903-910.

III. Faculty Biography

David Solomon is a neuropathologist and cancer researcher at the University of California, San Francisco with a dedicated interest in the genetic alterations that drive cancer development. He completed his MD/PhD training at Georgetown University School of Medicine, where his thesis research was focused on identifying novel transforming pathways in glioblastoma. He then completed an Anatomic Pathology Residency and Neuropathology Fellowship at UCSF, where he has subsequently joined the faculty in the Department of Pathology in July 2016. His clinical interests focus on diagnostic neuropathology, particularly CNS tumors. He worked with the UCSF Clinical Cancer Genomics Laboratory to develop a capture-based NGS test (the UCSF500 Cancer Panel) that was launched for patient care in early 2015 and has been revolutionizing the diagnosis and treatment of brain tumors since its implementation. Dr. Solomon's ongoing research efforts include genomic analysis of rare brain tumor variants including choroid plexus tumors, pineal parenchymal tumors, and chordoid gliomas. His prior work led to the discovery of frequent inactivating mutations of the cohesin complex gene STAG2 in glioblastoma, urothelial carcinoma, and Ewing sarcoma that define molecular subtypes of these tumors with distinct clinical outcomes. Current research in his laboratory is focused on understanding the function of the cohesin complex during tumorigenesis and developing novel targeted therapies for the many cancers harboring cohesin gene mutations. Dr. Solomon is funded by the NIH Director's Early Independence Award and the UCSF Physician-Scientist Scholar Program.

SPECIAL COURSE

The 2016 CNS WHO Classification: One Year Later

Time: 9:45 am – 10:30 am

David N. Louis, MD, *Pathologist-in-Chief, Massachusetts General Hospital, Benjamin Castleman Professor of Pathology, Harvard Medical School*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Evaluate strategies for practical implementation of the 2016 WHO CNS tumor classification.
2. Summarize some practical effects of the 2016 WHO CNS tumor classification.

II. Abstract & Relevant References

The 2016 World Health Organization Classification of Tumors of the Central Nervous System has been both a conceptual and practical advance over its 2007 predecessor. For the first time, the WHO classification of CNS tumors used molecular parameters in addition to histology to define many tumor entities, thus formulating a concept for how CNS tumor diagnoses should be structured in the molecular era. For example, the 2016 CNS WHO restructured the diffuse gliomas, medulloblastomas and other embryonal tumors in major ways, and incorporated new entities that are defined by both histology and molecular features. Since its publication a year ago, these changes have resulted in practical challenges for neuropathologists, which have included: specific diagnostic challenges. e.g., relating to the diffuse gliomas; the use of layered reports and of standardized reporting structures; the time required to reach an integrated diagnosis; the availability of molecular testing technologies and the use of surrogate markers; the process of keeping the WHO classification both current and practical for all users. These practical challenges and ongoing developments to address these challenges will be discussed.

References:

1. Louis DN. Perry A, Burger P, Ellison DW, Reifenberger G, von Deimling A, Aldape K, Brat D, Collins VP, Eberhart C, Figarella-Branger D, Fuller GN, Giangaspero F, Giannini C, Hawkins C, Kleihues P, Korshunov A, Kros JM, Lopes MB, Ng HK, Ohgaki H, Paulus W, Pietsch T, Rosenblum M, Rushing E, Soylemezoglu F, Wiestler O, Wesseling P. International Society of Neuropathology-Haarlem consensus guidelines for nervous system tumor classification and grading. Brain Pathol 24:429-435, 2014.
2. Louis DN, Ohgaki H, Wiestler OD, Cavenee WK (eds.). World Health Organization Histological Classification of Tumours of the Central Nervous System. Lyon: International Agency for Research on Cancer, 2016.
3. Louis DN, Perry A, Reifenberger G, von Deimling A, Figarella-Branger D, Cavenee WK, Ohgaki H, Wiestler OD, Kleihues P, Ellison DW. The 2016 WHO classification of tumours of the central nervous system: a summary. Acta Neuropathol 131:803-820, 2016.
4. Reifenberger G, Wirsching HG, Knobbe-Thomsen CB, Weller M. Advances in the molecular genetics of gliomas - implications for classification and therapy. Nat Rev Clin Oncol. 2016
5. Ellis DW, Srigley J. Does standardised structured reporting contribute to quality in diagnostic pathology? The importance of evidence-based datasets. Virchows Arch. 468:51-59, 2016.
6. Louis, DN, Aldape K, Brat DJ, Capper D, Ellison DW, Hawkins C, Paulus W, Perry A, Reifenberger G, Figarella-Branger D, Wesseling P, Batchelor TT, Cairncross JG, Pfister SM, Rutkowski, Weller M, Wick W, von Deimling A. Announcing cIMPACT-NOW: The Consortium to Inform Molecular and Practical Approaches to CNS Tumor Taxonomy. Acta Neuropathol 2017 (in press)

7. Louis, DN, Aldape K, Brat DJ, Capper D, Ellison DW, Hawkins C, Paulus W, Perry A, Reifenberger G, Figarella-Branger D, Wesseling P, Batchelor TT, Cairncross JG, Pfister SM, Rutkowski, Weller M, Wick W, von Deimling A. cIMPACT-NOW (the Consortium to Inform Molecular and Practical Approaches to CNS Tumor Taxonomy): a new initiative in advancing nervous system tumor classification. Brain Pathol 2017 (in press)
8. Paulus W. WHO 2016: Open questions and practical implications. Acta Neurochir (Wien). 159:419-422, 2017.

III. Faculty Biography

David N. Louis, MD, is the Benjamin Castleman Professor of Pathology at Harvard Medical School and pathologist-in-chief at Massachusetts General Hospital. Pathology at MGH has nearly 100 faculty members, over 100 trainees and over 700 employees, and performs about 12 million laboratory tests as well as 80,000 surgical pathology evaluations, 400,000 microbiology analyses, and 50,000 cytologies each year. Dr. Louis' own clinical neuropathology practice and research focuses on brain tumors, with an emphasis on the molecular basis of malignant gliomas and the application of molecular diagnostics to glioma classification. He has published close to 300 original articles, as well as numerous reviews, chapters and books. His laboratory was the first to demonstrate that molecular approaches could be used to subdivide malignant gliomas in a biologically relevant manner, and that molecular approaches could be used to predict the response of particular malignant gliomas to specific therapies. This work has contributed to worldwide adoption of molecular testing for the management of patients with these tumors. Dr. Louis has received a number of prestigious awards for his work in brain tumors and was lead editor of the 2007 and 2016 World Health Organization Classification of Central Nervous System Tumors. Dr. Louis served as President of the American Association of Neuropathologists in 2009-10, gave the Saul Korey Lecture at the 2008 meeting and the Matthew Moore Lecture at the 2014 meeting, has given multiple other talks at AANP meetings, and has participated in many AANP committees.

SPECIAL COURSE

Metagenomic Sequencing for Diagnosis of Central Nervous System Infections

Time: 11:00 am – 11:45 am

Steve Miller, MD, PhD, *Department of Laboratory Medicine, University of California San Francisco, San Francisco, CA*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Explain the clinical approach to molecular diagnostics in CSF.
2. Describe the complexities of metagenomics sequencing analysis and interpretation.
3. Correlate the findings of metagenomics sequencing to clinical presentation and diagnosis.

II. Abstract & Relevant References

Metagenomic next-generation sequencing (mNGS) for pathogen detection allows for unbiased identification of infectious agent nucleic acid in clinical samples. We have implemented this assay in the UCSF clinical laboratory for diagnosis of meningitis / encephalitis using optimized library preparation and bioinformatics processing steps, with case discussion and decision support through the Clinical Microbial Sequencing Board. This talk will outline the assay development and performance, and highlight cases showing the diagnostic utility of mNGS for infectious disease.

References:

1. Wilson MR, Naccache SN, Samayoa E, Biagtan M, Bashir H, Yu G, Salamat SM, Somasekar S, Federman S, Miller S, Sokolic R, Garabedian E, Candotti F, Buckley RH, Reed KD, Meyer TL, Seroogy CM, Galloway R, Henderson SL, Gern JE, DeRisi JL and Chiu CY. Actionable diagnosis of neuroleptospirosis by next-generation sequencing. The New England Journal of Medicine 2014;370:2408-2417.
2. Naccache SN, Federman S, Veeraraghavan N, Zaharia M, Lee D, Samayoa E, Bouquet J, Greninger AL, Luk K-C, Enge B, Wadford DA, Messenger SL, Genrich GL, Pellegrino K, Grard G, Leroy E, Schneider BS, Fair JN, Martinez MA, Isa P, Crump JA, DeRisi JL, Sittler T, Hackett J, Miller S and Chiu CY. A cloud-compatible bioinformatics pipeline for ultrarapid pathogen identification from next-generation sequencing of clinical samples. Genome Research 2014;24:1180-1192.
3. Naccache SN, Peggs KS, Mattes FM, Phadke R, Garson JA, Grant P, Samayoa E, Federman S, Miller S, Lunn MP, Gant V, Chiu CY. Diagnosis of neuroinvasive astrovirus infection in an immunocompromised adult with encephalitis by unbiased next-generation sequencing. Clinical Infectious Disease 2015;60:919-923.
4. Yu G, Greninger AL, Isa P, Phan TG, Martinez MA, Sanchez ML, Contreras JF, Santos-Preciado JI, Parsonnett J, Miller S, DeRisi JL, Delwart E, Arias CF and Chiu CY. Discovery of a Novel Polyomavirus in Acute Diarrheal Samples from Children. PLoS One 2012;7:e49449.
5. Greninger AL, Chen EC, Sittler T, Schneierman A, Roubinian N, Yu G, Kim E, Pillai D, Guyard C, Mazzulli T, Isa P, Arias CF, Hackett J, Schochetman G, Miller S, Tang P and Chiu CY. A Metagenomic Analysis of Pandemic Influenza A (2009 H1N1) Infection in Patients from North America. PLoS One 2010;5:e13381.
6. Mongkolrattanothai K, Naccache S, Bender J, Samayoa E, Pham E, Yu G, Miller S, Aldrovandi G, Chiu C. Neurobrucellosis: Unexpected answer from metagenomic next-generation sequencing. Journal of the Pediatric Infectious Disease Society 2016;*in press*.

III. Faculty Biography

Steve Miller MD, PhD is Director of the Clinical Microbiology Laboratory at the University of California San Francisco and Associate Professor in the Department of Laboratory Medicine. Dr. Miller trained at the Albert Einstein College of Medicine in Bronx, New York. His research involves translation of novel molecular methods for diagnosis and monitoring of clinical infectious disease, with over 40 publications. Dr. Miller has received several grants to provide clinically actionable results from large sequence data sets, including the California Initiative to Advance Precision Medicine project for clinical implementation of metagenomic next-generation sequencing for precision diagnosis of acute infectious diseases.

SPECIAL COURSE

Neuromuscular Disease: Using Biopsy Pathology to Predict or Confirm Genetic Diagnoses

Time: 11:45 am – 12:30 pm

Steven A. Moore, MD, PhD, *Professor, The University of Iowa, Department of Pathology, Co-Director Iowa Wellstone Muscular Dystrophy Cooperative Research Center (MDCRC)*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Apply muscle biopsy evaluation to the prediction of genetic neuromuscular diseases.
2. Integrate histopathology and molecular genetics in the diagnosis of neuromuscular diseases.
3. Navigate online resources related to genetic neuromuscular disorders.

II. Abstract & Relevant References

Muscle biopsy evaluation has a long history of contributing information critically important for the diagnosis of neuromuscular disorders. Structural, ultrastructural, and histochemical pathology were initially used to classify disease. As the genetic basis for neuromuscular disorders unfolded, biopsy pathology took on the role of predicting the underlying mutant gene. The additional tool of immunohistochemistry is particularly helpful in this regard. Technological advances in molecular genetic evaluation have altered the landscape and pushed biopsy pathology into the role of confirming the pathogenesis of novel gene variants or variants of unknown significance in known genes. However, diagnostic approaches vary widely among clinicians who evaluate neuromuscular disease patients. In order to best serve patients and clinical colleagues, muscle pathologists today should rely on old-school acumen for the recognition of classic neuromuscular pathology and hone their ability to integrate molecular genetic data with currently available tissue diagnostic tools. A broad spectrum of genetic neuromuscular disease examples will be provided as a general guide to the present day practice of muscle pathology.

References and Web-Based Resources:

1. Gene Table of Neuromuscular Disorders. http://www.musclegenetable.fr
2. GeneReviews. https://www.ncbi.nlm.nih.gov/books/NBK1116/
3. Leiden Muscular Dystrophy Pages. http://www.dmd.nl
4. Online Mendelian Inheritance in Man. https://www.ncbi.nlm.nih.gov/omim
5. Monoclonal Antibodies for Muscular Dystrophy Research. http://www.glennmorris.org.uk/mabs/WCIND.htm
6. Developmental Studies Hybridoma Bank. http://dshb.biology.uiowa.edu

III. Faculty Biography

Dr. Moore is a Professor in the Department of Pathology at the University of Iowa. He is also Co-Director of the NIH-funded Wellstone Muscular Dystrophy Cooperative Research Center (MDCRC) at Iowa. Originally from Indiana where he obtained a bachelor's degree at Purdue University and both PhD and MD degrees at Indiana University Medical Center, he moved to Iowa in 1982 for a residency in anatomic pathology followed by a fellowship in neuropathology, both at the University of Iowa. Starting around 1997, Dr. Moore's clinical and research interests have increasingly focused on neuromuscular disorders. The Iowa Wellstone MDCRC was first funded in 2005. One component of the MDCRC is a biorepository of muscle biopsies and cultured fibroblasts. Clinical and basic science projects in the MDCRC focus on the dystroglycanopathies. Over the past few years, Iowa has become a central surgical biopsy site and muscle biopsy processing and storage site for clinical trials related to the treatment of Duchenne muscular dystrophy. Dr. Moore is actively involved in assay and protocol development related to the use of muscle biopsy endpoints for these clinical trials.

SPECIAL COURSE

Neuropathology of Autism

Time: 1:30 pm – 2:15 pm

Manuel F. Casanova, MD, *SmartState Endowed Chair for Childhood Neurotherapeutics, University of South Carolina*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Explain the role of neuropathology in the ongoing neurodiversity debate on autism.
2. Describe evidence indicating that autism spectrum disorder is a multifactorial or complex condition.
3. Outline evidence for abnormalities of cell migration in autism spectrum disorder.

II. Abstract & Relevant References

There is a growing populist movement (Neurodiversity) claiming that autism spectrum disorder (ASD) falls within the normal variability of human behavior. This movement decries the medical profession's attempts at research and treatment for this and other mental disorders. Opposing the Neurodiversity viewpoint is mounting evidence that ASD is the result of abnormally migrating postmitotic neuroblasts. This migratory defect affects the final cytoarchitecture and physiology of the cerebral cortex, brainstem and cerebellum. This lecture will emphasize the results of neuropathological studies that indicate a high prevalence of heterotopias and cortical malformations in this patient population. In addition, we will discuss recent neuroimaging findings that indicate a blurring of the gray/white matter junction in ASD individuals. Higher resolution studies suggest that this blurring is the result of cells migrating to the cerebral cortex that get stuck in the subplate region. Similarly, diffusion tensor imaging (DTI) of the cerebral cortex of ASD individuals has shown abnormalities in diffusivity that correspond to minicolumnar disorganization resulting from an improper alignment of neuroblasts migrating to the cerebral cortex. The findings discussed in this lecture strongly support ASD as a neurodevelopmental disorder. The large variety of mechanisms involved in cellular migration as well as the large time span wherein abnormalities in these mechanisms have been noted help explain some of the clinical heterogeneity observed in ASD.

References:

1. Reference #1 Casanova MF, Buxhoeveden D, Switala A, Roy E: Minicolumnar pathology in autism. Neurology, 58:428-432, 2002.
2. Reference #2 Casanova MF, Buxhoeveden D, Gomez J: Disruption in the inhibitory architecture of the cell minicolumn: implications for autism. The Neuroscientist, 9(6):496-507, 2003.
3. Reference #3 Casanova MF, van Kooten IAJ, Switala AE, van Engeland H, Heinsen H, Steinbusch HWM, Hof PR, Trippe J, Stone J, Schmitz C. Minicolumnar abnormalities in autism. Acta Neuropathologica 112(3):287-303, 2006.
4. Reference #4 Casanova MF. The neuropathology of autism. Brain Pathology 14(1): 101-118, 2008.
5. Reference #5 Casanova MF. The neuropathology of autism. In Fred Volkmar, Kevin Pelphrey, Rhea Paul, Sally Rogers (eds). Handbook of Autism and Pervasive Developmental Disorders 4th edition, ch. 21. Pp. 497-531, 2014.

III. Faculty Biography

Dr. Casanova belonged to the founding board of the National Alliance for Autism Research (now Autism Speaks) and the Autism Tissue Board. He has served on the Board of Directors or Scientific Advisory Board of numerous organizations (e.g., Autism Research Institute, Generation Rescue, On Mental Health, Families for Effective Autism Treatment, Clearly Present Foundation) and is presently on the editorial board of fifteen different medical journals. Among many honors Dr. Casanova served as Chairperson of the Developmental Brain Disorders Study Section, has received a Physicians Recognition Award by the American Medical Association, a National Research

Service Award by the Public Health Service, the Stanley Scholars title, a Distinguished Faculty Award by the Medical College of Georgia, Senior Scientist Award by the Winter Workshop on Schizophrenia, the title of Honorary Professor from the University of Krasnoyarsk, and a Contributing Piece Award by FEAT. In 2015 Dr. Casanova received the High-end Foreign Expert Award from the State Administration of Foreign Expert Affairs from Bejing Normal University in China. More recently, in 2016 Dr. Casanova was elected president of the International Consortium of Autism Institutes (ICAI). Dr. Casanova has provided a large number of plenary lectures including a magisterial presentation at the World Congress of Autism. His research has been recognized by a EUREKA award from the NIMH for the introduction of repetitive Transcranial Magnetic Stimulation (rTMS) in the Therapy of autism spectrum disorders. At present Dr. Casanova is the SmartState Endowed Chair for Childhood Neurotherapeutics at the University of South Carolina and the Greenville Health System.

SPECIAL COURSE

Recent Progress in the Genomics of Autism Spectrum Disorders

Time: 2:15 pm – 3:00 pm

Matthew State, MD, PhD, *Oberndorf Family Distinguished Professor in Psychiatry, Department of Psychiatry Chair, Director of the Langley Porter Psychiatric Institute and Hospital, Weill Institute for Neurosciences*, *University of California San Francisco, San Francisco, CA*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Explain how the psychiatric genetics field has transitioned from the identification of candidate loci to the discovery of bona fide risk genes
2. Identify at least one specific genes and genomic region that markedly increase the risk for Autism Spectrum Disorder.
3. Describe the differences in expected effect size between common transmitted and rare de novo risk variants.

II. Abstract & Relevant References

Recent advances in high throughput genomic technologies, coupled with large patient cohorts and an evolving culture of rapid data sharing have led to remarkable advances in the understanding of the genetics of autism spectrum disorders. To date, the lion's share of this progress has been with regard to the contribution of rare spontaneous mutations, both in DNA sequence and chromosomal structure. The ability now to reliably and systematically identify ASD risk genes provides important initial insights into both the opportunities as well as the challenges the field now faces in moving from gene discovery to an actionable understanding of the mechanisms underlying these complex common neurodevelopmental syndromes. The lecture will provide an overview of what is now known about the specific risk mutations associated with ASD, address the particular challenges posed by the discovery of mutations that have large biological effect but low population frequency, and consider the role that whole genome sequencing will play in the near future in enhancing the understanding of the developmental aspects of ASD risk.

References:

1. Reference #1: Sanders, S. J. *et al.* Insights into Autism Spectrum Disorder Genomic Architecture and Biology from 71 Risk Loci. *Neuron* **87**, 1215-1233, doi:10.1016/j.neuron.2015.09.016 (2015).
2. Reference #2: Sanders, S. J. *et al.* Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron* **70**, 863-885, doi:10.1016/j.neuron.2011.05.002 (2011).
3. Reference #3: Sanders, S. J. *et al.* De novo mutations revealed by whole-exome sequencing are strongly associated with autism. *Nature* **485**, 237-241, doi:10.1038/nature10945 (2012).
4. Reference #4: Iossifov, I. *et al.* The contribution of de novo coding mutations to autism spectrum disorder. *Nature* **515**, 216-221, doi:10.1038/nature13908 (2014)
5. Reference #5: Willsey AJ *et al*.: Coexpression networks implicate human midfetal deep cortical projection neurons in the pathogenesis of autism. *Cell* **155**:997-1007.

III. Faculty Biography

Matthew State MD, PhD is a child and adolescent psychiatrist and human geneticist who is currently the Oberndorf Family Distinguished Professor of Psychiatry, Chair of the Department of Psychiatry, Director of the Langley Porter Psychiatric Institute, and member of the Weill Institute for Neurosciences at the University of California San Francisco.

He received his MD from Stanford University, completed a residency in psychiatry and fellowship in child psychiatry at UCLA, and earned a PhD in genetics from Yale University, where he joined the faculty in 2001, until moving to UCSF in 2013. Over the past 15 years, his laboratory has played a leading role in elaborating the contribution of rare mutations to the etiology of autism spectrum and Tourette disorders. He has been the recipient of numerous awards, including the Tarjan Award from AACAP, the Ruane Prize from the Brain and Behavior Research Foundation, and was elected to membership in the National Academy of Medicine in 2013.

SPECIAL COURSE

Cellular and Neural Networks in Autism

Time: 3:30 pm – 4:15 pm

Matthew P. Anderson, MD, PhD, *Associate Professor, Harvard Medical School*; *Director of Neuropathology, Beth Israel Deaconess Medical Center*; *Faculty, Program in Neuroscience, Harvard Medical School*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Explain the core and comorbid behavioural and neurological symptoms found in autism spectrum disorder.
2. Describe how Cre-loxP conditional genetics and chemogenetics help to map the neuronal circuits underlying the behavioural problems in human neuropsychiatric genetic diseases.
3. Identify a neuronal circuit where at least one genetic ASD may give rise to autism's sociability deficits.
4. Describe where and how post-mortem brain cases of autism spectrum disorder are now being banked and made available to researchers.

II. Abstract & Relevant References

Diverse genetic defects, immunological insults, and certain epilepsies have been implicated in the pathophysiology of autism spectrum disorder (ASD). These varied aetiologies may converge to disrupt specific molecular interactions in specialized neuronal subtypes to produce autism's sociability deficits. Maternally inherited 15q11-13 chromosomal triplications cause a frequent and highly penetrant type of autism linked to increased gene dosages of UBE3A, which encodes a ubiquitin ligase with transcriptional co-regulatory functions. Here, using in vivo mouse genetics, we show that increasing UBE3A in the nucleus downregulates the glutamatergic synapse organizer Cbln1, which is needed for sociability in mice. Epileptic seizures also repress Cbln1 and are found to expose sociability impairments in mice with asymptomatic increases in UBE3A. This Ube3a–seizure synergy maps to glutamate neurons of the midbrain ventral tegmental area (VTA), where Cbln1 deletions impair sociability and weaken glutamatergic transmission. We provide preclinical evidence that viral-vector-based chemogenetic activation of, or restoration of Cbln1 in, VTA glutamatergic neurons reverses the sociability deficits induced by Ube3a and/or seizures. Our results suggest that gene and seizure interactions in VTA glutamatergic neurons impair sociability by downregulating Cbln1, a key node in the expanding protein interaction network of autism genes.

References:

1. Chen, J. A., Peñagarikano, O., Belgard, T. G., Swarup, V. & Geschwind, D. H. The emerging picture of autism spectrum disorder: genetics and pathology. *Annu. Rev. Pathol.* 10, 111–144 (2015).
2. El Achkar, C. M. & Spence, S. J. Clinical characteristics of children and young adults with co-occurring autism spectrum disorder and epilepsy. *Epilepsy Behav.* 47, 183–190 (2015).
3. Glessner, J. T. *et al.* Autism genome-wide copy number variation reveals ubiquitin and neuronal genes. *Nature* 459, 569–573 (2009).
4. Smith, S. E. *et al.* Increased gene dosage of *Ube3a* results in autism traits and decreased glutamate synaptic transmission in mice. *Sci. Transl. Med.* 3, 103ra97 (2011).
5. Jiang, Y. H. *et al.* Mutation of the Angelman ubiquitin ligase in mice causes increased cytoplasmic p53 and deficits of contextual learning and long-term potentiation. *Neuron* 21, 799–811 (1998).
6. Kaphzan, H. *et al.* Genetic reduction of the α1 subunit of Na/K-ATPase corrects multiple hippocampal phenotypes in Angelman syndrome. *Cell Reports* 4, 405–412 (2013).
7. Matsuura, T. *et al. De novo* truncating mutations in E6-AP ubiquitin-protein ligase gene (*UBE3A*) in Angelman syndrome. *Nat. Genet*. 15, 74–77 (1997).
8. Wallace, M. L., Burette, A. C., Weinberg, R. J. & Philpot, B. D. Maternal loss of *Ube3a* produces an excitatory/inhibitory imbalance through neuron type-specific synaptic defects. *Neuron* 74, 793–800 (2012).
9. Scheffner, M., Huibregtse, J. M., Vierstra, R. D. & Howley, P. M. The HPV-16 E6 and E6-AP complex functions as a ubiquitin-protein ligase in the ubiquitination of p53. *Cell* 75, 495–505 (1993).
10. Nawaz, Z. *et al.* The Angelman syndrome-associated protein, E6-AP, is a coactivator for the nuclear hormone receptor superfamily. *Mol. Cell. Biol*. 19, 1182–1189 (1999).
11. Scoles, H. A., Urraca, N., Chadwick, S. W., Reiter, L. T. & Lasalle, J. M. Increased copy number for methylated maternal 15q duplications leads to changes in gene and protein expression in human cortical samples. *Mol. Autism* 2, 19 (2011).

12. Basu, S. N., Kollu, R. & Banerjee-Basu, S. AutDB: a gene reference resource for autism research. *Nucleic Acids Res*. 37, D832–D836 (2009).
13. Matsuda, K. & Yuzaki, M. Cbln family proteins promote synapse formation by regulating distinct neurexin signaling pathways in various brain regions. *Eur. J. Neurosci*. 33, 1447–1461 (2011).
14. Wei, P. *et al.* The Cbln family of proteins interact with multiple signaling pathways. *J. Neurochem*. 121, 717–729 (2012).
15. Elegheert, J. *et al.* Structural basis for integration of GluD receptors within synaptic organizer complexes. *Science* 353, 295–299 (2016).
16. Matsuda, K. *et al.* Cbln1 is a ligand for an orphan glutamate receptor δ2, a bidirectional synapse organizer. *Science* 328, 363–368 (2010).
17. Uemura, T. *et al. Trans*-synaptic interaction of GluRδ2 and neurexin through Cbln1 mediates synapse formation in the cerebellum. *Cell* 141, 1068–1079 (2010).
18. Ryu, K., Yokoyama, M., Yamashita, M. & Hirano, T. Induction of excitatory and inhibitory presynaptic differentiation by GluD1. *Biochem. Biophys. Res. Commun*. 417, 157–161 (2012).
19. Hirai, H. *et al.* Cbln1 is essential for synaptic integrity and plasticity in the cerebellum. *Nat. Neurosci*. 8, 1534–1541 (2005).
20. Bao, D. *et al.* Cbln1 is essential for interaction-dependent secretion of Cbln3. *Mol. Cell. Biol*. 26, 9327–9337 (2006).
21. Grier, M. D., Carson, R. P. & Lagrange, A. H. Of mothers and myelin: Aberrant myelination phenotypes in mouse model of Angelman syndrome are dependent on maternal and dietary influences. *Behav. Brain Res*. 291, 260–267 (2015).
22. Yang, M., Silverman, J. L. & Crawley, J. N. Automated three-chambered social approach task for mice. *Curr. Protoc. Neurosci.* Chapter 8, Unit 8.26 (2011).
23. Yadav, R. *et al.* Deletion of glutamate delta-1 receptor in mouse leads to aberrant emotional and social behaviors. *PLoS One* 7, e32969 (2012).
24. Iijima, T., Emi, K. & Yuzaki, M. Activity-dependent repression of Cbln1 expression: mechanism for developmental and homeostatic regulation of synapses in the cerebellum. *J. Neurosci*. 29, 5425–5434 (2009).
25. Takechi, K., Suemaru, K., Kiyoi, T., Tanaka, A. & Araki, H. The α4β2 nicotinic acetylcholine receptor modulates autism-like behavioral and motor abnormalities in pentylenetetrazol-kindled mice. *Eur. J. Pharmacol*. 775, 57–66 (2016).
26. Taylor, S. R. *et al.* GABAergic and glutamatergic efferents of the mouse ventral tegmental area. *J. Comp. Neurol.* 522, 3308–3334 (2014).
27. Zhang, S. *et al.* Dopaminergic and glutamatergic microdomains in a subset of rodent mesoaccumbens axons. *Nat. Neurosci*. 18, 386–392 (2015).
28. Gunaydin, L. A. *et al.* Natural neural projection dynamics underlying social behavior. *Cell* 157, 1535–1551 (2014).
29. Yamaguchi, T., Qi, J., Wang, H. L., Zhang, S. & Morales, M. Glutamatergic and dopaminergic neurons in the mouse ventral tegmental area. *Eur. J. Neurosci*. 41, 760–772 (2015).
30. Greer, P. L. *et al.* The Angelman Syndrome protein Ube3A regulates synapse development by ubiquitinating Arc. *Cell* 140, 704–716 (2010).
31. Khan, O. Y. *et al.* Multifunction steroid receptor coactivator, E6-associated protein, is involved in development of the prostate gland. *Mol. Endocrinol*. 20, 544–559 (2006).
32. Kühnle, S., Mothes, B., Matentzoglu, K & Scheffner, M. Role of the ubiquitin ligase E6AP/UBE3A in controlling levels of the synaptic protein Arc. *Proc. Natl Acad. Sci. USA* 110, 8888–8893 (2013).
33. Kim, S., Chahrour, M., Ben-Shachar, S. & Lim, J. Ube3a/E6AP is involved in a subset of MeCP2 functions. *Biochem. Biophys. Res. Commun*. 437, 67–73 (2013).
34. Lyst, M. J. *et al.* Rett syndrome mutations abolish the interaction of MeCP2 with the NCoR/SMRT co-repressor. *Nat. Neurosci*. 16, 898–902 (2013).
35. McClelland, S. *et al.* The transcription factor NRSF contributes to epileptogenesis by selective repression of a subset of target genes. *eLife* 3, e01267 (2014).
36. Liu, M. *et al.* Neuronal conditional knockout of NRSF decreases vulnerability to seizures induced by pentylenetetrazol in mice. *Acta Biochim. Biophys. Sin. (Shanghai)* 44, 476–482 (2012).
37. Kent, W. J. *et al.* The human genome browser at UCSC. *Genome Res/* 12, 996–1006 (2002).
38. Yi, J. J. *et al.* An autism-linked mutation disables phosphorylation control of UBE3A. *Cell* 162, 795–807 (2015).
39. Ronchi, V. P., Klein, J. M., Edwards, D. J. & Haas, A. L. The active form of E6-associated protein (E6AP)/UBE3A ubiquitin ligase is an oligomer. *J. Biol. Chem.* 289, 1033–1048 (2014).
40. Gossan, N. C. *et al.* The E3 ubiquitin ligase UBE3A is an integral component of the molecular circadian clock through regulating the BMAL1 transcription factor. *Nucleic Acids Res*. 42, 5765–5775 (2014).
41. Berrios, J. *et al.* Loss of UBE3A from TH-expressing neurons suppresses GABA co-release and enhances VTA-NAc optical self-stimulation. *Nat. Commun*. 7, 10702 (2016).
42. Riday, T. T. *et al.* Pathway-specific dopaminergic deficits in a mouse model of Angelman syndrome. *J. Clin. Invest.* 122, 4544–4554 (2012).

III. Faculty Biography

Matthew P. Anderson, M.D., Ph.D. is Associate Professor in Neurology and Pathology, Harvard Medical School; Faculty, Harvard Medical School Neuroscience PhD Program and Boston Children's Hospital IDDRC; Director of Neuropathology, Beth Israel Deaconess Medical Center; Director (Boston Node) and Neuropathologist of Autism BrainNET and Morphometric Core of The Center for SUDEP Research (CSR). He completed M.D. and Ph.D. degrees

at the University of Iowa where he uncovered the ion channel and regulatory functions of the cystic fibrosis gene training in epithelial ion transport, ion channel structure-function and regulation using site-directed mutagenesis and viral vectors, and human genetics. He completed anatomic pathology training and then trained in neuropathology in the Harvard Longwood Neuropathology Training Program (Brigham and Women's and Boston Children's Hospitals). He then did research at the Brain and Cognitive Sciences Department and Picower Institute of Learning and Memory at MIT with Nobel Laureate Susumu Tonegawa learning Cre-LoxP conditional mouse genetics, brain slice and *in vivo* (Matt Wilson, MIT) electrophysiology, and behavioral neurosciences. In his own laboratory at Harvard Medical School, they identified the first human genetic epilepsy disorder due to defective postnatal developmental pruning and maturation of glutamatergic circuits (Zhou et al. Nature Medicine 2009). They then developed mouse models of human genetic autism spectrum disorder reporting that increased Ube3a gene dosages as found in human maternal 15q11-13 duplications underlie the autism-related behavioral deficits and identified deficits in glutamatergic synapses in cortex (Smith et al. Sci. Transl. Med. 2011). More recently, they localized the sociability deficits arising from increased Ube3a gene dosage and from seizures to the midbrain ventral tegmental nucleus of the midbrain (Krishnan et al. Nature 2017). Here Ube3a and seizures synergize to repress expression of synapse organizing gene Cbln1 in glutamatergic neurons causing an impairment of glutamatergic synaptic transmission that impaired sociability. They have also performed electrophysiology studies of hypothalamic circuits as equally contributing first and senior authorships to a study establishing postnatal embryonic cell transplantation into hypothalamus improves obesity/diabetes by reconstituting complex circuitries to rescues leptin receptor deficiency (Czupryn et al. Science 2011).

SPECIAL COURSE

Young Neuron Migration in Human Brain: Insights into Neurodevelopmental Diseases

Time: 4:15 pm – 5:00 pm

Eric J. Huang, MD, PhD, *Professor of Pathology, Department of Pathology, University of California San Francisco, San Francisco, CA*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Explain the structural bases of human brain development from the 2nd trimester to early postnatal ages.
2. Describe the origins of GABAergic interneurons, and the timing of their migration and integration into the neural circuits in the frontal lobe.
3. Summarize how mutations in Coffin-Siris syndrome affect young neuron migration in human brain development.

II. Abstract & Relevant References

The first few months after birth are critical to human brain development. As a child begins to interact with the environment, the brain's frontal lobe, important for cognition, social behavior and executive function, increases in size and complexity. In a series of studies, we describe a collection of neurons that migrate and integrate widely into the frontal lobe during infancy. Chains of young neurons move tangentially close to the walls of the lateral ventricles and along blood vessels. They then individually disperse long distances to reach cortical tissue where they differentiate and contribute to inhibitory circuits. Our more recent work identifies the origin of these interneurons and determines how mutations in autism genes may affect the prenatal migration and/or differentiation of these young neurons, and contribute to the pathogenesis of neurodevelopmental disorders.

References:

1. Sanai N, Nguyen T, Ihrie RA, Mirzadeh Z, Tsai HH, Wong M, Gupta N, Berger MS, **Huang E**, Garcia-Verdugo JM, Rowitch DH, Alvarez-Buylla A. Corridors of migrating neurons in the human brain and their decline during infancy. Nature. 2011 Sep 28;478(7369):382-6.
2. Kwan KY, Lam MM, Johnson MB, Dube U, Shim S, Rašin MR, Sousa AM, Fertuzinhos S, Chen JG, Arellano JI, Chan DW, Pletikos M, Vasung L, Rowitch DH, **Huang EJ**, Schwartz ML, Willemsen R, Oostra BA, Rakic P, Heffer M, Kostović I, Judaš M, Sestan N. Species-dependent posttranscriptional regulation of NOS1 by FMRP in the developing cerebral cortex. Cell. 2012 May 11;149(4):899-911.
3. Yang D, Baumann JM, Sun YY, Tang M, Dunn RS, Akeson AL, Kernie SG, Kallapur S, Lindquist DM, **Huang EJ**, Potter SS, Liang HC, Kuan CY. Overexpression of vascular endothelial growth factor in the germinal matrix induces neurovascular proteases and intraventricular hemorrhage. Sci Transl Med. 2013 Jul 10;5(193):193ra90.
4. Fancy SP, Harrington EP, Baranzini SE, Silbereis JC, Shiow LR, Yuen TJ, **Huang EJ**, Lomvardas S, Rowitch DH. Parallel states of pathological Wnt signaling in neonatal brain injury and colon cancer. Nat Neurosci. 2014 Apr;17(4):506-12.
5. Paredes MF, James D, Gil-Perotin S, Kim H, Cotter JA, Ng C, Sandoval K, Rowitch DH, Xu D, McQuillen PS, Garcia-Verdugo JM, **Huang EJ**, Alvarez-Buylla A. Extensive migration of young neurons into the infant human frontal lobe. Science. 2016 Oct 7;354(6308). pii: aaf7073.

III. Faculty Biography

Dr. Eric Huang received his MD from the National Taiwan University School of Medicine in 1986, and PhD in Molecular Biology from Cornell University/Sloan Kettering Institute in New York City in 1993. He was trained in the combined Anatomic Pathology/Neuropathology (AP/NP) program in the Department of Pathology at UCSF from 1993 to 1997. Following his residency/fellowship, Dr. Huang pursued postdoctoral training in Developmental Neurobiology with Dr. Louis Reichardt at the Howard Hughes Medical Institute at UCSF from 1997 to 2000. In 2000, he was recruited to a joint position in the Department of Pathology at UCSF and the San Francisco VA Medical Center. He is currently a Professor of Pathology at UCSF.

Dr. Huang is the recipient of the following awards/Honors: **1997-99**, Postdoctoral Research Fellowship for Physicians (Howard Hughes Medical Institute); **1998**, Weil Award for the best paper in Experimental Neuropathology (AANP Annual Meeting); **2000-05**, Presidential Early Career Award for Scientists and Engineers (PECASE); **2002-07**, Independent Scientist Award (NINDS/NIH); **2009-14**, Mid-career Investigator Award in Mouse Pathobiology, NCRR/NIH; **2016**, The DeArmond Lecture (AANP Annual Meeting); **2017**, The Stowell Lecture (UC Davis).

Dr. Huang has a long-standing interest in the cellular and molecular mechanisms of human neurodevelopmental diseases. His approach combines the strengths of animal models and direct investigations in human brain tissues to understand the dynamic mechanisms of neurogenesis, neuronal migration and gliogenesis during embryonic and early postnatal life, and how disease process, such as genetic perturbations and environmental insults, might impact on human brain development. To achieve this goal, Dr. Huang spearheaded the efforts to establish a fetal/pediatric brain tissue banking system at UCSF. The success of this endeavor is underscored by our extensive collaborations with colleagues within and outside UCSF, which lead to many high impact publications that elucidate the fundamental mechanisms for newborn neuron migration and glial responses to hypoxic ischemic injury in early postnatal life. These studies revealed previously unrecognized role of neural circuit dysfunctions in neurodevelopmental disorders, and re-define our approaches toward investigating the molecular and cellular pathways in these devastating diseases.

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Overview: Scientific Sessions

Friday, June 9, 2017 &
Saturday, June 10, 2017

All abstracts of the papers presented in this program are published in the June 2017 issue of the *Journal of Neuropathology and Experimental Neurology.*

FRIDAY PLATFORMS 1 & 2

	Platform Session 1: ***Neurodegenerative: Alzheimer*** ***Grand Ballroom ABCD*** **Moderators: Edward B. Lee, MD, PhD; Julia Kofler, MD**	**Platform Session 2:** ***Tumors: Adult*** **Grand Ballroom EGF** **Moderators: Melike Pekmezci, MD; David Ellison, MD, PhD**
8:00 am – 8:15 am	**1** **Primary age-related tauopathy (PART) and brain arteriolosclerosis contribute to mild cognitive impairment** Peter Nelson, Erin Abner, Richard Kryscio, Frederick Schmitt, David Fardo, Daniela Moga, Eseosa Ighodaro, Gregory Jicha, Lei Yu, Hiroko Dodge, Chengjie Xiong, Randall Woltjer, Julie Schneider, Nigel Cairns, David Bennett	**9** **Multidimensional Scaling of Diffuse Gliomas: Application of the WHO's 2016 Revised Classification with Molecular Subgroup Discovery** Patrick Cimino, Michael Zager, Lisa McFerrin, Eric Holland
8:15 am – 8:30 am	**2** **The Expression of *MSRB3*, Implicated in Decreased Hippocampal Volume, Changes in Alzheimer's Disease and Associates With Synaptic Vesicles** Ivana Delalle, Stephanie Adams, Laurent Benayoun, Jayandra Himali, Kathy Tilton, Sudha Seshadri	**10** **Targeted NGS of Paired Tumor and Normal DNA Reveals Frequent Cancer Predisposing Germline Alterations in Neuro-oncology Patients** David Solomon, Nancy Joseph, Jessica van Ziffle, Eric Talevich, Courtney Onodera, James Grenert, Iwei Yeh, Boris Bastian
8:30 am – 8:45 am	**3** **The atomic structures of Tau filaments from Alzheimer disease brain** Anthony Fitzpatrick, Ben Falcon, Shaoda He, Alexey Murzin, Garib Murshudov, Holly Garringer, R. Anthony Crowther, Bernardino Ghetti, Michel Goedert, Sjors Scheres	**11** **Alternative splicing of neurofibromin 1 is an alternate mechanism of MAPK pathway activation in high grade astrocytoma** Cynthia Hawkins, Arun Ramani, Man Yu, Michael Brudno, Robert Siddaway
8:45 am – 9:00 am	**4** **Novel fluid biomarkers for brain amyloid, dementia risk, and body mass index yield new insights into preclinical Alzheimer disease** Richard Perrin, Hua Weng, John Morris, Tammie Benzinger, Anne Fagan, Chengjie Xiong, David Holtzman	**12** **Young adult glioblastomas harbor mutational profiles and driver events distinct from classic adult glioblastoma** Shakti Ramkissoon, Eric Severson, Adrienne Johnson, Laurie Gay, Jo-Anne Vergilio, Julia Elvin, James Suh, Sugganth Daniel, Alexa Schrock, Glenn Lesser, Sonika Dahiya, David Fabrizio, Prasanth Reddy, Caitlin Connelly, Garrett Frampton, Siraj Ali, James Sun, Philip Stephens, Vincent Miller, Jeffery Ross
9:00 am – 9:15 am	**5** **Locus coeruleus tauopathy in aging and early Alzheimer Disease.** Edward Plowey, Margaret Flanagan, Anusha Bharadwaj	**13** **SETD2 Mutations in CNS tumors** Angela Viaene, Mariarita Santi, MacLean Nasrallah
9:15 am – 9:30 am	**6** **Cryptic exon splicing repression by TDP-43 represents its major function compromised in Alzheimer's disease and ALS/FTD** Liam Chen, Mingkuan Sun, Robert Bell, Olga Pletnikova, Juan Troncoso, Philip Wong	**14** **Genomic analysis of high-grade meningiomas** Malak Abedalthagafi, Linda Bi, Noah Greenwald, Jeremiah Wala, Pankaj Agarwalla, Peleg Horowitz, Ossama Al-Mefty, Gavin Dunn, Sandro Santagata, Ian Dunn, Rameen Beroukhim
9:30 am – 9:45 am	**7** **Modeling Alzheimer's Disease via Human Induced Pluripotent Stem Cell derived Cerebral Organoids** Anita Huttner	**15** **Distinct Expression Patterns of Hypoxia Pathway Proteins in Selected Morphological Variants of Meningioma** Rati Chkheidze, Kimmo Hatanpaa, Charles White, Dennis Burns, Jack Raisanen, Chunyu Cai
9:45 am – 10:00 am	**8** **Apolipoprotein E4 Is Associated with Lewy Body Distribution and Pathologic Burden Independent of Alzheimer's Disease Pathology** Dennis Dickson, Michael Heckman, Melissa Murray, Alexandra Soto-Ortolaza, Ronald Walton, Nancy Diehl, Jay van Gerpen, Ryan Uitti, Zbigniew Wszolek, Nilufer Ertekin-Taner, David Knopman, Ronald Petersen, Neill Graff-Radford, Bradley Boeve, Guojun Bu, Tanis Ferman, Owen Ross	**16** **GATA3 Immunoreactivity is Characteristic of Gonadotroph Adenomas** William McDonald, Amber Wang, Nilanjana Banerji, Joseph Pollei, Bridget Ho, Kelsey McDonald

FRIDAY PLATFORMS 3 & 4

	Platform Session 3 ***Muscle, Nerve and Eye Pathology*** **Grand Ballroom ABCD** **Moderators: Charles Eberhart, MD, PhD; Mariarita Santi-Vicini, MD, PhD**	**Platform Session 4** *Tumors: Pediatric* **Grand Ballroom EFC** **Moderators: Fausto Rodriguez, MD; David Solomon, MD, PhD**
2:00 pm – 2:15 pm	**17** **The E274D Variant of p62/SQSTM1 Increases the Risk of Sporadic Inclusion Body Myositis** Marta Margeta, Jennifer Cotter, Xianhong Wang, Natasa Djordjevic	**25** **Global reduction in H3K27me3 is a molecular surrogate for pediatric posterior fossa- group A ependymomas** Pooja Panwalkar, Jonathan Clark, Vijay Ramaswamy, Debra Hawes, Fuseng Yang, Christopher Dunham, Stephen Yip, Juliette Hukin, Yilun Sun, Matthew Schipper, Ashley Margol, Melike Pekmezci, Chan Chung, Adam Banda, Sarah J. Curry, Mariarita Santi, Fausto Rodriguez, Matija Snuderl, Amanda M. Saratsis, Craig Horbinski, Marcel Kool, Stephan M. Pfister, Michael D. Taylor, Cynthia Hawkins, Andrey Korshunov, Alexander R. Judkins, Sriram Venneti
2:15 pm – 2:30 pm	**18** **Muscle Pathology in Patients with the *FKRP* Mexican Founder Mutation** Karra Jones, Angela Lee, Steven Moore	**26** **Molecular Heterogeneity Among Pediatric Posterior Fossa Ependymomas** David Ellison, Kristian Pajtler, Ji Wen, Martin Sill, Tong Lin, Jens Hübner, Vijay Ramaswamy, David Jones, Hendrik Witt, Lukas Chavez, Ruth Tatevossian, Richard Grundy, Thomas Merchant, Arzu Onar-Thomas, Michael Taylor, Stefan Pfister, Andrey Korshunov, Marcel Kool
2:30 pm – 2:45 pm	**19** **Restoration of truncated dystrophin expression with AAV-microdystrophin in the mouse model of Duchenne muscular dystrophy** Michael Lawlor, Kristy Brown, Margaret Beatka, Brittany Fickau, Hui Meng, Jennifer Tinklenberg, Samuel Ayers, Sharla Birch, Dongsheng Duan, Joel Schneider	**27** **Genetic Features of Astroblastoma by Targeted Next-Generation Sequencing: Lack of Unifying Alterations Across Eight Cases** Matthew Wood, Arie Perry, Andrey Korshunov, Geeta Chacko, Cunfeng Pu, Christopher Payne, Serguei Bannykh, Clinton Turner, Tarik Tihan, David Solomon
2:45 pm – 3:00 pm	**20** **Total and Regulatory T Cell Counts are Higher in HIV-associated than Sporadic T Cell-mediated Inflammatory Myopathies** Jennifer Cotter, Marta Margeta	**28** **Oligodendrogliomas in pediatric and teenage patients only rarely exhibit molecular markers and have excellent survivals** Ho Keung Ng, Yanxi Li, Aden Chan, Zhifeng Shi, Hong Chen, Jinsong Wu
3:00 pm – 3:15 pm	**21** **Differential Diagnostic Considerations for Giant Cell Myositis** Joshua Klonoski, Meghan Driscoll, Joshua Sonnen, Cheryl Palmer	**29** ***BRAF*-fused and 1p-deleted Glioneuronal Tumors: a Spectrum of Dissemination** Jason Chiang, Julie Harreld, Brent Orr, Suash Sharma, Azzam Ismail, Kamran Badizadegan, Annette Segura, David Ellison
3:15 pm – 3:30 pm	**22** **Diagnostic utility of C5b-9 immunostain in peripheral neuropathies** Paul Yell, Evan Dittmar, Dennis Burns, Chunyu Cai	**30** **Genetic Susceptibility and Evolution of Pediatric IDH-Mutant Infiltrating Astrocytomas** Cheng-Ying Ho, Peter Burger, Fausto Rodriguez, David Solomon, Sabine Mueller, Daniel Boue, Brent Orr, Alberto Broniscer, Mahtab Tehrani, James Dollar, Jeremy Goecks
3:30 pm – 3:45 pm	**23** **Identifying corneal infections in formalin fixed specimens using next generation sequencing** Charles Eberhart, Zhigang Li, Florian Breitwieser, Jennifer Lu, Albert Jun, Steven Salzberg	**31** **Medulloblastoma genomic subgroup-specific outcomes in irradiated children above 3 years treated at King Fahad medical city (KFMC)** Malak Abedalthagafi, Nahla Mobark, Musa AlHarbi, Othman Mosleh, Sandro Santagata
3:45 pm – 4:00 pm	**24** **Sebaceous adenoma of the eyelid and the Muir-Torre syndrome: a case report and a review of the literature** Yuan Rong, Hope Richard	**32** **Non-hematopoietic central nervous system metastases in children: clinicopathologic features of 24 cases** Jiancong Liang

FRIDAY POSTERS #33-#51

Friday, June 9, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	33	**Morphological Spectrum of Acute White Matter Lesions in Multiple Sclerosis** Murad Alturkustani, Basem Bahakeem, Qi Zhang, Lee-Cyn Ang
	34	**CD8⁺ T-Cell Leukoencephalitis With Astrocytopathy Clinically Presenting As Neuromyelitis Optica** Diana Thomas, Geoffrey Murdoch, Clayton Wiley
	35	**ALK Positive CNS Parenchymal Tumor with Inflammatory Myofibroblastic Tumor Histological Features** Yelena Fudym, Marc Rosenblum, Joseph Fullmer
	36	**Weston Hurst Syndrome Presenting As A Single Lesion** Nichole Allen, Aaron Wagner, Gary Pearl, Nicholas Avgeropoulos
	37	**Anti-PD-1-associated inflammatory-demyelinating lesions in patients with brain metastases** Peyman Samghabadi, Sherif Makar, Reena Thomas, Judith Pelpola, Sunil Reddy, Eric Tranvinh, Allyson Spence, Hannes Vogel, Donald Born, Raymond Sobel
	38	**Idiopathic granulomatous hypophysitis presenting with pituitary dysfunction and mass lesion: Two-case report.** Vaibhav Chumbalkar, Jiang Qian
	39	**Neuropathology of Lgi1 antibody limbic encephalitis** Alejandro Perez, Gustavo Roman, Suzanne Powell, Joseph Masdeu, Ronald Fisher, Andreana Rivera, Matthew Cykowski
	40	**Cerebral mycobacterial spindle cell pseudotumor in an HIV-infected patient.** Ewa Borys, Oluwatobi Odetola, Stefan Pambuccian
	41	**A case of meningo-encephalitis with unusual neuropathology** Aditya Shivane, Philip Edwards, David Hilton, Richard Cunningham, Laura Nabarro, Timothy Harrower
	42	**Citrobacter meningitis in an adult patient with unsuspected disseminated strongyloidiasis--a clinical case report with literature review** Yang Liu, Joshua Kagan, Nigar Anjuman-Khurram, Rong Xia, Caitlin Otto, Carmencita Yudis, Jenny Libien, Jianying Zeng
	43	**Neuropathological study of two cases with false positive real time quake- induced conversion result of cerebrospinal fluid for prion protein** Shigeo Murayama, Rie Motoyama, Toshihiko Shimizu, Yuta Nakano, Junko Fujigasaki, Renpei Sengoku, Katsuya Sato, Masaki Takao
	44	**Cerebral Sparganosis Masquerading as a Brain Tumor in a Young Adult** Khalda Ibrahim, Hasan Khalidi, Bryan Schmitt, James Snyder, Omar Choudhri, Eyas Hattab
	45	**Acute Hydrocephalus secondary to Holocord Epidural Abscess in an IV drug abuser that presented with abdominal pain: A Case Report** Yasir Al-Khalili, Shaymaa Ashi, Colin Kanach, Christos Katsetos
	46	**The effect of cerebral amyloid angiopathy on integrity of the blood brain barrier in Alzheimer disease** Shino Magaki, Zhaoyi Tang, Spencer Tung, Darrick Lo, William Yong, Negar Khanlou, Harry Vinters
	47	**Subcranial Cholinergic Ganglia in Alzheimer's Disease** Ralf Schober, Isabel Hilbrich, Max Holzer
	48	**An Autopsy Case of Early Onset Alzheimer's Disease with G378E Mutation in *PSEN1* Showing Widespread Tau and Aβ Pathologies** Hajime Miyata, Taizen Nakase, Takeshi Ikeuchi, Yasuji Yoshida
	49	**Targeting Pathological Proteins in Alzheimer's Disease** Krystal Herline, Fernando Goni, Thomas Wisniewski
	50	**Severe α-synucleinopathy in Alzheimer Disease associated with the *Presenilin 1* p.A396T mutation** Dibson Gondim, Owen Ross, Rose Richardson, Bernardino Ghetti
	51	**Alzheimer Disease with Psychiatric Features Associated with a Presenilin 2 Deletion** Kathryn Scherpelz, Stephanie Bucks, Thomas Bird, MIchael Dorschner, Caitlin Latimer, Debby Tsuang, Suman Jayadev, Luis Gonzalez-Cuyar, C. Dirk Keene

Posters are not offered for CME credit

FRIDAY POSTERS #52-#71

Friday, June 9, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	52	**Astrocytic tau deposition is frequent in Alzheimer disease** Amber Nolan, Elisa De Paula Franca Resende, Cathrine Petersen, Alexander Ehrenberg, Salvatore Spina, Bruce Miller, William Seeley, Lea Grinberg
	53	**Application of a condensed NIA-AA protocol for the pathologic assessment of Alzheimer's disease in an academic hospital autopsy service** Rajnish Bharadwaj, Patrick Cimino, Margaret Flanagan, Caitlin Latimer, Luis Gonzalez-Cuyar, Gordana Juric-Sekhar, Thomas Montine, Desiree Marshall, Christopher Keene
	54	**Severe Leukoencephalopathy in a Patient with Early-Onset Alzheimer Disease and Diffuse Lewy Body Disease** Jose Bonnin, Holly Garringer, Rose Richardson, Francine Epperson, Bernardino Ghetti
	55	**Gender Differences in Alzheimer's Disease: Brain Atrophy, Histopathology Burden and Cognition** Geidy Serrano, Jessica Walker, Jessica Filon, Lucia Sue, Thomas Beach
	56	**TDP-43 pathological changes in early onset Alzheimer's disease and late onset Alzheimer's disease** William Bell, Collin English, Jonathan Ling, Yusuke Kageyama, Philip Wong, Juan Troncoso
	57	**Antemortem-Postmortem Correlation of Florbetapir (^{18}F) PET Amyloid Imaging with Quantitative Biochemical Measures of Aβ40 and Aβ42** Thomas G. Beach, Chera L. Maarouf, Anthony Intorcia,Lucia I. Sue, Geidy E. Serrano, Alex E. Roher
	58	**Hippocampal sclerosis in the very old Finns (*Equal Contribution)** Mia Kero , Anna Raunio , Tuomo Polvikoski , Pentti Tienari , Anders Paetau *, Liisa Myllykangas *
	59	**New Insight to the Diabetic Lacy Vacuolation of Iris Pigment Epithelium** Alireza Ghaffarieh
	60	**PAX8 Expression in Adenocarcinoma of the Retinal Pigment Epithelium** Mehenaz Hanbazazh, Bradley Thuro, Kymberly Gyure
	61	**Ependymoma Can Arise as Part of Multiple Endocrine Neoplasia Type 1 (MEN1) Syndrome: Molecular Confirmation** Areli K. Cuevas-Ocampo, Andrew W. Bollen, Benjamin Goode, Kristian Pajtler, Lucas Chavez, Tanvi Sharma, Sun-Chuan Dai, Michael McDermott, Arie Perry, Andrey Korshunov, David Solomon
	62	**Hemophagocytic Lymphohistiocytosis in Adults with Intraocular Involvement: Clinicopathologic Features of 3 Cases** Maria Vizcaino Villalobos, Charles Eberhart, Fausto Rodriguez
	63	**Ocular Manifestations of Behçet's disease: a Clinicopathologic Case Description with Literature Review.** Bartholomew White, Michael Wilkinson, Charles Specht
	64	**Quadruple Neoplasms Following Radiation Therapy for Bilateral Retinoblastoma** Linda Szymanski, Maria Sibug Saber, John Go, Jonathan Kim, Gabriel Zada, Narsing Rao, Kyle Hurth
	65	**Striatal Cholinergic Neurons in Cervical Dystonia** Karin Mente, Nancy Edwards, Drew Pratt, Diego Iacono, Demelio Urbano, Abhik Ray-Chaudhury, Mark Hallett
	66	**Correlation of intraepidermal nerve fiber area in formalin-fixed skin tissue with small unmyelinated fibers on sural nerve biopsies** Darren Groh, Grant Jolly, John Donahue
	67	**Low-grade fibromyxoid sarcoma arising within the median nerve** Amy Swanson, Caterina Giannini, Andrew Folpe, Daniel Van Dyke, Rachael Vaubel
	68	**Intracranial Malignant Peripheral Nerve Sheath Tumor with Loss of H3K27me3 Expression** Rosalind Sharain, Jorge Torres-Mora, Aditya Raghunathan
	69	**Pathologic features of neonatal centronuclear myopathy associated with A618T dynamin-2 mutation** Amber Nolan, Marta Margeta
	70	**Diagnostic genetic testing for facioscapulohumeral dystrophy types 1 and 2 (FSHD1 and FSHD2)** Steven Moore
	71	**Calciphylaxis on Muscle Biopsy: A Case Report with Clinicopathologic Findings and Literature Review.** Bartholomew White, Lisa Carpenter, Gregory Emkey, Charles Specht

Posters are not offered for CME credit

FRIDAY POSTERS #72-#89

Friday, June 9, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	72	**Large fiber predominant axonopathy and respiratory insufficiency as the presentation of a patient with a BAG3 mutation** Caitlin Latimer, B. Distad, Bradley Rolf, Fuki Hisama, C. Keene, Luis Gonzalez-Cuyar
	73	**Giant Cell Polymyositis and Myocarditis in a Patient with Thymoma and Myasthenia Gravis: A Post-viral Autoimmune Process?** David Priemer, Darrell Davidson, Patrick Loehrer, Sunil Badve
	74	**Pediatric Diffuse Midline Glioma, H3 K27M-Mutant, With Ependymal Differentiation. Report of a Case** Christine Fuller, Francesco Mangano, James Leach, Ruby Khoury, David Ellison, Ralph Salloum
	75	**Extending the Neuroanatomic Territory of Diffuse Midline Glioma, K27M-Mutant: Pineal Region Origin.** Andrea Gilbert, Wafik Zaky, Murat Gokden, Christine Fuller, Eylem Ocal, Norman Leeds, Gregory Fuller
	76	**Anaplastic Diffuse Leptomeningeal Glioneuronal Tumor, two cases** Juan Mercado-Acosta, Kenneth Fallon, James Hackney, Arie Perry
	77	**A Pediatric Case of Diffuse Glioma Diagnosed at Autopsy** Jennifer Ross, Adriana Olar, Christine Fuller
	78	**Triple Trouble: A Case of Three Distinct Synchronous Brain Malignant Neoplasms in a Patient with Li-Fraumeni Syndrome.** Jorge Novo, Hussein Alnajar, Mehmet Kocak, Lorenzo Munoz, Clement Pillainayagam, Leonidas Arvanitis
	79	**Pediatric Diffuse Midline Glioma, H3 K27M-Mutant, With Ganglionic Differentiation. Case Report.** Sheryl Johnson, Christine Fuller, Mariko DeWire, James Leach, Amber D'Souza
	80	**Assessment of Focal Gene Amplifications and EGFRvIII Mutation in Glioblastoma Through Next-Generation Sequencing.** Michael Miller, Fei Ye, Jessica Tome-Garcia, David Zhang, Nadejda Tsankova
	81	**Anaplastic Transformation in a Pilocytic Astrocytoma** Giselle Lopez, Arie Perry, Marilyn Li, Brian Harding, Mariarita Santi
	82	**Histology, surrogate markers and prognosis of ependymoma, *RELA* fusion-positive: Multicentric Japanese study on ependymoma** Atsushi Sasaki, Junko Hirato, Takanori Hirose, Naohito Hashimoto, Keisuke Ishizawa, Koichi Ichimura, Hiroaki Sakamoto, Ryo Nishikawa
	83	**Synchronous gemistocytic astrocytoma IDH-mutant and oligodendroglioma IDH-mutant and 1p19q-codeleted in a patient with CCDC26 polymorphism** Rachael Vaubel, Thomas Kollmeyer, Alissa Caron, Emily Barr Fritcher, Jesse Voss, Haohai Liang, Robert Jenkins, Benjamin Kipp, Caterina Giannini
	84	**Rapid extracranial metastasis of BRAF-mutant high grade glioma with complete loss of glial immunophenotype** Nishant Tiwari, Jennifer Cotter
	85	**Adult Brainstem Gliomas with H3 K27M Mutation: Radiology, Pathology, and Prognosis.** Elena Daoud, Veena Rajaram, Chunyu Cai, Robert Oberle, Gregory Martin, Kevin King, Bruce Mickey, Jack Raisanen, Kimmo Hatanpaa
	86	**Clinicopathologic Features of the New Entity CNS High-Grade Neuroepithelial Tumor with BCOR Alteration** Sean Ferris, Joanna Phillips, Andrew Bollen, Tarik Tihan, Arie Perry, David Solomon
	87	**Diffuse Midline Glioma, H3 K27M-Mutant Tumor Presenting as a Diffuse Leptomeningeal Tumor. Case Report.** Sheryl Johnson, Christine Fuller, Trent Hummel, James Leach, Maryam Fouldadi
	88	**Epithelioid Glioblastoma (E-GBM): Histological And Molecular Characterization Of A Mini Series.** Yasha Chickabasaviah, Parul Jain, Bevinahalli Nandeesh, Harsha Sugur, Vani Santosh
	89	**Metastatic Glioblastoma to the Parotid Gland and Neck Lymph Nodes** Meghan Driscoll, Benjamin Witt, Cheryl Palmer, Karen L. Salzman

Posters are not offered for CME credit

FRIDAY POSTERS #90-#109

Friday, June 9, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	90	**A genome-wide analysis of rapidly progressive IDH-mutated astrocytomas** Timothy Richardson, Matija Snuderl, Jonathan Serrano, Matthias Karajannis, Adriana Heguy, Dwight Oliver, Jack Raisanen, Elizabeth Maher, Edward Pan, Samuel Barnett, Chunyu Cai, Amyn Habib, Robert Bachoo, Kimmo Hatanpaa
	91	**Copy number alteration on SNP arrays of long-standing oligodendroglioma** Takashi Komori, Kenta Masui, Masayuki Nitta, Takashi Maruyama, Yoshihiro Muragaki, Takakazu Kawamata
	92	**Non-Midline H3 K27M-Mutant Glioma** Vivian Snyder, Christina Di Loreto, James Chen, Lawrence Hansen, Karra Jones
	93	**The Costimulatory Ligand CD70 Is Expressed In High-Grade Diffuse Gliomas** Drew Pratt, Maryknoll Palisoc, Stefania Pittaluga, Markku Miettinen, Zied Abdullaev, Svetlana Pack, Martha Quezado
	94	**The Role of NOTCH1 and PI3K Pathways In The Progression of Oligodendrogliomas** Jose Velazquez Vega, Sameer Halani, Safoora Yousefi, Fatemeh Amrollahi, Chad Holder, Laila Poisson, Brent Griffith, Jennifer Eschbacher, Michael Nalisnik, Jeffrey Olson, Lee Cooper, Daniel Brat
	95	**Neoplasia Arising from Exogenous Stem Cell Introduction** Michael Miller, Saad Mir, Indira Guleria, John Chi, Keith Ligon, Aaron Berkowitz
	96	**A novel *GIT2-BRAF* fusion event in pilocytic astrocytoma.** Jeffrey Helgager, Hart Lidov, Mark Kieran, Keith Ligon, Sanda Alexandrescu
	97	**Epithelioid glioblastoma arising in an anaplastic pleomorphic xanthoastrocytoma: supporting the 'consanguinity' concept** Suash Sharma, Ravindra Kolhe, Scott Rahimi, Amyn Rojiani
	98	**Anaplastic Myxopapillary Ependymoma In An Infant: A Case Report and Literature Review** Darshan Trivedi, Zhenggang Xiong
	99	**Rare Variants of Gliosarcoma: Histologic and Molecular Findings** Vivian Snyder, Christina Di Loreto, Denise Malicki, Lawrence Hansen
	100	**Diffuse astrocytoma, IDH-wildtype: A vanishing diagnosis** Werner Paulus, David Reuss, Mohammed Jaber, Annkatrin Bibo, Anika Witten, Oliver Grauer, Stephanie Terwey, Uta Schick, Heinrich Ebel, Walter Stummer, Andreas von Deimling, Martin Hasselblatt
	101	**Diffuse astrocytoma WHO grade II with novel MYCN amplification** Won Lee, Ravindra Kolhe, Suash Sharma
	102	**Late-Onset Myxopapillary Ependymoma Metastasis Presenting As An Isolated Sellar Lesion: A Case Report** Jorge Novo, Hussein Alnajar, Mehmet Kocak, Lorenzo Munoz, Clement Pillainayagam, Leonidas Arvanitis
	103	**Clinicopathological analysis of diffuse midline glioma, H3 K27M-mutant** Seong-Ik Kim, Yujin Lee, Jae Kyung Won, Sung-Hye Park
	104	**Anaplastic Ganglioglioma in a Septuagenarian: a case report** Tiffany Hollenbeck, Douglas Miller, Kathy Newell
	105	**Subependymomas of the spinal cord: A report of four cases** Michael Paolini, William Krauss, Caterina Giannini, Michelle Clarke, Aditya Raghunathan
	106	**ATRX Mutation in Anaplastic Pilocytic Astrocytoma** Milad Webb, Kyle Conway, Mark Hoddinott, Kathryn McFadden, Sandra Camelo-Piragua
	107	**Radiation-induced glioblastoma at the site of a treated cerebellar medulloblastoma: A case report** Hussein Alnajar, Hussein Alnajar, Jason Chiang, Jorge Novo, Mehmet Kocak, Clement Pillainayagam, Lorenzo Munoz, Leonidas Arvanitis
	108	**Somatic Truncating Mutation of TSC1 in a Patient with Subependymal Giant Cell Astrocytoma** David Pisapia, Andrea Sboner, Mark Souweidane, Ehud Lavi, Himisha Beltran, Mark Rubin
	109	**Clinicopathologic differences between Latino American patients with gross total resection of glioblastoma compared to those without** Masoud Movassaghi, Mari Perez-Rosendahl, Kyeosuk Im, Gregory Lucey, Sergey Mareninov, Denise W. Ng, Randy Tashjian, Shino Magaki, Albert Lai, Phioanh (Leia) Nghiemphu, Timothy F. Cloughesy, Linda M. Liau, William H. Yong

Posters are not offered for CME credit

SATURDAY PLATFORMS 5 & 6

	Platform Session 5 ***Neurodegenerative: FTD/Lewy Body/Parkinson/ Vascular/Prion*** **Grand Ballroom ABCD** **Moderators: Richard Perrin, MD, PhD; Edward Plowey, MD, PhD**	**Platform Session 6** ***Developmental/Pediatric*** **Grand Ballroom EFG** **Moderators: Brian Harding, DPhil, FRCPath; Rebecca D. Folkerth, MD**
8:00 am – 8:15 am	**110** **Rapidly Progressive Frontotemporal Lobar Degeneration with TDP-43 Inclusions** Edward Lee, Silvia Porta, G Baer, Yan Xu, EunRan Suh, Linda Kwong, Lauren Elman, Murray Grossman, Virginia Lee, David Irwin, Vivianna Van Deerlin, John Trojanowski	**118** **Disruption of the ATXN1-CIC complex causes a spectrum of neurobehavioral phenotypes in mice and humans** Hsiang-Chih Lu, Qiumin Tan, Maxime Rousseaux, Wei Wang, Ji-Yoen Kim, Ronald Richman, Ying-Wooi Wan, Szu-Ying Yeh, Jay Patel, Xiuyun Liu, Tao Lin, Yoontae Lee, John Fryer, Jing Han, Maria Chahrour, Richard Finnell, Yunping Lei, Maria Zurita-Jimenez, Priyanka Ahimaz, Kwame Anyane-Yeboa, Lionel Van Maldergem, Daphne Lehalle, Nolwenn Jean-Marcais, Anne-Laure Mosca-Boidron, Julien Thevenon; Margot Cousin; Della Bro; Brendan Lanpher; Eric Klee; Nora Alexander; Mattherw Bainbridge; Harry Orr; Roy Sillitoe; Cecilia Ljungberg; Zhandong Liu; Christian Schaaf; Hua Zoghbi
8:15 am – 8:30 am	**111** **Amyotrophic lateral sclerosis-linked mutations increase the viscosity of liquid-like TDP-43 RNP granules in neurons** Pallavi Gopal, Jeffrey Nirschl, Eva Klinman, Erika Holzbaur	**119** **Partial Corpus Callosum Dysgenesis: A Previously Unreported Association With PTPN11 Gene Mutation in Noonan Syndrome.** Pryia Skaria, Sahibu Sultan Habeebu, Melissa Gener
8:30 am – 8:45 am	**112** ***GRN* mutations decrease the proportion of PGRN-positive CA1 microglia** Qinwen Mao, Missia Kohler, Jayson Wilson, Zachary Parton, Haibin Xia, Sandra Weintraub, Marsel Mesulam, Eileen Bigio	**120** **Alterations of Behavior, Motor Functions and Brain Morphology in Young Rats Following Exposure to Common Organic Solvents in Infancy** Mirna Lechpammer, Krista Thongphanh, Hilary Gonzales, Joanne Chan, Veronica Martínez-Cerdeño, Viswanathan Krishnan, Evgeny Nudler, David Zagzag, Robert Berman
8:45 am – 9:00 am	**113** **Identification of Novel Pathways in C9ORF72 Frontotemporal Dementia and Amyotrophic Lateral Sclerosis** Daniel Mordes, Mercedes Prudencio, Joseph Klim, Robert Moccia, Olli Pietilainen, Yingying Zhang, Dennis Dickson, Leonard Petrucelli, Kevin Eggan	**121** **Developmental Changes in Tau Protein Expression and Splicing** Marco Hefti, Kurt Farrell, Kathryn Bowles, Mary Fowkes, Towfique Raj, John Crary
9:00 am – 9:15 am	**114** **Elucidating the roles of the NBIA-mutated protein c19orf12 in mitochondrial function and metal homeostasis** Rajnish Bharadwaj, Leo Pallanck	**122** **Trophic Factors Involved In Developmental and Adult Hippocampal Neurogenesis In Humans.** Homa Adle-Biassette, Sara Cipriani, Isidre Ferrer, Jeannette Nardelli, Gabor Kovacs, Eleonora Aronica, Fabien Guimiot, Catherine Verney, Pierre Gressens
9:15 am – 9:30 am	**115** **Identification of Alpha-Synuclein in Enteroendocrine Cells Suggests a Direct Route of Pathologic Spread in Parkinson's Disease** Annie Hiniker, Rashmi Chandra, Yien-Ming Kuo, Robert Nussbaum, Rodger Liddle	**123** **Altered synaptic transmission and maturation in hippocampal CA1 neurons of a mouse model of human chromosome 16p11.2 microdeletion** Di Tian, Hung-Chi Lu
9:30 am – 9:45 am	**116** **A broader approach to the neuropathological assessment of vascular disease in cognitive impairment** Seth Love, Scott Miners	**124** **Incidence and Risk Factors for Neonatal Seizures in US natality records from 2005-2013. A Population-Based Study** Ahmed Gilani, Zainab Siddiq
9:45 am – 10:00 am	**117** **The Comparative Study Of Protease-Resistant Prion Protein Typically Associated With CJD And GSS Reveals An Unexpected Basic Similarity** Laura Cracco, Silvio Notari, Ignazio Cali, Tetsuyuki Kitamoto, Jose Bonnin, James Mastrianni, Bernardino Ghetti, Pierluigi Gambetti	**125** **Genetic mapping of developing brainstem motor neuron subtypes: implications for their differential susceptibility to disease** Matthew F. Rose, Max A. Tischfield, Alon Gelber, Alicia A. Nugent, Phillip Ang, Sarah Izen, Wentao Huang, Rahul Satija, Orit Rozenblatt-Rosen, Aviv Regev, Elizabeth C. Engle

SATURDAY PLATFORMS 7 & 8

	Platform Session 7 ***CTE/Pediatric Forensic/Trauma*** **Grand Ballroom ABCD** **Moderators: Veena Rajaram, MD; Jennifer Cotter, MD**	**Platform Session 8** ***Tumor Diagnostic Tools and Social Media*** **Grand Ballroom EFG** **Moderators: Sriram Venneti, MD, PhD; Cynthia Hawkins, MD, PhD**
1:30 pm – 1:45 pm	**126** **Clinicopathological Findings in 8 Young Ice Hockey Players** Ann McKee, Patrick Kiernan, Raymond Nicks, Victor Alvarez, Bobak Abdolmohammadi, Michael Alosco, Jesse Mez, Thor Stein, Bertrand Huber, Christopher Nowinski, Kerry Cormier, Caroline Kubilus, Douglas Katz, Robert Stern, Robert Cantu, Neil Kowall, Lee Goldstein	**134** **Stimulated Raman histology in brain tumors: A label free microscopic technique utilizing fresh unprocessed tissue.** Sandra Camelo-Piragua, Sandra Camelo-Piragua, Balaji Pandian, Yashar Niknafs, Todd Hollon, Julianne Boyle, Spencer Lewis, Mia Garrard, Shawn Hervey-Jumper, Hugh Garton, Cormac Maher, Jason Heth, Oren Sagher, D. Andrew Wilkinson, Matija Snuderl, Sriram Venneti, Shakti Ramkissoon, Kathryn McFadden, Amanda Fisher-Hubbard, Andrew Lieberman, Timothy Johnson, X. Sunney Xie, Jay Trautman, Christian Freudiger, Daniel Orringer
1:45 pm – 2:00 pm	**127** **Utility of TDP-43 immunohistochemistry in differentiating chronic traumatic encephalopathy from other tauopathies of aging** Travis Danielsen, Robert Reichard, Ping Shang, Charles White	**135** **Immediate Label-free Ex Vivo Evaluation of Human Brain Tumor Biopsies with Confocal Reflectance Microscopy** Jennifer Eschbacher, Joseph Georges, Eugene Belykh, Mohammadhassan Izady Yazdanabadi, Nikolay Martirosyan, Emily Szeto, Catherine Seiler, Kendall Van Keuren-Jensen, Mark Preul, Stephen Coons, Shwetal Mehta, Peter Nakaji
2:00 pm – 2:15 pm	**128** **The Pathological Distribution of Lewy Body Disease in Chronic Traumatic Encephalopathy** Jason Adams, Victor Alvarez, B. Huber, Weiming Xia, Ann McKee, Thor Stein	**136** **Using Intraoperative Cytology Preparations for Molecular Testing in Gliomas, a Method to Reduce Tissue Use and Turnaround Times** Angela Viaene, Robyn Sussman, Jason Rosenbaum, Renee McNamara, Jennifer Morrissette, MacLean Nasrallah
2:15 pm – 2:30 pm	**129** **Neuropathologic Examination in Sudden Unexpected Deaths in Infancy and Childhood: Recommendations for Diagnostic Yield** Rebecca D. Folkerth, Jacqueline Nunez, Zhanna Georgevskaya, Declan McGuone	**137** **Frozen foresight: Rapid IDH and *MGMT* testing in diffuse gliomas initiated at the time of frozen section diagnosis** John DeWitt, Justin Jordan, Julie Batten, Long Le, Michelle Toups, Daniel Cahill, Matthew Frosch, A. Iafrate, David Louis, Tracy Batchelor, Jochen Lennerz
2:30 pm – 2:45 pm	**130** **Methods and Procedures for the Neuropathologic Evaluation of the Infant Cervical Spine** Evan Matshes, Sam Andrews, Odey Ukpo, David Fowler	**138** **Brain Tumors and Neuroanatomical Imaging by Microscopy with UV Surface Excitation (MUSE)** Nicholas Coley, Miao Tian, Ferez Fereidouni, Zachary Harmany, Alexander Borowsky, David Zagzag, Richard Levenson, Mirna Lechpammer
2:45 pm – 3:00 pm	**131** **Pediatric and Peripartum Neuropathologic Findings at Autopsy: Interesting Cases in Forensics** Lyndsey Emery	**139** **Insulinoma-associated 1 (INSM1) is a novel nuclear marker of immature neuronal central nervous system neoplasms** Heather Ames, Lisa Rooper, Charles Eberhart, Fausto Rodriguez
3:00 pm – 3:15 pm	**132** **Filling A Void: Creating a Systematic Approach to Examining Post Mortem Brains of Unexpected Child Deaths** Arline Faustin, Ross Reichard, Cheddhi Thomas, Timothy Shepherd, Brooke O'Connell, Laura Crandall, Declan McGuone, Christopher William, Matija Snuderl, Thomas Wisniewski, Orrin Devinsky	**140** **Applying Whole Exome Sequencing to Pediatric Tumors of the Central Nervous System: Experience at Weill Cornell Medicine** David Pisapia, Prajwal Rajappa, Andrea Sboner, Olivier Elemento, Mark Souweidane, Mark Rubin, Himisha Beltran, Jeffrey Greenfield
3:15 pm – 3:30 pm	**133** **Traumatic Axonal Injury of Concussion** Melissa Blessing, R. Reichard	**141** **Neuropathology In Social Media** James Nix, Jerad Gardner, Murat Gokden, Brian Moore, Fausto Rodriguez, Maria Martinez-Lage, Felipe Costa, Douglas Anthony

SATURDAY POSTERS #142-#161

Saturday, June 10, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	142	**Cortical Mimic of Pilocytic Astrocytoma in Infantile Alexander Disease: A Case Report** Seth Lummus, Reuben Antony, Lee-Way Jin, Hannes Vogel
	143	**Opiate Cerebellar Toxicity: Correlation of Imaging and Neuropathology in a Child.** Brian Harding, Arastoo Voosough
	144	**Pacinian Hamartoma in Association with Occult Spinal Dysraphism** Rong Li, Brandon Rocque, David Kelly
	145	**Developing a Neuropathology of Autism Through Autism BrainNET** Marcello DiStasio, Ikue Nagakura, Matthew Anderson
	146	**Expression of transcription factors in colloid and Rathke cleft cysts** Janice Ahn, Kymberly Gyure
	147	**Agenesis of bilateral internal carotid arteries: A report of a rare congenital vascular anomaly identified at autopsy** Michael Paolini, Melanie Bois, Joseph Parisi, Jonathan Morris, Mark Jentoft
	148	**Vanishing white matter disease: an autopsy case report** Natalie Ellington, Veena Rajaram, Charles Timmons
	149	**FUS/TLS proteinopathy presenting as atypical parkinsonism** Kevin Bieniek, Dennis Dickson
	150	**Rod-shaped Microglia Show Disease and Regional Hippocampal Specificity** Missia Kohler, Jayson Wilson, Zachary Parton, Sandra Weintraub, Marsel Mesulam, Qinwen Mao, Eileen Bigio
	151	**Amyotrophic lateral sclerosis and parkinsonism-dementia complex that related to the focal area in Kii peninsula of Japan** Maya Mimuro, Maya Mimuro, Yasumasa Kokubo, Ryogen Sasaki, Yasushi Iwasaki, Mari Yoshida, Shigeki Kuzuhara
	152	**Lack and relative lack of vagus nerve alpha-synuclein pathology in an autopsy series of 49 normal elderly and 18 with incidental Lewy body disease.** Thomas G. Beach, Lucia I. Sue, Geidy E. Serrano, Anthony Intorcia, Holly A. Shill, Erika Driver-Dunckley, Shyamal Mehta, David Shprecher, Marwan N. Sabbagh and Charles H. Adler
	153	**Brain biopsy in patients presenting with suspected Cretutzfeldt-Jakob disease.** Francesca Brett, Daphne Chen, Alan Beausang, Jane Cryan, Farrell Michael, Looby Seamus, Heffernan Josephine, Heany Ciara, Cunningham Florence, Teresa Loftus
	154	**Wolfram syndrome with hypothalamic preservation and associated Wernicke-Korsakov syndrome** Derek Oakley, Jeremy Schmahmann, Anat Stemmer-Rachamimov, Matthew Frosch
	155	**TDP-43 Protein Variants as Biomarkers in Amyotrophic Lateral Sclerosis.** Galam Khan, Stephanie Williams, Brent Harris, John Ravits, Michael Sierks
	156	**Pathogenesis of prion-infected mice treated with 2-aminothiazole therapeutics** Brittany Dugger, David Berry, Rigo Roman-Albarran, Abby Oehler, Carlo Condello, Julian Castaneda, Yevgeniy Freyman, Kurt Giles, Stanley Prusiner
	157	**Progressive Ataxia and Palatal Tremor: Two Autopsy Cases of a Novel Tauopathy** Andrew Gao, Achinoam Faust-Socher, Maryam Al-Murshed, Marc Del Bigio, Anthony Lang, David Munoz
	158	**A rare case of leukoencephalopathy with intracranial calcifications and cysts (LCC)** Ewa Borys, Swathi Chidambaram, Stefan Pambuccian
	159	**Creutzfeldt-Jakob disease associated with PRNP V180I-129M mutation as a cause of dementia in elderly individuals** Masaki Takao, Hioaki Kimura, Ban Mihara, Takashi Kanda, Kazuo Yoshizawa, Mizuho Koide, Kimihito Arai, Yasumichi Arai, Nobuyoshi Hirose, Masaru Mimura, Tetsuyuki Kitamoto
	160	**TDP43 associations in the Nun Study and Honolulu Asian Aging Study** Margaret Flanagan, Laura Hemmy, Brenna Cholerton, Steven Edland, Kathleen Montine, Lon White, Thomas Montine
	161	**A Case of Multiple System Atrophy in the Medical Examiner Setting** Christopher Liverman, Eric Huang, Harminder Narula, Ellen Moffatt

Posters are not offered for CME credit

SATURDAY POSTERS #162-#181

Saturday, June 10, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	162	**Alterations to the glymphatic system in chronic traumatic encephalopathy through redistribution of aquaporin-4** Bertrand Huber, Victor Ventrano, Brock Knapp, Jonathan Cherry, Victor Alvarez, Thor Stein, Ann McKee
	163	**Clinical, Genetic, and Histopathologic Heterogeneity of Hereditary Diffuse Leukoencephalopathy with Spheroids: A Report of Two Cases** Caitlin Latimer, Suman Jayadev, Kathryn Scherpelz, Luis Gonzalez-Cuyar, Michael Dorschner, Thomas Bird, C. Keene
	164	**Ultrasensitive and selective detection of 3-repeat tau seeding activity in Pick disease brain and cerebrospinal fluid by tau RT-QuIC** Eri Saijo, Bernardino Ghetti, Gianluigi Zanusso, Adrian Oblak, Jennifer Furman, Allison Kraus, Byron Caughey
	165	**Cerebellar cortical axonal swellings ("torpedoes") in humans; increase with age, MSA, PSP, FTLD-TDP** John Hedreen
	166	**Hereditary Diffuse Leukoencephalopathy with Spheroids, associated with mutation p.F849del in the *CSF1R* gene** Dibson Gondim, Holly Garringer, Shannon Risacher, Francine Epperson, Andrew Saykin, Martin Farlow, Jose Bonnin, Bernardino Ghetti
	167	**Development of a novel resource to study military-related TBI/CTE neuropathology.** Desiree Marshall, Allison Beller, Zachary Hoffer, John Lacy, Thomas Clark, Elaine Peskind, C. Keene
	168	**Neuroimaging of White Matter Pathology in Two *CSF1R* p.F849del Mutation Carriers** Shannon Risacher, Dibson Gondim, Joey Contreras, Holly Garringer, Francine Epperson, Rose Richardson, Martin Farlow, Andrew Saykin, Bernardino Ghetti
	169	**The role of astrocyte senescence and p53 isotypes in neurodegenerative diseases and neurocognitive dysfunction after radiation or chemotherapy** Brent Harris, Casmir Turnquist, Ifeyinwa Obiorah, Suhua Han, Jessica Beck, Ingrid Bremer, Heather Ames, Izumi Horikawa, Curtis Harris
	170	**Neuropathological findings in an NBIA (Neurodegeneration with Brain Iron Accumulation) patient with a C19orf12 gene mutation.** Hemant Varma, Norris Hollie, David Simon, Matthew Anderson
	171	**Applying CRISPR/Cas9-Mediated Genome Editing To Generate Mouse Models Of Autism Spectrum Disorders** Di Tian, Hung-Chi Lu
	172	**Patient K.C.: Neuropathology of a Unique Case of Memory Impairment** Andrew Gao, Shayna Rosenbaum, Fuqiang Gao, Morris Moscovitch, Endel Tulving, Sandra Black, Julia Keith
	173	**Leptomeningeal Cyst: A Case Study and Review of the Literature** E. Kelly Mrachek, Shayan Moosa, Farooq Choudhry, Mark Shaffrey, M. Beatriz Lopes
	174	**Histopathology of the Cat Somatosensory cortex after chronic electrical stimulation** Nishant Tiwari, Carol Miller, Douglas McCreery
	175	**Primary Cerebral Amyloidoma Mimicking a Cavernous Malformation** Suzanna Logan, Jose Velazquez Vega, Matthew Schniederjan
	176	**DLPFC Transcriptome Defines Two Molecular Subtypes of Schizophrenia** C. Harker Rhodes, Eli Bowen, Caitlyn Lee, Jack Burgess, Richard Granger
	177	**Meningothelial Hyperplasia: Expanding the Clinicopathologic Spectrum** Amyn Rojiani, Bruce Gilbert, Mark Cohen
	178	**Rhabdomyosarcoma in association with pituitary adenoma: Common ancestor with subsequent genetic divergence.** Astin Powers, Christopher Trindade, Keith Killian, Prashant Chittiboina, Nina Afsar, Lynnette Nieman, Martha Quezado
	179	**Rapid onset cognitive decline in a middle-aged lady** Francesca Brett, Daphne Chen, Seamus Looby, Allan McCarthy, Cillian De Gascun, Elaine Jaffe, Richard Flavin
	180	**Aggressive pituitary adenomas may depend on suppression of immune response: a whole transcriptome analysis** Timothy Richardson, Chao Xing, Mohammed Kanchwala, Zhong-Jian Shen, James Malter, Samuel Barnett, Jack Raisanen, Dennis Burns, Charles White, Kimmo Hatanpaa
	181	**Genomic Profiling of Anaplastic Meningioma** Rebecca Yoda, Yigit Karasozen, Josh Warrick, Michael Dorschner, Manuel Ferriera, Patrick Cimino

Posters are not offered for CME credit

SATURDAY POSTERS #182-#200

Saturday, June 10, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	182	**Isolated Classical Hodgkin Lymphoma of Central Nervous System** Kwok Ling Kam, Teresa Scordino, Michael Sughrue, Kar-Ming Fung
	183	**The perivascular microenvironment in primary central nervous system Epstein-Barr virus-positive lymphomas: the role of PD-1 and PD-L1** Yasuo Sugita, Takuya Furuta, Satoru Komaki, Junko Miyoshi, Koichi Ohshima, Hideyuki Abe, Yoshihiro Tsukamoto, Hitoshi Takahashi, Akiyoshi Kakita
	184	**ASCL1 Expression in Atypical Teratoid Rhabdoid Tumors in Children** Debra Hawes, Benita Tamrazi
	185	**Somatostatin receptor 2A and Progesterone Receptor may aid in distinguishing Endolymphatic sac tumor from Meningioma** Aaron Halfpenny, Randall Woltjer
	186	**Lymphomatoid Granulomatosis (LYG), Presenting as Spinal Cord Lesion: A Case Report.** Aseeb Rehman, Llewellyn Foulke, Jiang Qian
	187	**Mosaic Postzygotic Mutations Leading to Cancer Predisposition in Patients with Neural Tumors** Lea Surrey, Gozde Akgumus, Kristin Zelley, Michael Fisher, Minjie Luo, Brian Harding, Marilyn Li, Mariarita Santi
	188	**Rare Case of Crystal Storing Histiocytosis Presenting Exclusively in the Central Nervous System (CNS) (*Contributed equally as first authors)** Timothy Richardson*, James Nix*, Jon Wilson, Jack Raisanen, Murat Gokden
	189	**Analysis of Skull Base Tumors by Raman Spectroscopy** Gordana Juric-Sekhar, Peter Chiarelli, Natasha Hippel, Laligam Sekhar
	190	**Pituitary adenomas in pediatric and adolescent populations** Jie Chen, Robert Schmidt, Sonika Dahiya
	191	**High-Grade B-Cell Lymphoma (HGBCL) Involving the Central Nervous System (CNS)** James Nix, Giovanni Insuasti-Beltran, Ryan Fitzgerald, Murat Gokden
	192	**PD-L1 is Not Expressed by CNS Embryonal Tumors** Xinhai Zhang, Mitra Harati, William Yong, Jeremy Deisch
	193	**Anaplastic Meningioma Presenting as a Pelvic Mass** Kimberly Stogner-Underwood, Sami Belakhlef, Mustafa Yousif
	194	**Erdheim-Chester disease with multifocal central nervous system involvement –A rare and challenging diagnosis** Michael Paolini, Natalie Parks, Joon Uhm, Aditya Raghunathan
	195	**LIN28A Expression In CNS Embryonal Tumours** Yasha Chickabasaviah, Shilpa Rao, R Rajeswarie, Bevinahalli Nandeesh, Arimappamagan Arivazhagan, Vani Santosh
	196	**Lung Adenocarcinoma Metastatic To The Brain: Most Common Histologic Pattern and Molecular Aberration** Anna Mathew, Ali Saad, Varsha Manucha
	197	**PD-L1 and PD-1 Expression in CNS Germinoma: A Preliminary Study of 22 Cases** Miriam Wildeman, Matthew Shepard, Oldfield Edward, M. Beatriz Lopes
	198	**Cerebellopontine Angle Atypical Teratoid/Rhabdoid Tumor Involving the Internal Acoustic Canal in an 11 Year Old** Kimberly Stogner-Underwood
	199	**Congenital neurovascular hamartoma is a clue to search for rhabdoid tumors** Sanda Alexandrescu, Birgitta Schmidt, Mark Kieran, Liliana Goumnerova, Junne Kamihara, Marilyn Liang
	200	**Primary Central Nervous System T-Cell Lymphomas: A Single Center Experience** Mercia Bezerra Gondim, Jiehao Zhou

Posters are not offered for CME credit

SATURDAY POSTERS #201-#219

Saturday, June 10, 2017		
Time:	**Poster #:**	**Garden 2&3, N. Tower**
8:00 am – 5:00 pm	201	**Cavernous Hemangioma Presenting as an Extradural Lesion in the Spine of an Adult** Nishant Tiwari, Josh Lucas, Chia-Shang Liu, John Liu, Kyle Hurth
	202	**LEF1 Immunohistochemistry Identifies CTNNB1-mutated Medulloblastomas** Diana Thomas, Geoffrey Murdoch, Ronald Hamilton
	203	**Tumor-to-tumor metastasis: Mucinous non-small cell lung carcinoma to sphenoid wing meningioma** Colin Kanach, Yasir Al-Khalili, Chelsey Gracia, Christos Katsetos
	204	**Genomic analysis of choroid plexus tumors reveals TERT alterations in atypical papillomas and recurrent chromosomal gains** Julieann Lee, Eric Talevich, Courtney Onodera, Jessica Van Ziffle, Nancy Joseph, Iwei Yeh, Boris Bastian, Aleli Siongco, Claudia Greco, Joanna Phillips, Andrew Bollen, Tarik Tihan, Arie Perry, David Solomon
	205	**Lymphatic Malformation Occurring Within the Lateral Ventricle: A Case Report** Seth Lummus, Gregory Scott, Samuel Cheshier, Donald Born
	206	**A Case of Schwannomatosis: Comparison of Histologic Features in 18 Different Tumors from a Single Patient** Douglas Miller
	207	**Secretory Meningioma of the Internal Auditory Canal** Caitlin Latimer, Victoria Lee, Lynn McGrath, Manuel Ferreira, Jay Rubinstein, Luis Gonzalez-Cuyar
	208	**Lack of Angiocentricity for the EBV Negative Diffuse Large B-cell Lymphoma of the CNS** Zhe Piao, Vaninder Chhabra, Vartan Tashjian, Todd Goldenberg
	209	**Establishment of the first orthotopic primary human chordoma xenograft model.** Homa Adle-Biassette, Henri Salle, Marc Pocard, Shahrazad Bouazza, Sebastien Froelich
	210	**Clinicopathologic features and pathogenesis of melanocytic colonization in meningioma** Mitra Dehghan Harati, Andrew Yu, Kyuseok Im, Mari Perez-Rosendahl, Young Park, Marvin Bergsneider, William Yong
	211	**A case of meningeal melanocytoma in a 42-year-old male.** Jeffrey Helgager, David Meredith, Ian Dunn, Umberto De Girolami
	212	**Multifocal Central Nervous System AIDS Related Epstein Barr Virus Associated Malignant Myopericytoma** Linda Szymanski, Melissa Barger, James Hu, Emily Blodget, Anandh Rajamohan, Gabriel Zada, Kyle Hurth
	213	**CNS Metastasis of a Pulmonary Angiosarcoma** Kyle S. Conway, Milad Webb, Jonathan McHugh, David Lucas, Sriram Venneti
	214	**Cerebral Segmental Arterial Mediolysis In a Pediatric Patient: A Novel Case Report** Peyman Samghabadi, Hannes Vogel
	215	**Caveolin-1 prevents cerebral edema in stroke by protecting junctional proteins on blood brain barrier** Min-Cheol LEE, Kyung-Hwa LEE, Kang-Ho Choi, Hyung-Seok KIM, Kyung-Ah Cho
	216	**Fatal Refractory Shock Following Endovascular Catheter Emboli: A Case Series** Marco Hefti, Daniel Wei, Mary Fowkes, John Crary, Reade De Leacy, Stefan Mayer
	217	**Lymphoplasmacytic Lymphoma Presenting as Giant Cell Arteritis: A Novel Case Report** Peyman Samghabadi, Gerald Berry, Yasodha Natkunam, Hannes Vogel, Stanford Shoor, Donald Born
	218	**Inflammatory cerebral amyloid angiopathy: a case report and review of the literature** Yuan Rong, Hope Richard
	219	**Cranial nerve cavernous angioma: a rare lesion of the oculomotor nerve** Patricia Pittman, Thomas Cummings

Posters are not offered for CME credit

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Endowed Lectureships

Friday, June 9, 2017

I. Parisi Lecture
II. DeArmond Lecture

Saturday, June 10, 2017

I. Saul R. Korey Lecture

Sunday, June 11, 2017

I. Matthew T. Moore Lecture

ENDOWED LECTURESHIP

PARISI LECTURE

The *Parisi Lecture* was established in 2007. The lecture was named the *Parisi Lectureship* in honor of one of the American Association of Neuropathologists' exceptional members, Dr. Joseph E. Parisi. He has published seminal neuropathological studies on a wide range of diseases affecting the nervous system, with particular focus on neurodegenerative diseases and multiple sclerosis. He has held virtually every office of the Society, including President, and has served on several AANP committees. In 2006, his dedication and generosity were recognized with the Award for Meritorious Contributions to Neuropathology. He is considered by many the heart and soul of the association and a man worth emulating.

We are pleased to have **Sean J. Pittock** join our list of distinguished speakers.

2008	Claudia Lucchinetti	The Spectrum of CNS Inflammatory Demyelinating Diseases: *From Pathology to Pathogenesis*
2009	Hans Lassmann	Inflammation Induced Mitochondrial Injury: A Major Mechanism of Neurodegeneration
2010	Joseph Dalmau	Autoimmune Synaptic Encephalitis
2011	Steven S. Scherer	Molecular Pathologies at the Nodes of Ranvier
2012	Bruce D. Trapp	Neuronal Damage in Multiple Sclerosis
2013	Albee Messing	GFAP: Friend or Foe
2014	Clayton Wiley	Human Parechovirus Encephalitis
2015	Bruce T. Lamb	The Role of Innate Immunity in Neurodegenerative Disease Pathogenesis
2016	Bette Kleinschmidt-DeMasters	CNS White Matter Disorders with Viral Causation and Association
2017	Sean J. Pittock	Autoimmune Gliopathies: A Journey of Discovery

PARISI LECTURE

Autoimmune Gliopathies: A Journey of Discovery

Time: 10:30 am – 11:30 am

Date: Friday, June 9, 2017

Sean J. Pittock, MD, *Professor of Neurology, Mayo Clinic; Director of the Center for Multiple Sclerosis and Autoimmune Neurology at Mayo Clinic; Director of Neuroimmunology Laboratory at Mayo Clinic*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Recognize the evolving spectrum of AQP4, GFAP, and MOG autoimmunity.
2. Explain the molecular bases of disease and recognize novel therapeutic targets
3. Outline the alternative molecular-based approach to classification.

II. Abstract & Relevant References

This lecture will provide an overview of novel biomarker discoveries and advances being made in the study of autoimmune gliopathies. Firstly, neuromyelitis optica (NMO) spectrum disorders (SDs) considered under the umbrella term "Autoimmune Aquaporin-4 Channelopathy" represents an evolving spectrum of CNS-inflammatory-autoimmune-demyelinating diseases for which a specific antigen has been identified-the astrocytic water channel aquaporin-4 (AQP4). MS lacks a distinguishing biomarker. The discovery of AQP4-IgG represents a significant shift from emphasis on the oligodendrocyte and myelin to the astrocyte and was the first proven autoimmune gliopathy. The NMO of today represents a relapsing spectrum of disease (SD), extending beyond the optic nerves and spinal cord to include brain (especially in children) and rarely muscle. Most patients have MRI brain abnormalities and these are consistent with MS in up to 10% of patients. Typical NMOSD lesions are found in areas that highly express AQP4, including the circumventricular organs (accounting for intractable nausea and vomiting) and the diencephalon (accounting for sleep disorders, endocrinopathies and syndrome of inappropriate antidiuresis). Continued progress in our understanding of the immunobiology of AQP4 autoimmunity necessitates continuing revision of the clinical diagnostic criteria for NMO spectrum disorders. As the clinical spectrum broadens, the importance of highly specific assays that detect pathogenic AQP4-IgG targeting extracellular epitopes of AQP4 cannot be overemphasized. Individual patient-specific NMO therapies are likely to result from advanced serological interpretive insights, coupled with increased understanding of the pathogenic impact of binding of AQP4-IgG to its target on the astrocytic end foot (complement activation, AQP4 downregulation and coupled glutamate transporter downregulation). IgG targeting myelin oligodendrocyte glycoprotein (MOG), when detected using cell-based assays expressing recombinant MOG, is now recognized as a biomarker of an autoimmune oligodendrogliopathy (distinct from multiple sclerosis) considered an autoimmune "MOG-opathy". Its phenotype has many similarities to NMO. Recently our group reported a novel immunopathological biomarker, GFAP antibody, which when detected in CSF, unifies a spectrum of immunotherapy-responsive autoimmune inflammatory CNS disorders, termed "autoimmune GFAP astrocytopathy" distinct from infectious meningoencephalitis and idiopathic inflammatory CNS disorders such as multiple sclerosis, vasculitis, and sarcoidosis.

References:

1. Lennon VA, Wingerchuk DM, Kryzer TJ, **Pittock SJ**, Lucchinetti CF, Fujihara K, Nakashima I, Weinshenker BG. A serum autoantibody marker of neuromyelitis optica: distinction from multiple sclerosis. Lancet 2004 Dec 11-17; 364(9451):2106-12
2. Lennon VA, Kryzer TJ, **Pittock SJ**, Verkman AS, Hinson SR. IgG marker of optic-spinal multiple sclerosis binds to the aquaporin-4 water channel. J Exp Med 2005 Aug 15; 202(4):473-7
3. Roemer SF, Parisi JE, Lennon VA, Benarroch EE, Lassmann H, Bruck W, Mandler RN, Weinshenker BG, **Pittock SJ**, Wingerchuk DM, Lucchinetti CF. Pattern-specific loss of aquaporin-4 immunoreactivity distinguishes neuromyelitis optica from multiple sclerosis. Brain 2007 May; 130(Pt 5):1194-205

4. Hinson SR, Kryzer TJ, Lucchinetti CF, **Pittock SJ**, Roemer SF, Lennon VA. (2007) Pathogenic potential of IgG binding water channel extracellular domain in neuromyelitis optica. Neurology 2007; 69(24):2221-31.
5. Iorio R, Lucchinetti CF, Lennon VA, Farrugia G, Pasricha PJ, Weinshenker BG, **Pittock SJ**. Intractable nausea and vomiting from antibodies against a brain water channel. Clin Gastroenterol Hepatol 2013 Mar; 11(3):240-5.
6. **Pittock SJ**, Lennon VA, McKeon A, Mandrekar J, Weinshenker BG, Lucchinetti CF, O'Toole O, Wingerchuk DM. Eculizumab in AQP4-IgG-positive relapsing neuromyelitis optica spectrum disorders: an open-label pilot study. Lancet Neurol 2013 Jun;12(6):554-62.
7. **Pittock SJ**, Lucchinette CF. Neuromyelitis optica and the evolving spectrum of autoimmune aquaporin-4 channelopathies: a decade later. Ann N Y Acad Sci. 2015 June 10.
8. Flanagan EP, Cabre P, Weinshenker BG, St Sauver J, Jacobson DJ, Majed M, Lennon VA, Lucchinetti CF, McKeon A, Matiello M, Kale N, Wingerchuk DM, Mandrekar J, Sagen JA, Fryer JP, Borders Robinson A, **Pittock SJ**. Epidemiology of aquaporin-4 autoimmunity and neuromyelitis optica spectrum. Ann Neurol. 2016 Feb 17. doi: 10.1002/ana.24617.
9. Hinson SR, Lennon VA, **Pittock SJ**. Autoimmune AQP4 channelopathies and neuromyelitis optica spectrum disorders. In: *Handbook of Clinical Neurology, 3rd series: Autoimmune Neurology* (A Vincent, **SJ Pittock**, eds.), Oxford:Elsevier. 2016;133:377-403.
10. Flanagan EP, Hinson SR, Lennon VA, Fang B, Aksamit AJ, Morris PP, Basal E, Honorat JA, Alfugham NB, Linnoila JJ, Weinshenker BG, **Pittock SJ**, McKeon A.. Ann Neurol. 2017 Feb;81(2):298-309. doi: 10.1002/ana.24881.

III. Faculty Biography

Dr. Sean J. Pittock is Professor of Neurology at the Mayo Clinic, with a joint appointment in the Departments of Neurology and Laboratory Medicine and Pathology. He currently directs the Neuroimmunology Laboratory providing comprehensive neural antibody testing for 150,000 patients annually. He is the Director of the Center for MS and Autoimmune Neurology at the Mayo Clinic. He is the co-founder of the American Academy of Neurology's Autoimmune Neurology Section of which he is the vice chair. His expertise is in the diagnosis and management of immune mediated neurological disorders. In 2006, he set up the Autoimmune Neurology Clinic at the Mayo Clinic, the first dedicated Clinic of its type in the USA. This Clinic provides a multidisciplinary approach to the evaluation and treatment of patients with a broad range of autoimmune neurological disorders considered "rare orphan diseases". These disorders have often been misdiagnosed as untreatable neurodegenerative diseases; but are, in fact, responsive to immunotherapy and reversible. Dr Pittock's research is translational, and is focused on 1) the identification of novel biomarkers of autoimmune neurological diseases, 2) the clinical application of laboratory-based tests in diagnosis and outcome prediction for patients with autoimmune neurological disorders and 3) optimizing the clinical management of autoimmune neurological disorders. In the last decade, his work has focused on the diagnostic characteristics and immunotherapy outcomes of patients with autoimmune CNS demyelinating disorders (targets include aquaporin-4 and GFAP), epilepsies (targets include LGI1, CASPR2, NMDA, GABAB receptors) and gastrointestinal dysmotility (targets include ganglionic acetylcholine receptors and DPPX). This unique translational practice extending the laboratory's serologic findings directly to the bedside has allowed the creation of diagnostic decision trees which will optimize triaging of such patients for further phenotype analysis and biomarker discovery. Dr Pittock is also an educator in the field of autoimmune neurology and is actively involved in educational courses at both national (American Academy of Neurology, American Neurological Association) and international (World Congress of Neurology) meetings. He has received research support from Mayo Clinic, NIH (R01), the Guthy-Jackson Charitable Foundation and industry.

ENDOWED LECTURESHIP

DEARMOND LECTURE

The *DeArmond Lecture* was established in recognition of Stephen J. DeArmond's excellent leadership and organization of the scientific program for the 2006 International Congress of Neuropathology. This successful meeting garnered significant support intended for the future advancement of the mission of the American Association of Neuropathologists. To continue these intended goals and recognize Dr. DeArmond's contributions, the American Association of Neuropathologists has honored him by establishing the *DeArmond Lecture*. Dr. DeArmond is a leading authority on prion disease, where his work has been fundamental in demonstrating mechanisms of transmission and routes to therapeutics. The DeArmond Lecture focuses on honoring those making major advances in the field of neurodegeneration and aging with a particular emphasis on translating these findings to patient care.

We are pleased to have **David C. Van Essen** join our list of distinguished speakers.

2008	Virginia M.Y. Lee	TDP-43, A New Class of Proteinopathies in Neurodegenerative Diseases
2009	Rudy Tanzi	Decoding Alzheimer's Disease Gene by Gene
2010	Todd Golde	Alzheimer's Disease: Models and Therapeutics
2011	Beverley L. Davidson	Emerging Therapies for Neurogenetic Diseases
2012	Krystof Bankiewicz	New Therapies for Parkinson Disease
2013	Stanley Prusiner	A Unifying Role for Prions in Neurodegenerative Diseases
2014	Dale Bredesen	Prionic Loops, Dependence Receptors, and a New Approach to Alzheimer's Disease
2015	William W. Seeley	Frontotemporal Dementia: Onset and Spread
2016	Eric J. Huang	FTD and ALS: Genes, Circuits and Therapeutic Targets
2017	David C. Van Essen	Structure, Function, Connectivity, Development and Evolution of Human Cerebral Cortex

DEARMOND LECTURE

Structure, Function, Connectivity, Development and Evolution of Human Cerebral Cortex

Time: 4:45 pm – 5:45 pm

Date: Friday, June 9, 2017

David C. Van Essen, PhD, *Alumni Endowed Professor, Department of Neuroscience, Washington University in St. Louis, St. Louis, MO*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Summarize the current understanding of human cortical parcellation in individuals as well as group averages.
2. Explain the basic principles of distributed cortical connectivity in humans and nonhuman primates.
3. Summarize the key objectives and major accomplishments of the Human Connectome Project

II. Abstract & Relevant References

The cerebral cortex is the dominant structure of the mammalian brain, and it plays critical but diverse roles in cognition, perception, emotion, and motor control. This lecture will review recent progress in elucidating the structure, function, connectivity, development, and evolution of cerebral cortex in humans and nonhuman primates. Underlying methodological themes will include the power of surface-based analysis and visualization and the importance of user-friendly data sharing for accelerating progress in exploring these issues. Consideration of cortical development will include questions of why the cortex is a sheet whose convolutions vary across species and across individuals. Advances in elucidating functional organization include a recent multimodal human cortical parcellation, based on data from the Human Connectome Project (HCP), that reveals 180 distinct areas in each hemisphere. The ability to accurately parcellate the cortex in individual subjects will enable systematic analyses of individual variability in relation to many neurobiologically informative features as well as hundreds of behavioral measures that are part of the freely shared HCP data. Comparisons with nonhuman primates, including chimpanzees as well as macaque monkeys, provide intriguing evolutionary insights regarding the dramatic expansion of neocortical regions associated with higher cognition in the human lineage.

References:

1. Glasser, M.F., T.S. Coalson, E.C. Robinson, C.D. Hacker, J. Harwell, E. Yacoub, K. Ugurbil, J. Andersson, C.F. Beckmann, M. Jenkinson, S.M. Smith, and D.C. Van Essen. 2016. A multi-modal parcellation of human cerebral cortex. Nature 536: 171-178.
2. Glasser, M.F., Smith, S.M., Marcus, D.S., Andersson, J., Auerback, E.J., Behrens, T.E.J., Coalson, T.S., Harms, M.P., Jenkinson, M., Moeller, S., Robinson, E.C., Sotiropoulos, S.N., Xy, J., Yacoub, E., Ugurbil, K. and Van Essen, D.C. (2016) The Human Connectome Project's neuroimaging approach. Nature Neuroscience 19: 1175-1187
3. Van Essen DC, Smith S, Barch D, Behrens TEJ, Yacoub E, Ugurbil K (2013) The WU-Minn Human Connectome Project: an Overview. Neuroimage 80:62-79.
4. Van Essen, D.C. (1997) A tension-based theory of morphogenesis and compact wiring in the central nervous system. Nature 385:313-318
5. Felleman, D.J. and Van Essen, D.C. (1991) Distributed hierarchical processing in primate cerebral cortex, *Cerebral Cortex*, 1: 1-47

III. Faculty Biography

David Van Essen is the Alumni Endowed Professor of Neurobiology at Washington University in St Louis. He is internationally known for his research on the structure, function, connectivity, evolution, and development of cerebral cortex in humans and nonhuman primates. He has developed powerful methods of computerized brain mapping and has been a pioneer in neuroinformatics and data sharing for two decades. He has written more than 200 peer-reviewed articles and invited publications.

Dr. Van Essen received his undergraduate degree from Caltech and his Ph.D. from Harvard University. He joined the Caltech faculty in 1976 and moved to Washington University in 1992. He has been a leader in two major professional societies, is a Fellow of the American Association for the Advancement of Science, and has received many awards, including several for teaching excellence

ENDOWED LECTURESHIP

SAUL R. KOREY LECTURE

The *Korey Lecture* was established by Dr. Robert D. Terry in honor of Dr. Saul R. Korey, the founder and first Chair of the Department of Neurology at Albert Einstein College of Medicine. Dr. Korey's vision of an interdisciplinary approach to the study of neurological diseases by basic and clinical scientists has inspired generations of colleagues and trainees. Dr. Terry, a close collaborator and colleague of Dr. Korey, was the first recipient of the prestigious *Potamkin Prize for Pick's and Alzheimer's Disease* in 1988, in recognition of his seminal observations of the pathological changes in Alzheimer's disease. Dr. Terry generously contributed a portion of the prize funds to endow the *Korey Lectureship*, to be administered by the American Association of Neuropathologists, with the lecturer to be chosen annually by the President in conjunction with the Nominating Committee and the Chair of the Program Committee. Dr. Terry has summarized the qualities of the Korey lecturer as someone who has "…been an active member of the Association…a working MD or MD/PhD neuropathologist…responsible for diagnostic work as well as teaching and research. The lecture should be aimed at the members of the Association, and the lecturer might well serve as a role model for younger members."

We are pleased to have **Eliezer Masliah** join our list of distinguished speakers.

Year	***Lecturer***	***Title***
1989	Nicholas K. Gonatas	MG-60, a Novel Sialoglycoprotein of Medial Cisternae of the Neuronal Golgi Apparatus: Implications and Applications
1990	Henry M. Wisniewski	Amyloidosis in Alzheimer's Disease and the Spongiform Encephalopathies
1991	Robert D. Terry	Alzheimer's Disease as Seen by a Lucky Morphologist
1992	Henry de Forest Webster	Formation and Regeneration of Myelin
1993	Kunihiko Suzuki	Molecular Genetics of Tay-Sachs and Related Disorders: The Legacy of Saul Korey
1994	*No Lecture*	*XIIth International Congress (Toronto)*
1995	Blas Frangione	Amyloid Genes and Chaperones in Alzheimer Disease
1996	Floyd Gilles	The 3R's of Neuro-oncology – Recording, Reliability and Reporting
1997	Donald L. Price	The Role of Neuropathologists in the Analyses of Models of Neurodegenerative Disease
1998	Sandra H. Bigner	Molecular Genetics of Medulloblastoma
1999	William F. Hickey	Key Participants in the Initiation of Inflammation in the Central Nervous System
2000	Mary E. Case	Neuropathology and Forensic Pathology: A Natural Synergism
2001	Paul H. Kleihues	Molecular Biology of Brain Tumors
2002	James E. Goldman	Astrocytes, Intermediate Filaments, Cellular Stress and Neuropathology
2003	Samuel K. Ludwin	Pathology and Pathogenesis in Multiple Sclerosis
2004	James M. Powers	The Road Not Taken
2005	Bernardino Ghetti	Deciphering Hereditary Presenile Dementias: Neuropathology at the Crossroads of Neuropsychiatry and Molecular Genetics
2006	Donna M. Ferriero	Molecular Mechanisms of Hypoxic-Ischemic Injury in the Developing Nervous System
2007	Dennis W. Dickson	Neuropathology and Genetics of Parkinsonism
2008	David N. Louis	Brain Tumor Classification: Little Steps and Big Jumps

2009	Stephen J. DeArmond	Mechanisms of Neurodegeneration in Prion Disease Originating from the Neuronal Plasma Membrane
2010	Peter C. Burger	A Long-Term Perspective on Pediatric CNS Tumors
2011	Hans H. Goebel	Protein Aggregate Myopathies
2012	Michael Norenberg	Astrocyte Pathobiology
2013	Harry Vinters	Gain and Pain from Cerebral Microvessels – Adventures in Vascular Neuropathology
2014	Thomas J. Montine	Alzheimer's Disease and Related Dementias
2015	Matthew Frosch	Working at the Crossroads of Neurodegeneration and Cerebrovascular Disease
2016	Eileen H. Bigio	The FTLD-ALS Connection
2017	Eliezer Masliah	Disease Modifying Therapeutical Approaches for Synucleinopathies of the Aging Population

SAUL R. KOREY LECTURE

Disease Modifying Therapeutical Approaches for Synucleinopathies of the Aging Population

Time: 4:45 pm – 5:45 pm

Date: Saturday, June 10, 2017

Eliezer Masliah, MD, *Director, Division of Nuerosciences, National Institute on Aging, NIH, Bethesda, MD*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Describe the role of synuclein in pathogenesis of neurodegeneration in parkinsonism and dementia.
2. Explain targeting synuclein in neurodegenerative disorders.
3. Name some potential strategies to reduce synuclein accumulation in treatment of synucleinopathies.

II. Abstract & Relevant References

Parkinson's Disease (PD), Dementia with Lewy bodies (DLB), and Multiple System Atrophy (MSA) are neurodegenerative disorders of the aging, characterized by progressive accumulation of alpha-synuclein. This synaptic protein has also been found to accumulate in other neurodegenerative disorders with dementia such as Alzheimer's disease and jointly are known as synucleinopathies. Together over 6.5 million people are affected in the US and currently no disease modifying therapies are available. Alterations in the balance between clearance, synthesis, aggregation and extracellular release/uptake of alpha-synuclein might play a central role in the pathogenesis. Strategies directed at targeting toxic alpha-synuclein species as well as reducing alpha-synuclein propagation or inflammation associate with alpha-synuclein spreading have been developed in recent years and they might be of therapeutical value at managing synucleonopathies. Strategies to reduce alpha-synuclein expression includes the use of anti-sense and miRNA, to increase clearance and reduce propagation includes compounds and gene therapy targeting lysosomal pathways, anti-aggregation small molecules has shown some promise as well, finally immunotherapy with antibodies against alpha-synuclein have shown to operate by increasing autophagy, microglial clearance, promoting anti-inflammatory responses and reducing propagation. Passive and active immunotherapy approaches are in early stage clinical development and hold considerable promise. The lecture will review the progress to date with these strategies.

References:

1. Reference #1 The many faces of α-synuclein: from structure and toxicity to therapeutic target.Lashuel HA, Overk CR, Oueslati A, Masliah E. Nat Rev Neurosci. 2013 Jan;14(1):38-48. doi: 10.1038/nrn3406. Review.
2. Reference #2 A de novo compound targeting α-synuclein improves deficits in models of Parkinson's disease.Wrasidlo W, Tsigelny IF, Price DL, Dutta G, Rockenstein E, Schwarz TC, Ledolter K, Bonhaus D, Paulino A, Eleuteri S, Skjevik ÅA, Kouznetsova VL, Spencer B, Desplats P, Gonzalez-Ruelas T, Trejo-Morales M, Overk CR, Winter S, Zhu C, Chesselet MF, Meier D, Moessler H, Konrat R, Masliah E. Brain. 2016 Dec;139(Pt 12):3217-3236. Epub 2016 Sep 27.
3. Reference #3 Passive immunization reduces behavioral and neuropathological deficits in an alpha-synuclein transgenic model of Lewy body disease. Masliah E, Rockenstein E, Mante M, Crews L, Spencer B, Adame A, Patrick C, Trejo M, Ubhi K, Rohn TT, Mueller-Steiner S, Seubert P, Barbour R, McConlogue L, Buttini M, Games D, Schenk D. PLoS One. 2011 Apr 29;6(4):e19338. doi: 10.1371/journal.pone.0019338.
4. Reference #4 Therapeutic approaches in Parkinson's disease and related disorders.Valera E, Masliah E. J Neurochem. 2016 Oct;139 Suppl 1:346-352. doi: 10.1111/jnc.13529. Epub 2016 Feb 10. Review.26749150

5. Reference #5 Combination therapies: The next logical Step for the treatment of synucleinopathies?Valera E, Masliah E Mov Disord. 2016 Feb;31(2):225-34. doi: 10.1002/mds.26428. Epub 2015 Sep 21. Review. PMID:26388203

III. Faculty Biography

Dr. Masliah joined the NIA/NIH as Director of the Division of Neurosciences in the summer of 2016. Dr. Masliah received his M.D. from the National Autonomous University of Mexico in 1982. He completed a postgraduate residency training in pathology at the National Institutes of Health in Mexico City in 1986. Following a fellowship in neuropathology and neurodegenerative disorders at the University of California, San Diego (UCSD), before joining NIA he held joint appointments as tenured track Professor at the Departments of Neurosciences and Pathology and as Director of the Autopsy Service at UCSD-Medical Center. As head of UCSD's Experimental Neuropathology Laboratory, he investigated synaptic damage in neurodegenerative disorders such as Alzheimer's disease, Parkinson's disease, dementia with Lewy bodies, multiple system atrophy and AIDS-related dementia. His laboratory developed novel models of neurodegeneration as well as new gene therapies, small molecules and experimental immunotherapies for Alzheimer's disease and Parkinson's disease. Four of the experimental therapeutic approaches developed at his laboratory have now passed Phase I clinical trials. He also directed the neuropathology core of the NIA-supported Shirley-Marcos Alzheimer's Disease Research Center. A prolific author with approximately 800 original research articles, 70 book chapters and dozens of patents, Dr. Masliah has familiarity with NIA as a past member of the NIA National Advisory Council on Aging, the NIA Neuroscience of Aging Study Section, and the Cellular and Molecular Biology of Neurodegeneration Study Section. He has also served as an advisor in the expert panels to revise the criteria for the neuropathological diagnosis of Alzheimer's Disease organized by the NIA and the Alzheimer's Association, at expert meetings and workshops to advise in the use of preclinical models for Alzheimer's Disease and at a series of NIH-hosted Summits on Alzheimer's and related dementias. Masliah served as a member of the Scientific Advisory Board of the Alzheimer's Association from 2010-2016". As Director of the Division of Neurosciences at NIA, Dr. Masliah is responsible for managing the portfolios and providing leadership on NIH sponsored programs dedicated at better understanding brain aging and Alzheimer's Disease, the Division plays a key role in developing the implementation research milestones targeting the ultimate goal of the National Plan to Address Alzheimer's Disease, which calls for the nation to identify effective ways to treat or prevent Alzheimer's disease and related dementias by 2025.

ENDOWED LECTURESHIP

MATTHEW T. MOORE LECTURE

In 1970, Dr. Matthew T. Moore made a contribution to the AANP to establish the Moore Award, which is given annually to recognize the "Best Paper on Clinico-Pathological Correlation Presented at the Annual Meeting." In 1987, Rechelle Fishman, a former patient of Dr. Moore, bequeathed $75,000 to the Moore Award Fund. Dr. Moore requested that this bequest be used to establish a "Rachelle Fishman-Matthew Moore Distinguished Lectureship" (later shortened to just the "*Moore Lectureship*"), which is "to be given by a distinguished lecturer, on a subject which represents the leading edge of advanced research in neuropathological subjects of contemporary interest. The lecture is to take place on the day of the Presidential Address." In 1988, it was decided that this Lectureship would replace the "Distinguished Lectureship" that had been sponsored each year by the Association. The Moore Lecturer is selected annually by the President in conjunction with the Nominating Committee and the Chair of the Program Committee.

We are pleased to have **M. Beatriz S. Lopes** join our list of distinguished speakers.

Year	Lecturer	Title
1990	Robert H. Horvitz	The Genetic Control of GABAergic and Serotonergic Neuronal Differentiation and of Programmed and Pathological Cell Death in a Nematode Nervous System
1991	Charles Janeway	Induction, Mediation and Continuation of Immune Responses
1991	Ramzi S. Contran	Cytokine-Endothelial Interactions in inflammation, Immunity and Vascular Injury
1992	D. Carleton Gajdusek	The genetic Control of Spontaneous Generation of Infectious Amyloids: Kuru-CJD-GSS-Scrapie-BSE
1995	Leroy Hood	Deciphering the Human Genome: Implications for Medicine of the 21st Century
1996	Martin Raff	Programmed Cell Death--Mechanisms and Social Controls
1998	James Eberwine	Single Cell Molecular Neuropathology
1999	Richard T. Johnson	Viral Pathogenesis, an Overview
2000	Seymour Benzer	The Neuropathology of Drosophila
2001	Dennis Choi	Ischemia-Induced Perturbations in Neuronal Ionic Homeostasis
2002	J. William Langston,	MPTP: Its impact on Parkinson's Disease Research
2003	Carolyn C. Meltzer	Future of PET in the Study of Neurological Disease
2004	Henry L. Paulson	Toward Understanding the Pathogenesis of Repeat Expansion Diseases
2005	Peter St. George Hyslop	Molecular Genetics and Biology of Alzheimer Disease Generate Clues for Therapeutics
2006	Keith L. Ligon	Stem and Progenitor Cell Insights into Gliomas: Novel Origins, Markers and Targets
2008	William Mobley	Trafficking Trophic Signals to Prevent Neurodegeneration
2009	Donald W. Cleveland	From Charcot to Lou Gehrig: Mechanisms and Treatment of ALS
2011	Mark Gilbert	RTOG: Clinical Trials and the Increasing Role of Neuropathology
2012	Kevin P. Campbell	Mechanistic and Molecular Insights into the Pathogenesis of Glycosylation – Deficient Muscular Dystrophy
2013	Bradley Hyman	How does Alzheimer's Disease know Neuroanatomy?
2014	David N. Louis	WHO's Next? Guidelines for the Next WHO Classification of Brain Tumors
2015	Eric C. Holland	Brain Tumors in Mouse and Man
2016	Ted M. Dawson	Unlocking the Secrets of Parkinson's
2017	M. Beatriz S. Lopes	An Update of the WHO Classification of Tumors of the Pituitary Gland, 4th Edition

MATTHEW T. MOORE LECTURE

An Update of the WHO Classification of Tumors of the Pituitary Gland, 4th Edition

Time: 8:05 am – 9:00 pm

Date: Sunday, June 11, 2017

M. Beatriz S. Lopes, MD, PhD, *Professor of Pathology and Neurological Surgery, Chief of Neuropathology and Autopsy, Director of Neuropathology Fellowship Program, University of Virginia School of Medicine, Charlottesville, VA*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Recall the main rationale for the changes proposed by the 4th Edition of the WHO Classification of Tumors of the Pituitary Gland
2. Outline the changes in neuroendocrine pituitary tumors classification.
3. Discuss the changes in non-neuroendocrine pituitary tumors classification.
4. Summarize some practical guidelines for the diagnosis of pituitary tumors.

II. Abstract & Relevant References

Tumors of the pituitary gland and sellar region represent approximately 15% of all brain tumors. Several categories of tumors may involve the sellar region, reflecting its complex anatomy. The most common tumors are, by far, the pituitary adenomas representing the third most common primary intracranial tumor in neurosurgical practice, outnumbered only by gliomas and meningiomas. In this lecture, we will review the main changes on the upcoming 4th Edition of the WHO classification of tumors of the pituitary gland emphasizing histopathological and molecular genetics aspects of pituitary neuroendocrine (i.e. adenomas) and non-neuroendocrine tumors involving the pituitary gland.

The majority of pituitary neuroendocrine tumors are pituitary adenomas; pituitary carcinomas are a rare condition. Adenomas are benign neoplasms confined to the sella turcica; however, invasive adenomas are frequent. Pituitary adenomas predominantly affect females between the third and sixth decades; however, no age group is spared. In the pediatric population adenomas are uncommon, and most tumors of childhood are clinically functioning adenomas.

Pituitary adenomas are clinically classified in *clinically functioning adenomas* and the *clinically non-functioning adenomas.* Most adenomas are functioning tumors presenting with a clinical syndrome of hormone excess and these include Prolactin (PRL)-secreting, Growth Hormone (GH)-secreting, Adrenocorticotropic Hormone (ACTH)-secreting, and Thyrotropin-Stimulating Hormone (TSH)-secreting adenomas. About a third of pituitary adenomas are unassociated with either clinical or biochemical evidence of hormone excess. This group includes the gonadotroph adenomas that produce Follicle-Stimulating Hormone (FSH) and Luteinizing Hormone (LH), and the null-cell adenomas. The so-called clinically non-functioning adenomas commonly present with symptoms related to local mass effect such as headaches, neurologic deficits in the cranial nerves including visual field disturbances, and mild hyperprolactinemia due to pituitary stalk compression (the so-called "stalk effect"). In addition, adenomas may be designated as "silent adenomas" when patients do not show any clinical signs or hormonal elevation in the serum but the tumor demonstrates hormone production by immunohistochemistry.

Pituitary adenomas are histopathologically classified by the WHO classification according to the hormone content of the tumor cells as assessed by immunohistochemical stains (Table). This immunohistochemical classification provides significant information for clinical practice. Moreover, the revised WHO classification recognizes the role of transcription factors, mainly Pit-1, SF-1, and Tpit, in tumor differentiation according to cellular lineage(s), in

regulation of specific pituitary hormones, and in possible tumorigenesis. Recognizing this new data, the current WHO classification has adopted the pituitary-cell lineage for designation of the adenomas.

In the previous edition of the WHO Tumor Classification of Endocrine Organs (2004), the pituitary neuroendocrine tumors were divided into *typical adenoma*, *atypical adenoma*, and *carcinoma*. The majority of pituitary neuroendocrine tumors are typical adenomas with bland histological features, infrequent mitotic figures, and a low Ki-67 proliferative index (less than 3%). Atypical adenomas were defined as adenomas with histological features suggestive of an aggressive clinical behavior including elevated mitotic index and a Ki-67 labeling index greater than 3%, and overexpression of the p53 protein by immunohistochemistry. Using these criteria, the incidence of atypical adenoma is relatively variable (2-15%), and its prognostic value not yet established despite more than 10 years of classification. In the upcoming WHO classification, the term of *atypical adenoma* is no longer recommended. However, assessment of the tumor proliferative potential by mitotic count and Ki-67 index, in addition to other clinical parameters such as tumor invasion (by MRI studies and/or intra-operative impression), is strongly recommended in individual cases for consideration of clinically aggressive adenomas.

The classification also recognizes some subtypes of adenomas that show an aggressive behavior; patients with such tumors should be carefully followed clinically. These include the sparsely-granulated somatotroph adenoma, the Crooke's cell adenoma, the silent corticotroph adenoma, and the plurihormonal Pit-1 positive adenoma (previously known as silent subtype III).

The WHO classification reiterates the definition of pituitary carcinomas as tumors demonstrating metastatic spread by either craniospinal dissemination or systemic metastases. Pituitary carcinomas are very rare, comprising less than 1% of all pituitary neoplasms. The majority of carcinomas are hormonally active tumors; the most common are PRL-secreting tumors, followed by ACTH-secreting. Clinically non-functioning carcinomas constitute about 15-20% of the cases including gonadotroph, silent corticotroph, and rarely null cell carcinomas. There are no morphologic criteria to distinguish locally aggressive or even markedly atypical adenomas from pituitary carcinomas when the tumor is still confined to the sella turcica. Morphologic features associated with malignancy including hypercellularity, nuclear and cellular pleomorphism, increased mitotic activity, necrosis, and dural/bony invasion are commonly present but are not necessarily diagnostic of carcinoma.

Although much less frequent than pituitary adenomas, non-neuroendocrine tumors arising in the pituitary are intrinsic tumors of the gland that are important in the differentiation diagnosis of sellar masses. The main entities considered in this group of tumors and discussed in this lecture are the *pituicytomas*, the *granular cell tumors of the neurohypophysis* and the *spindle cell oncocytomas*.

All three tumors are low-grade, non-neuroendocrine neoplasms of the sella that can clinically and radiologically mimic non-functioning pituitary adenomas. Patient's clinical presentation is mostly related to tumor size, with signs and symptoms of compression of adjacent structures and the pituitary stalk including visual disturbances, headaches, hyperprolactinemia and amenorrhea, and fatigue.

Although these three tumor entities have specific histological features, pituicytomas, granular cell tumors and spindle cell oncocytomas show a common immunohistochemical features, diffuse nuclear expression for thyroid transcription factor-1 (TTF-1), similar to that seen in pituicytes, the specialized glia of the neurohypophysis and pituitary stalk. Furthermore, the ultrastructural morphology of these tumors shows similarities with the described variants of normal pituicytes, another indication of a possible common pituicyte-lineage. The current hypothesis is that these non-neuroendocrine pituitary tumors would originate from specific variants of pituicytes, i.e. pituicytomas from the light/major variant, granular cell tumors from the granular variant, and the spindle cell oncocytomas from the oncocytic variant.

References:

1. Alband N, Korbonits M. Familial pituitary tumors. Handb Clin Neurol. 2014;124:339-60. PMID: 25248598
2. Brat DJ, Wesseling P, Fuller GN, Roncaroli F. Pituicytoma. In: Louis DN, Ohgaki H, Wiestler OD, Cavenne C (Eds). WHO Classification of Tumours of the Central Nervous System. Revised 4th Edition. IARC: Lyon, 2016, pp 332-333.
3. Chatzellis E, Alexandraki KI, Androulakis II, Kaltsas G. Aggressive pituitary tumors. Neuroendocrinology. 2015;101(2):87-104. PMID: 25571935
4. Fuller GN, Brat DJ, Wesseling P, Roncaroli F. Granular cell tumour of the sellar region. In: Louis DN, Ohgaki H, Wiestler OD, Cavenne C (Eds). WHO Classification of Tumours of the Central Nervous System. Revised 4th Edition. IARC: Lyon, 2016, pp 329-331.
5. Lopes MB. Growth hormone-secreting adenomas: pathology and cell biology. Neurosurg Focus. 2010;29(4):E2. PMID: 20887127
6. Lopes MBS, Fuller GN, Roncaroli F, Wesseling P. Spindle cell oncocytoma. In: Louis DN, Ohgaki H, Wiestler OD, Cavenne C (Eds). WHO Classification of Tumours of the Central Nervous System. Revised 4th Edition. IARC: Lyon, 2016, pp 334-336.
7. Mete O, Gomez-Hernandez K, Kucharczyk W, Ridout R, Zadeh G, Gentili F, Ezzat S, Asa SL. Silent subtype 3 pituitary adenomas are not always silent and represent poorly differentiated monomorphous plurihormonal Pit-1 lineage adenomas. Mod Pathol. 2016;29(2):131-42. PubMed PMID: 26743473.
8. Nishioka H, Inoshita N, Mete O, Asa SL, Hayashi K, Takeshita A, et al. The complementary role of transcription factors in the accurate diagnosis of clinically nonfunctioning pituitary adenomas. Endocr Pathol. 2015;26(4):349-55. PMID: 26481628
9. Raverot G, Vasiljevic A, Jouanneau E, Trouillas J. A prognostic clinicopathologic classification of pituitary endocrine tumors. Endocrinol Metab Clin North Am. 2015;44(1):11-8. PMID: 25732637.
10. Zhu X, Rosenfeld MG. Transcriptional control of precursor proliferation in the early phases of pituitary development. Curr Opin Genet Dev. 2004 Oct;14(5):567-74. PMID: 15380249.

III. Faculty Biography

M. Beatriz S. Lopes received her MD and PhD degrees at the University of São Paulo in São Paulo, Brazil. She completed her Anatomic Pathology and Neuropathology training at the University of São Paulo followed by a Clinical and Research Neuropathology Fellowship at the University of Virginia in Charlottesville, Virginia, USA, under the supervision of the late Prof. Lucien J. Rubinstein and Prof. Scott R. VandenBerg.

She joined the faculty of the Department of Pathology at the University of Virginia in 1993 where she is now the Harrison Distinguished Teaching Professor of Pathology and Neurological Surgery and the Chief of the Neuropathology and Autopsy Services. Dr. Lopes has been the Director of the ACGME-accredited Neuropathology Fellowship Program at that Institution since 2003.

Her clinical and research expertise is in brain tumors and pituitary pathology with over 140 peer-reviewed manuscripts, 40 textbook chapters, and one book in these areas. She has co-authored several chapters in both the WHO Classification of Brain and Pituitary Gland Tumors since 2000 and 2004, respectively, and was a member of the WHO Consensus and Editorial Meeting for the upcoming 4th Edition of the WHO Classification of Tumors of the Pituitary Gland.

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Award for Meritorious Contributions to Neuropathology

Friday, June 9, 2017
11:30 am – 11:45 am
Honoring Name Ronald C. Kim, MD
Presented by: Joseph E. Parisi, MD

Saturday, June 10, 2017
11:00 am – 11:15 am
Honoring Harry V. Vinters, MD
Presented by: William H. Yong, MD

AWARD FOR MERITORIOUS CONTRIBUTIONS TO NEUROPATHOLOGY

The *Award for Meritorious Contributions to Neuropathology* recognizes a member who has made significant contributions to the advancement of knowledge in neuropathology and provided service to the American Association of Neuropathologists. Each recipient of the award is nominated by the President, in conjunction with the Nominating Committee and with the approval of the Executive Council. The qualities of outstanding scientific achievement and service are embodied in this year's recipients, **Dr. Ronald C. Kim** and **Dr. Harry V. Vinters**. They join the rich roster of distinguished former award recipients.

Year	Recipient
1959	Armando Ferraro
	Arthur Weil
1960	Joseph H. Globus
	George B. Hassin
1968	Abner Wolf
	Paul I. Yakovlev
	Harry M. Zimmerman
1970	Webb E. Haymaker
1971	James W. Kernohan
1972	George A. Jervis
1979	Raymond D. Adams
	David Cowen
	Matthew T. Moore
1981	Richard Lindenberg
1983	Orville T. Bailey
1984	Margaret Murray
1985	Kenneth M. Earle
	Nathan Malamud
	Leon Roizin
1986	Martin G. Netsky
1987	*No Award Presented*
1988	Edward P. Richardson, Jr.
	F. Stephen Vogel
1989	Lucien J. Rubinstein
	Robert D. Terry
1991	Lysia K. S. Forno
1992	John Moossy
	Gabriele M. ZuRhein
1993	Peter W. Lampert
	Elias E. Manuelidis
1994	Murray B. Bornstein
	Samuel P. Hicks
	Lowell W. Lapham
1995	Amico Bignami
	Asao Hirano
1997	Henryk M. Wisniewski
1998	Richard L. Davis
	Wolfgang Zeman

Year	Recipient
1999	Lucy B. Rorke
2000	William R. Markesbery
2001	John J. Kepes
	Henry de Forest Webster
2002	Dikran S. Horoupian
	Fusahiro Ikuta
	Kurt A. Jellinger
2003	Bernardino F. Ghetti
2004	Michael N. Hart
2005	E. Tessa Hedley-Whyte
	Suzanne S. Mirra
2006	Joseph E. Parisi
	Jeannette J. Townsend
2007	James M. Powers
	Cedric S. Raine
2008	Kinuko Suzuki
	Margaret G. Norman
2009	Peter C. Burger
	Pierluigi Gambetti
	Nicholas K. Gonatas
2010	Stephen J. DeArmond
	Samuel K. Ludwin
2011	William W. Schlaepfer
	Leroy R. Sharer
2012	Bernd W. Scheithauer
	Donald L. Price
2013	Reid Heffner
	Dawna Armstrong
2014	Floyd Gilles
	Françoise Gray
2015	John Q. Trojanowski
	Bette K. Kleinschmidt-DeMasters
2016	Dennis W. Dickson
	Barbara J. Crain
2017	Ronald C. Kim
	Harry V. Vinters

AWARD FOR MERITORIOUS CONTRIBUTIONS TO NEUROPATHOLOGY

Biography: Ronald C. Kim, MD

I was born in New York City and grew up in Washington, DC.

After Japan attacked Pearl Harbor my father, a graduate student from Korea who had attended high school and college in Japan, was hired by the U.S. State Department to help interpret some of the documents seized from the offices of Japanese companies. There were only a handful of individuals in this country who were not of Japanese descent and who were fluent in Japanese and had a deep understanding of Japanese culture. After the war ended my father, who had never intended to reside permanently in the U.S., waited for Korea to recover so that he could return there with his family but; because the Korean War broke out a few years later, he and his children (me and my younger sister) ended up living here permanently.

My father insisted that I enter medical school and, after graduating from Jefferson Medical College, I was unsure what specialty to choose, and I did a rotating internship at San Francisco General Hospital. While there, I was drafted into the U.S. Air Force, where I spent two years, mostly at Kadena AFB in Okinawa. While there I noted that a number of servicemen who had married local women and had children under a year old were returning stateside, so I exempted them from vaccination. An Air Force general at Hickham AFB in Hawaii wanted to know who at Kadena was exempting these infants from vaccination, and I was called into my commanding officer's office to explain why I had done what I had done. I responded with a paper in *Pediatric Clinics of North America* that indicated that the risk of post-vaccinal acute disseminated encephalomyelitis was lowest if vaccination was undertaken at 12 months of age. I left his office thinking that I would be court-martialed and that my medical career was over but, as it turned out, he accepted my explanation.

After discharge from the USAF, I entered the Boston VAMC pathology residency training program, initially directed by Dr. Roger Coté (of SNOMED fame) and subsequently by Dr. George Kenneth Mallory (of Mallory-Weiss fame). Two neuropathologists there [Dr. Jacques B. Lamarche and Dr. Remedios ("Rose") Rosales] talked me into training in neuropathology, so I obtained an NIH fellowship to train in neuropathology at the College of Physicians & Surgeons of Columbia University, then under the direction of Dr. David Cowen.

While at P&S, I attended the twice-weekly morning lectures of Dr. Abner Wolf, at that time Professor Emeritus and Editor-in-Chief of the *Journal of Neuropathology & Experimental Pathology*. Dr. Wolf, who counted Dr. Alfons Jakob among his teachers, asked me to serve as his informal editorial assistant. For example, when the Editorial Board requested revision of a submitted manuscript and the manuscript was returned to the *JNEN* office, Dr. Wolf would ask me to review it in order to determine if the author(s) had complied with the Editorial Board's requests. I was also assigned the task of rendering into readable English any accepted manuscripts that were not properly written. I would estimate that, in the four years that I was at P&S, I spent at least 6-8 hours/week working for the *Journal*.

Despite the hours I invested on the *Journal's* behalf, I felt that the experience was beneficial. I got to know Dr. Wolf much better than my fellow trainees in neuropathology did. Although I had heard from others that he was a difficult man with whom to interact, I found him to be a very pleasant, forthright man with a real sense of humor, and he thoroughly enjoyed regaling me with his off-color jokes. As President of the International Society of Neuropathology he offered to pay my expenses to travel to the Society meeting in Budapest but, having a 10-month-old daughter, I respectfully declined.

Dr. Cowen, a patient and dedicated teacher, was a superb diagnostician. He himself had trained with Dr. Wolf. When I entered the program, they had been together for 40 years. I never saw Dr. Cowen lose his temper despite the travails visited upon him by some of his trainees. A lifelong New York City bachelor and amateur naturalist, he was fond of spending time wandering in forests of northern New England.

After completing my neuropathology training, I entered a year-long NIH fellowship in neuroanatomy with Dr. Malcolm Carpenter, undertaking autoradiographic studies on globus pallidus and nigrothalamic projections in rhesus monkeys.

My first faculty position was in the Department of Pathology at SUNY-Upstate Medical Center in Syracuse where, together with my colleague Dr. George Collins, we were able to establish a training program in neuropathology.
Because of personal issues related to my parents' well-being, I re-located to the Long Beach VAMC and the University of California Irvine, having been recruited jointly with Dr. Byung-Ho ("Ben") Choi. Dr. Choi, who had NIH funding to study the effects of mercury on the developing fetal brain, was a solid and creative neuroscientist and one of the most personable individuals I have ever known, and I considered myself most fortunate to have been one of his co-investigators.

I was assigned, among other things, to teach residents in pathology, neurology, and neurosurgery at UCI Medical Center and at the Long Beach VA and Long Beach Memorial Medical Centers. For over 20 years, I gave monthly CPCs at each of these institutions. I was also responsible, for 15 years or so, for coordinating the Pathology Grand Rounds at LBVAMC.

It was here that I first encountered the enormous LBVAMC Spinal Cord Injury (SCI) unit. Whereas most of the traumatic SCI autopsies were fairly straightforward, the non-traumatic myelopathies were not, and I had to struggle mightily to solve some of these cases. Indeed, some of these cases I could not solve. My presentations at national meetings reflected some of these problems. I became very interested in spinal vascular disease, especially venous infarction of the spinal cord. I also encountered a number of "one-of-a-kind" myelopathies, owing to the fact that the LBVAMC SCI unit was an international referral center for patients with spinal cord disease.

In the late 1980s, Dr. Joseph Parisi, then Director of Neuropathology at the Armed Forces Institute of Pathology, invited me to participate in their annual week-long course in neuropathology and to talk about neuropathology that was specific to the spinal cord (trauma, vascular disease, and miscellaneous myelopathies), and I have been doing so ever since. I am most grateful to Joe for having given me the opportunity to participate in this course, currently known as the Kenneth M. Earle Neuropathology Review Course (sponsored by the Inova Fairfax Medical Center in Fairfax, VA).

Because of my close relationship with successive faculty SCI Chiefs, I was asked to join the American Paraplegia Society where I have, for many years, participated in their Spinal Cord Medicine course. The organization is now the Academy for Spinal Cord Professionals, and the course is now sponsored by the Cleveland Clinic.

In 1984, the NIH funded five Alzheimer Disease Research Centers (ADRCs), including one established jointly by USC and UCI. The USC/UCI ADRC continued jointly until the 1990s, at which time the two institutions were asked to establish separate ADRCs, which we did. Dr. Choi and I were both involved in the Neuropathology Core. After Dr. Choi retired, I was the sole neuropathologist.

From 1989-94 and 1999-2001 I served as Director of the UCI Pathology Residency Training Program, and from 1999-2014 I served as Director of the UCI Course in General & Systemic Pathology which, for many years, received awards for being the outstanding course in the second-year medical school curriculum.

I have also served as co-investigator on the NIH-funded California NeuroAids Tissue Network and as co-investigator on an NIH grant to Dr. Mark Fisher (Chair Emeritus of Neurology) focused on hemorheological factors in cerebral ischemia.

I have served on the AANP Membership Committee (1984-86, 1995-97), Awards Committee (1990-92, 2003-04), and Professional Affairs Committee (1992-94), and I have served as a member of the Editorial Board of the *JNEN* (1997-2004).

I have two daughters, Helen Sujin (born in New York City in 1972) and Sujung (born in Syracuse in 1977) and have been married to my wife Youngja for 46 years. We have three grandchildren (Gabriela, 12 years; Owen, 9½ years; and Riley, 2½ years).

I have been a lifelong fan of classical music and am particularly fond of opera, especially German and Russian opera.

Dr. Ronald C. Kim "Contributions to the Field", written by Joseph E. Parisi, MD

Ronald C. Kim was born in New York City and had his formative years in the Washington, DC metro area. He attended Harvard College (Boston, MA) where he majored in Physics. He obtained his M.D. degree from Jefferson Medical College (Philadelphia, PA). A rotating internship at San Francisco General Hospital was interrupted by a two-year stint in the U.S. Air Force as a General Medical Officer at Kadena AFB in Okinawa. His interest in pathology took him back to Boston where he entered pathology residency training at the Boston VAMC, initially directed by Dr. Roger Coté (of SNOMED fame) and subsequently by Dr. George Kenneth Mallory (of Mallory-Weiss fame). His exposure to two giants in neuropathology (Drs. Jacques Lamarche and Remedios ("Rose") Rosales) inspired him to pursue neuropathology training at the College of Physicians & Surgeons of Columbia University (New York, NY), then under the direction of Dr. David Cowen. At P&S, his keen writing and editorial talents were recognized by Dr. Abner Wolf, at that time Editor-in-Chief of the *Journal of Neuropathology & Experimental Pathology*, who invited him to serve as his informal editorial assistant, an extraordinary experience for a young trainee that no doubt contributed to his encyclopedic knowledge of neuropathology. Following neuropathology training, he completed a NIH fellowship in neuroanatomy with Dr. Malcolm Carpenter, during which he performed autoradiographic studies on globus pallidus and nigrothalamic projections in rhesus monkeys.

Dr. Kim's first faculty position was in the Department of Pathology at SUNY-Upstate Medical Center (Syracuse, NY), where he and his colleague Dr. George Collins established a superb neuropathology service and training program.

His subsequent recruitment to the Long Beach VAMC and the University of California Irvine along with Dr. Byung-Ho ("Ben") Choi, resulted in another longstanding and successful collaboration.
At the VA and UCI, he quickly ascended through the academic ranks to become Health Sciences Clinical Professor of Pathology and Neurology, and Consultant in Neuropathology.
Dr. Kim's expertise as a superb diagnostic neuropathologist, teacher and collaborator is legendary. He is especially well known as an expert on spinal cord disorders, particularly spinal vascular disease and unusual myelopathies, the study of which has been a focus of much of his professional career. His research and observations have provided many important insights into our understanding of the pathogenesis of many of these devastating diseases.

He is a superb teacher, the recipient of several teaching awards and a widely sought guest lecturer. His uncanny ability to present complicated concepts in a clear and logical fashion consistently wins the acclaim of students, trainees and peers. For many years, he has been faculty at numerous specialty courses, including the Kenneth Earle Neuropathology Review and the Spinal Cord Medicine Courses. In addition, he has served as Director of the UCI Pathology Residency Training Program, and as Director of the UCI Course in General & Systemic Pathology.

Dr. Kim currently is co-director of the ADRC Neuropathology Core at UCI. He also has served as co-investigator on the NIH-funded California NeuroAids Tissue Network and an NIH study on flow properties of blood elements in cerebral ischemia.

He is the author of over 200 scientific publications that cover a wide range of topics in neuropathology. He has been a member of numerous professional advisory and organizing committees. He has been a long-time contributor to our annual meetings and organization, having served the AANP by membership on the JNEN editorial board, as well as the membership, awards, and professional affairs committees.

In addition to his recognized neuropathology expertise, Dr. Kim is the consummate renaissance and family man. He has a limitless curiosity and extensive range of intellectual interests. His passions include reading, music, and his family. He especially enjoys reading about history and music. He loves classical music and is particularly fond of German and Russian opera. His favorite composers are Beethoven and Wagner. He and his wonderful wife Youngja are enormously proud of their two daughters, Helen Sujin and Sujung and their families, and enjoy their three grandchildren, Gabriela (12 years), Owen (9½ years) and Riley (2½ years).

I was enormously fortunate to have been the first trainee under Drs. Collins and Kim. I learned a great deal, not only about neuropathology, but also about life. Dr Kim in particular taught me to how to critically evaluate an article and the importance of self-challenge, skills that I have tried to impart to my trainees.

Dr. Kim is knowledgeable, generous, kind and understanding, someone I am proud to consider my friend, mentor and colleague. Dr Kim embodies a spirit of excellence and excitement for neuropathology, and the personal qualities that represent our profession at its very best. These make him a very worthy recipient of the Award for Meritorious Service.

Biography: Harry V. Vinters

Harry V. Vinters grew up in Port Arthur (now Thunder Bay), Ontario, graduated from University College (Toronto) and the University of Toronto Medical School (1976), interned at the University of Alberta Hospitals (Edmonton, Canada) and trained in Neurology and Neuropathology at the University of Western Ontario Hospitals in London, Canada (1977-81).

At UWO, his mentors were John Kaufmann, Joe Gilbert and Melvyn Ball. He subsequently trained in Pediatric Neuropathology at Vancouver General Hospital in Vancouver, British Columbia, with Dr. Margaret Norman (1981). He completed a research fellowship, focusing on the neurobiology and cell biology of the blood-brain barrier and cerebral microvascular disease, with Dr. Michael N. Hart & Dr. Pasquale A. Cancilla at the University of Iowa in Iowa City, moving with Pat Cancilla to UCLA in 1982. Dr. Vinters was briefly (1984-85) on faculty at the University of Western Ontario (recently renamed Western University) in London, Canada, with a joint appointment in Pathology and Clinical Neurological Sciences. He has been on faculty at the David Geffen School of Medicine at UCLA in Los Angeles since 1985. He held the Daljit S. & Elaine Sarkaria Chair in Diagnostic Medicine (2005-2011), and is currently Distinguished Professor (Emeritus) of Pathology & Laboratory Medicine, and Neurology. He was Chief of the Section of Neuropathology at Ronald Reagan-UCLA Medical Center and the David Geffen School of Medicine (at UCLA) from 1993 to June, 2017. He is grateful for the amazing mentorship and support he has received from the individuals named above, and had the remarkable good fortune to work with two visionaries in the field of neurodegenerative diseases, Dr. Jeff Cummings and Dr. Helena C. Chui.

Dr. Vinters has published more than 500 articles, reviews and book chapters on various aspects of neuropathology, ranging from its clinical aspects to issues of molecular pathogenesis, tissue culture and animal models. He has also co-authored or edited six books, including all three editions of the widely-used ***Neuropathology—a Reference Text of CNS Pathology*** (Mosby, 3d edition, 2013). In addition to his clinical and teaching activities, he has had active research programs in several areas, including vascular dementias and the vascular component of Alzheimer disease (especially mediated through amyloid/congophilic angiopathy), neuropathologic substrates of intractable pediatric epilepsy, stroke and cerebrovascular disease—this work incorporates translational studies and investigation of animal models. In the early days of the AIDS epidemic, he had an active program in, and was among the first to characterize, the neurologic complications of HIV infection. In 1987, his laboratory contributed to discovery of one of the first 'neurotropic' strains of HIV (HIV-JR). He was the recipient (in 2002) of the Research Award of the Alzheimer's Association of Los Angeles, Riverside and San Bernardino Counties. He served as Editor-in-Chief of ***'Brain Pathology'***, from 2000-2006. In 2004-2005, he served as President of the American Association of Neuropathologists. Dr. Vinters' laboratory has hosted numerous international scholars and trainees, including Dr. Orestes Solis (from UST/ Manila), Dr. Remco Natte (Leiden University Medical Center, the Netherlands), and Dr. Sung Hye Park (Korea)—all of whom have gone on to distinguished careers in their home countries. He currently serves on several editorial boards of major scientific journals, including ***Neuropathology, Neuropathology & Applied Neurobiology, and Human Pathology***. He lives in (and greatly enjoys!) the eclectic Los Angeles beach community of Venice, California, where he has lived since 1989.

Dr. Harry V. Vinters "Contributions to the Field", written by William H. Yong, MD

Dr. Harry Valdis Vinters, who hails from icy Thunder Bay in Canada, graduated with his medical degree from the University of Toronto in 1976. He completed a residency in neuropathology at the University of Western Ontario Hospitals under the eminent Drs. John Kaufmann and Joe Gilbert. He subsequently trained in pediatric neuropathology with Dr. Margaret Norman at Vancouver General Hospital. Dr. Vinters became a Fellow of the Royal College of Physicians Canada and obtained certifications in neuropathology for both Canada and the United States. At the University of Iowa, he began a research fellowship in neuropathology under Drs. Michael Hart and Pasquale Cancilla. When Dr. Cancilla moved to UCLA, Dr. Vinters joined him to complete his fellowship. Of note, Drs. Norman, Hart and Cancilla are past recipients of the American Association of Neuropathologists (AANP) Meritorious Award as well. Dr. Vinters started in a faculty position at the University of Western Ontario and then was recruited to the University of California, Los Angeles (UCLA) to be an Assistant Professor. He rose easily and inexorably through the academic ranks to become a Distinguished Professor of Pathology and Laboratory Medicine. He was the Daljit S. and Elaine Sarkaria Chair in Diagnostic Medicine of the UCLA School of Medicine from 2006 to 2011 and served as Chief of Neuropathology at UCLA from 1993 to 2016. He served as Editor in Chief of Brain Pathology from 2001 to 2006 and as President of the AANP from 2004 to 2005. He is now emeritus faculty, but works part time- still sharing his eagle-sharp eyes and deep as the Mariana Trench expertise.

His curriculum vitae includes over 600 publications spanning a broad range of neuropathology topics. His seven days a week and dawn to late night work ethic indubitably contributed to this prodigious output. During the early fear-filled days of the AIDS epidemic, he performed many autopsies when many declined to do so and published extensively on the nervous system effects of AIDS. His

contributions to epilepsy neuropathology illuminating the clinical and pathologic features of Rasmussen's encephalitis, cortical dysplasia and tuberous sclerosis are well known. His other major area of study has been cerebrovascular neuropathology including

vascular malformations as well as cerebral amyloid angiopathy and other microangiopathies. Among other awards, he is the 2017 recipient of the Alfred Meyer Medal bestowed by the British Neuropathological Society for his contributions to neuropathology, in particular the neuropathology of vascular dementia and vascular cognitive impairment.

Dr. Vinters has many strong international ties. Though he made a home in balmy Venice Beach where he often hosts convivial celebrations for the neuropathology division, he frequently travels north to visit his Canadian family, friends, and colleagues. While an AANP member since 1984, he has been an even longer standing member of the Canadian Association of Neuropathologists (since 1982). A little known fact is that he has Latvian roots, which explains how he was able to give an entire neuropathology lecture in the Latvian language at a Baltic meeting! In 1997, he was inducted as a member of the Faculty of Pathology, Royal College of Physicians, Ireland. Dr. Vinters also undertook visiting professorships at the University of Leiden in the Netherlands and the University of Santo Tomas in the Philippines. He has a laudable record for hosting or training students and physicians from the United States and abroad. A number of his undergraduates have gone on to medical careers. He has chaired approximately 10 Ph.D. Committees and served on scores more. He has mentored many post-doctoral research fellows as well as clinical neuropathology fellows. Drs. Michael Farrell (Ireland), Sung-Hye Park (Korea), Hajime Miyata (Japan), Paul Mischel (U.S.A.), and myself are only a few of the colleagues or alumni blessed to have worked with this stalwart friend, incredible mentor, generous colleague, and giant intellect whose considerable contributions to our field and our association are well worthy of the Meritorious Award.

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

What Every Neuropathologist Needs to Know

Saturday, June 10, 2017

WHAT EVERY NEUROPATHOLOGIST NEEDS TO KNOW

The Updated WHO Classification and Pediatric Neuro-Oncology

Time: 10:30 am – 11:00 am

David W. Ellison, MD, PhD, *St. Jude Children's Research Hospital, Memphis, TN*

I. Learning Objectives:

At the end of this activity, learners should be able to:

1. Explain the clinical utility of a molecular classification of pediatric CNS tumors.
2. Describe how molecular and histopathological classifications of medulloblastoma are integrated.
3. Recognize technologies that demonstrate the molecular alterations of pediatric CNS tumors in FFPE tissues.

II. Abstract and Relevant Resources

The molecular characteristics of pediatric CNS tumors feature strongly in the updated WHO classification of 2016. In particular, molecular groups of medulloblastoma are now used alongside histopathologic variants in a 'tiered' approach to integrated diagnosis. Other notable new entities to impact the neuropathologist's diagnostic practice with respect to pediatric tumors include: ependymoma, RELA fusion-positive, and the diffuse midline glioma, H3 K27M-mutant. Some of the challenges that the field faces in this evolution to a molecular classification are: (i) keeping the classification current with demand from neuro-oncologists to report clinically useful molecular alterations and (ii) optimizing methods for detecting the molecular alterations described in the WHO classification in formalin fixed, paraffin embedded tissue.

References:

1. Louis, D. N., et al. (2016). "The 2016 World Health Organization Classification of Tumors of the Central Nervous System: a summary." Acta Neuropathol **131**(6): 803-820.
2. Chiang, J. C. and D. W. Ellison (2017). "Molecular pathology of paediatric central nervous system tumours." J Pathol **241**(2): 159-172.
3. Taylor, M. D., et al. (2012). "Molecular subgroups of medulloblastoma: the current consensus." Acta Neuropathol **123**(4): 465-472.
4. Korshunov, A., et al. (2014). "Embryonal tumor with abundant neuropil and true rosettes (ETANTR), ependymoblastoma, and medulloepithelioma share molecular similarity and comprise a single clinicopathological entity." Acta Neuropathol **128**(2): 279-289.
5. Wu, G., et al. (2014). "The genomic landscape of diffuse intrinsic pontine glioma and pediatric non-brainstem high-grade glioma." Nature genetics **46**(5): 444-450.
6. Broniscer, A., et al. (2014). "Clinical, radiological, histological and molecular characteristics of paediatric epithelioid glioblastoma." Neuropathol Appl Neurobiol **40**(3): 327-336.
7. Zhang, J., et al. (2013). "Whole-genome sequencing identifies genetic alterations in pediatric low-grade gliomas." Nature genetics **45**(6): 602-612.
8. Parker, M., et al. (2014). "C11orf95-RELA fusions drive oncogenic NF-kappaB signalling in ependymoma." Nature **506**(7489): 451-455.

III. Faculty Biography:

David Ellison is Chair of Pathology, Director of Neuropathology, and Joan and Roy Gignac Endowed Chair of Pathology and Laboratory Medicine at St. Jude Children's Research Hospital. After studying for an M.A. in neuropsychology at the University of Oxford, he graduated in medicine from the University of Cambridge, UK. Clinical training in general medicine and neurology preceded a Wellcome Trust research fellowship at Harvard

University / Massachusetts General Hospital, where neurotransmitter alterations in neurodegenerative diseases were the focus of his research in the laboratories of Dr. Flint Beal and Dr. E. P. Richardson.

Encouraged by his studies in Boston to train in neuropathology, Dr. Ellison returned to England and positions in Oxford and Southampton. Initially, he took a faculty position in Southampton as a clinician–educator, but a developing academic interest in pediatric brain tumors led to appointments as Reader, then Professor, in Neuropathology at the University of Newcastle and subsequent recruitment to St. Jude.

Dr. Ellison is a Fellow of the Royal Colleges of Physicians, Pathologists, and Paediatrics and Child Health. Current research interests include the molecular classification of low-grade gliomas and ependymomas and the development of clinical genomics and other precision medicine methodologies. Dr. Ellison has been an editor of two leading neuropathology textbooks: Greenfield's Neuropathology and Neuropathology, a reference text of CNS pathology. He was a senior advisor / editor for the 2016 updated WHO classification of CNS tumors and is a member of the IARC/WHO Committee for the ICD-O: Tumors of the Central Nervous System.

WHAT EVERY NEUROPATHOLOGIST NEEDS TO KNOW

Basic Electron Microscopy of Muscle

Time: 4:15 pm – 4:45 pm

Robert E. Mrak, MD, PhD, *Professor and Chairman, University of Toledo College of Medicine, Toledo, OH*

I. Learning Objectives:

At the end of this activity, learners should be able to:

1. Identify diseases for which electron microscopy of muscle is a helpful diagnostic tool
2. Explain the proper technique for muscle biopsy to include electron microscopy
3. Describe the ultrastructural features that are of diagnostic utility in muscle biopsies

II. Abstract and Relevant Resources

Electron microscopy continues to be a useful tool in the diagnostic evaluation of muscle biopsies, even as resident training in this classic pathology tool and availability of the technique declines. Ultrastructural results are best when a second, smaller specimen is taken at the time of original biopsy, clamped in a special muscle biopsy clamp, and promptly fixed in glutaraldehyde. However, useful information can also be obtained from unclamped or formalin-fixed muscle tissue. Ultrastructural examination is useful in the setting of congenital myopathies, where diagnostic features such as nemaline rods, structured and unstructured cores, and other less common abnormalities may be subtle histologically. Other genetic diseases in which there are characteristic ultrastructural features include myofibrillar myopathies (desminopathies), neonatal myotonic dystrophy (with increased numbers of satellite cells), and oculopharyngeal muscular dystrophy (with characteristic filamentous intranuclear inclusions). Mitochondrial diseases frequently show ultrastructural abnormalities, ranging from striking paracrystalline ("parking lot") inclusions, to more subtle alterations of mitochondrial shape, size, number, or internal structure. Diagnoses of glycogenoses and lipidoses can be supported by ultrastuctural examination, and electron microscopy of Pompe's disease, with membrane-bound glycogen accumulation, can be diagnostic. Additional diseases with helpful or diagnostic ultrastructural features include inclusion body myositis (sarcoplasmic degeneration with amorphous and myelinoid inclusions, and intranuclear filaments), acute quadriplegic myopathy (AKA intensive care myopathy; with loss of myosin filaments), and dermatomyositis (with tubuloreticular inclusions).

References:

1. K Kyriacou, B Kassianides, A Hadjisavvas, L Middleton, T Kyriakides (1997) The role of electron microscopy in the diagnosis of nonneoplastic muscle diseases. Ultrastuctural Pathol 21:243-252
2. E Bonilla, K Tanji (1998) Ultrastructural alterations in encephalomyopathies of mitochondrial origin. BioFactors 7:231–236
3. RE Mrak (2002) The 'Big Eye' in the 21st century: the role of electron microscopy in modern diagnostic neuropathology. J Neuropathol Exp Neurol 61:1027-1039, 2002
4. CA Sewry (2002) Mini-symposium: Diagnostic electron microscopy. Electron microscopy of human skeletal muscle: role in diagnosis. Current Diagnostic Pathol 8:225-231.
5. HB Sarnat, J. Marín-García (2005) Pathology of mitochondrial encephalomyopathies. Can J Neurol Sci 32:152-166
6. G Cenacchi, L Tarantino, A Corbu, LB De Giorgi, M Fanin, E Pegoraro, L Fulizio, C Angelini, G Martinelli (2007) The contemporary role of electron microscopy in neuromuscular pathology. Basic Applied Myology 17:167-171

7. E Curtis, C Sewry (2013) Chapter 3: Electron microscopy in skeletal muscle pathology. In: Diagnostic Electron Microscopy: A Practical Guide to Interpretation and Technique, 1st Ed. Edited by J. W. Stirling, A. Curry, B. Eyden. John Wiley & Sons, Ltd.
8. HH Goebel, W Stenzel (2013) Ultrastructural myopathology in the molecular era. Ultrastructural Pathol 37: 328–331
9. Olive M, Kley RA, Goldfarb LG (2013) Myofibrillar myopathies: new developments. Current Opinion Neurol 26:527-35

III. **Faculty Biography:**

Robert Mrak is a Professor and Chairman of Pathology at the University of Toledo College of Medicine. His training includes a BS from the University of California at Davis in Mathematics; MD and PhD from the University of California at Davis; residency at Vanderbilt University in Anatomic Pathology; and fellowship at Vanderbilt University in Molecular Biology and Neuropathology. Dr. Mrak is the Founder and Editor-in-Chief of the Journal of Neuroinflammation and is currently also a member of the Association of Pathology Chairs and chair of the Practice & Management Committee.

Selected Recent Publications:

1. Chen Y, Neve RN, Zheng H, Griffin WTS, Barger SW, Mrak RE. Cycle on wheels: Is APP key to the AppBp1 pathway? Austin Alzheimer's and Parkinson's Disease 1(2):7, 2014. PMID: 25568892
2. Thal DR, von Arnim CAF, Griffin WST, Mrak RE, Walker L, Attems J, Arzberger T. Frontotemporal lobar degeneration FTLD-tau: preclinical lesions, vascular, and Alzheimer-related co-pathologies. J Neural Transm 122:1007-18, 2015 PMID: 25556950
3. Zawada MW, Mrak RE, Biedermann J, Palmer QD, Gentleman SM, Aboud O, Griffin W. Loss of angiotensin II receptor expression in dopamine neurons in Parkinson's disease correlates with pathological progression and is accompanied by increases in Nox4- and 8-OH guanosine-related nucleic acid oxidation and caspase-3 activation. Acta Neuropathologica Communications 3:9, 2015. DOI: 10.1186/s40478-015-0189-z PMID: 25645462
4. Aboud O, Parcon PA, DeWall M, Liu L, Mrak RE, Griffin ST. Aging, Alzheimer's, and APOE genotype influence the expression and neuronal distribution patterns of microtubule motor protein dynactin-P50. Front Cell Neurosci 9:103, 2015. doi: 10.3389/fncel.2015.00103. PMID: 25859183
5. Romanovsky D, Mrak RE, Dobretsov M. Age-dependent decline in density of human nerve and spinal ganglia neurons expressing the alpha-3 isoform of Na/K-ATPase. Neuroscience 310:342-53, 2015. PMID: 26386295

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Diagnostic Slide Session

Saturday, June 10, 2017

Learning Objectives:

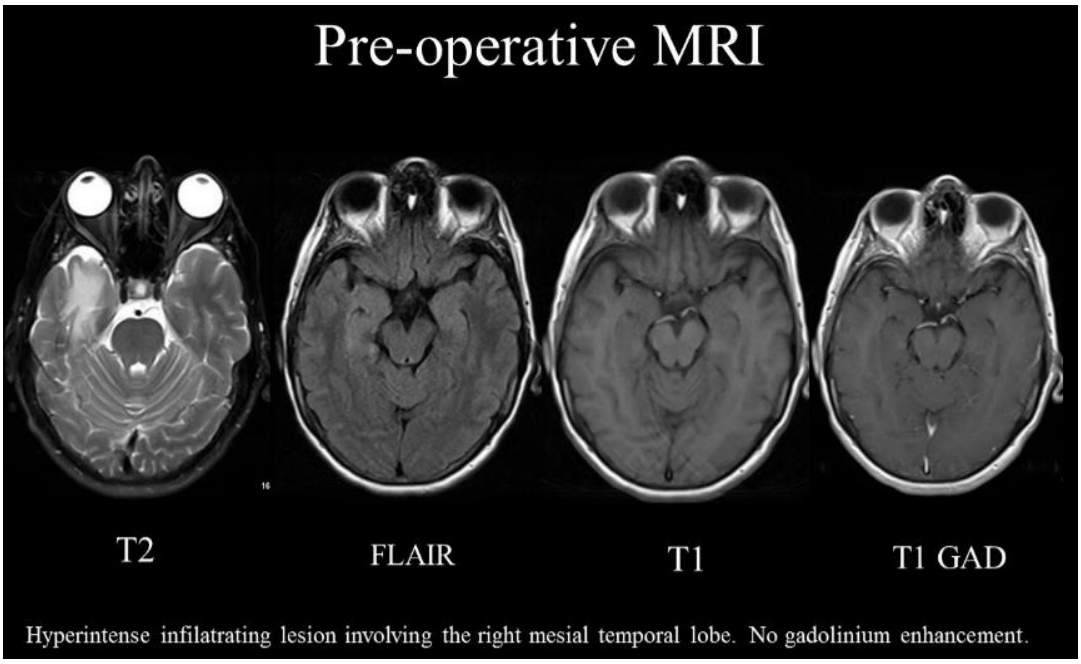
1. *Explain unusual tumors or patterns of tumor involvement in the nervous system, and their clinical, pathologic and molecular features.*
2. *Recognize complications of therapeutic irradiation.*
3. *Identify unusual nervous system infections and inflammatory reactions.*
4. *Outline pathologic characteristics and genetic underpinnings of syndromes including nervous system abnormalities.*
5. *Generate a differential diagnosis for neuromuscular disease in infants.*

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-1

Submitted By:
Natalya Hakim, Janna Neltner, Dianne Wilson, Peter Nelson, Vanessa D. Smith
University of Kentucky, Department of Pathology, 800 Rose Street, Lexington KY 40536

Clinical History:
The patient is a 51 year old female found to have a right temporal nonenhancing lesion upon workup for episodes of subacute progressive confusion and new onset seizures over the past two months. She also reports new frequent headaches and blurry vision. Her past medical history is significant for restless leg syndrome, anxiety and depression. Neurologic examination did not demonstrate any focal deficits. An MRI of the head demonstrated right temporal lobe cortical thickening with T2 hyperintense signal involving both the cortex and deep parenchyma, consistent with an infiltrating lesion. No enhancement was noted. The patient underwent a right temporal lobectomy, sparing the hippocampus and medial structures, with subtotal excision of the lesion.

Material Submitted: 1 H&E slide and pre-operative images of MRI head with and without contrast



Points for Discussion:
1. Clinical and radiographic presentation
2. Histologic and immunohistochemical characteristics
3. Prognosis

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-2

Submitted By:
Drew Williamson, C. Badve, A. Hdeib, L. Rogers, M. Couce, M. Cohen
University Hospitals Cleveland Medical Center, Cleveland Ohio

Clinical History:
November 2002: Radical orchiectomy demonstrating non-seminomatous germ cell tumor with yolk sac, embryonal, and syncytiotrophoblasts elements.

June 2003: 3 cm left frontal lobe metastasis resected followed by 40Gy whole brain radiation.

October 2003: Residual left frontal lobe tumor with new brain lesions treated with gamma knife radiosurgery. Seizures well controlled with Keppra.

April 2016: Left-sided headache, difficulty speaking, and progressive memory impairment. FDG-MRI-PET scan demonstrates left frontal and temporal mildly increased uptake consistent with viable tumor. EEG negative for seizures. Stereotactic biopsy demonstrates histopathological features consistent with radiation necrosis. Patient was not deemed a candidate for medical therapies to address the radiation necrosis and was observed.

November 2016: Breakthrough seizure activity with increased left anterior frontal lobe gyral swelling and enhancement compared with MRI 3 months previously. Area of greatest cortical enhancement biopsied.

Material submitted: H&E section from November 2016 biopsy

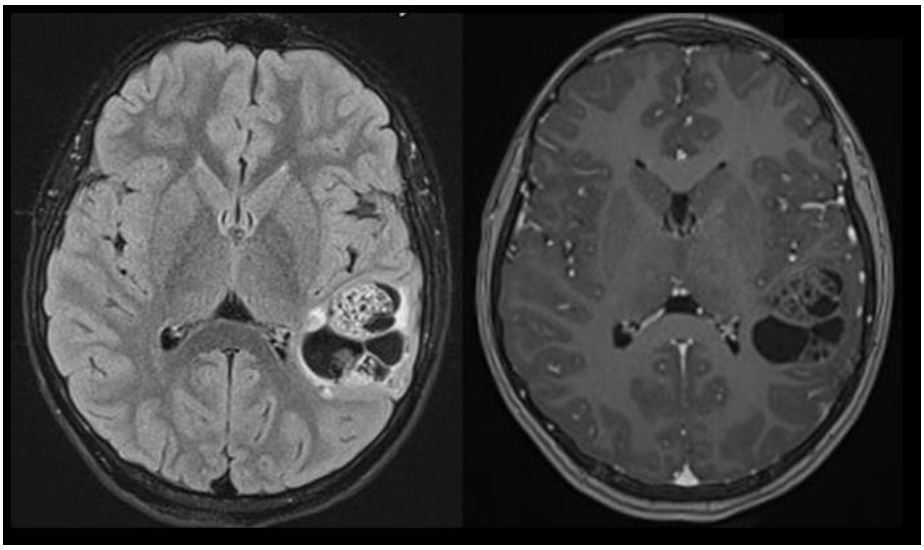
Points for discussion:
1. Provide 3 possible diagnoses in order of probability.
2. Postulate 2 possible pathogenetic mechanisms responsible for the cytological abnormalities seen within the lesion.
3. Name 2 eminent California neuropathologists on whose shoulders we are now standing.

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-3

Submitted By:
Andrew F. Gao[1], Lili-Naz Hazrati[1,2], and Cynthia E. Hawkins[1,2]
1. Department of Laboratory Medicine and Pathobiology, University of Toronto, Toronto, ON, Canada
2. Division of Pathology, Department of Pediatric Laboratory Medicine, Hospital for Sick Children, 555 University Avenue, Toronto, ON, Canada, M5G 1X8

Clinical History:
This 9-year-old girl from El Salvador presented with partial seizure and occasional headaches. A brain MRI showed a large complex solid-cystic cortical and subcortical mass lesion measuring 7.1 x 4.2 x 3.6 cm within the left superior temporal and parieto-occipital lobes. A faint rim of enhancement was noted along its medial margin. A first biopsy was performed in El Salvador and the diagnosis of oligodendroglioma was issued. The H&E of this first biopsy is presented in slide #1. After additional assessment and imaging, the patient was referred 6 months later to The Hospital for Sick Children for complete resection. The H&E of the second resection is presented in slide #2.

Material Submitted:
1. Representative digital images of H&E slides
2. Representative MRI images



Points for Discussion:
1. Differential Diagnosis (slides #1 and #2)
2. Molecular work up

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-4

Submitted By:
Erik A. Williams, MD, Matthew P. Frosch, MD, PhD, and E. Tessa Hedley-Whyte, MD
C S Kubik Laboratory of Neuropathology
Massachusetts General Hospital
55 Fruit Street,
Boston, MA 02114

Clinical History:
A 61-year-old man presented in the fall with headache, falls, and altered mental status. He had experienced several weeks of headache and malaise following known multiple tick bites in New Hampshire and treatment with doxycycline. His past medical history was significant for Crohn's disease maintained on adalimumab, atrial fibrillation on warfarin, and a prior right subcortical stroke (reportedly without prior deficits). On exam, he was noted to have jerking movements and rigidity on the right, and a left gaze preference. Lumbar puncture was performed and showed 430 nucleated cells, 5 RBCs, 96% lymphocytes, glucose 43, and total protein 133. MRI showed leptomeningeal enhancement involving the cerebral cortex, deep gray nuclei, midbrain, and cerebellum, and ill-defined enhancement and restricted diffusion within the left thalamocapsular region. He underwent biopsy of the right cerebellum.

Material Submitted:
One H&E stained virtual slide

Points for Discussion:
1. Etiology
2. Neuropathologic findings and differential diagnosis

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-5

Submitted By:
Meghan Driscoll, MD (Neuropathology Fellow), Jessica Corean, MD (Pathology Resident), and Cheryl Palmer, MD
University of Utah, Department of Pathology
1950 Circle of Hope Drive; Room N3105
Salt Lake City, UT 84112

Clinical History:
The patient is a 60-year-old woman who was employed as a third-grade teacher and presented to care with acute shortness of breath and heart failure. These findings were clinically attributed to pulmonary emboli. Upon admission, she was found to have acute heart failure, and developed cardiorenal syndrome. During her hospital stay, she was found to have *Klebsiella pneumoniae* bacteremia and became septic requiring inotropic support. Due to worsening cardiac function the patient and her family elected palliative care and she passed away 72 hours later.

Autopsy Findings:
Examination of the brain demonstrated a markedly enlarged pituitary gland that was 2.7 x 2.4 x 1.8 cm with a diffuse fleshy nodularity. The remainder of the brain and spinal cord examination was unremarkable.

Material Submitted:
One H&E stained slide and one unstained slide of the pituitary gland.

Points for Discussion:
1. Differential considerations and final diagnosis.
2. Relationship to general autopsy findings.

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-6

Submitted By:
Namita Sinha: Division of Neuropathology, Department of Pathology, Barnes-Jewish Hospital, Washington University School of Medicine, 660 S. Euclid Ave, Box 8118, St. Louis, MO 63110
Angelica Oviedo: Department of Pathology, IWK Health Centre, Dalhousie University Medical School, Halifax NS, Canada

Clinical History:
A 35-year-old G3P2 at 20w5d of gestation was referred for Maternal-Fetal Medicine consultation. A genetic sonogram revealed multiple congenital anomalies including dolicocephaly, elongated and parallel lateral ventricles, choroid plexus cysts, nuchal thickening, frontal bossing, midfacial hypoplasia, hypertelorism, bell-shaped chest, multiple echogenic intracardiac foci, and bilateral clubfeet. X-ray examination prior to autopsy showed absent mid-phalanx of 5th finger of hands, clefted lumbar vertebral bodies and 11 ribs. A male fetus was delivered vaginally at 22w5d of gestation.

Autopsy Findings:
Body length: 32 cm, Body weight: 526 g; Brain weight: 154.3 g; Right and left feet length were 2.9 and 3.4 cm respectively. Autopsy examination confirmed imaging findings and revealed vascular malformations in various organs and brain abnormalities (brain- figure 1, coronal section of brain- figure 2).

Material Submitted:
Images of brain, including coronal section, and H&E sections of the cerebrum

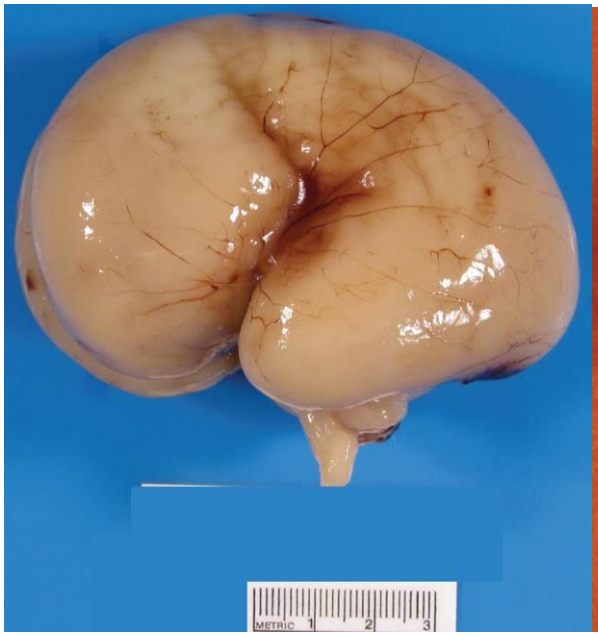


Figure 1

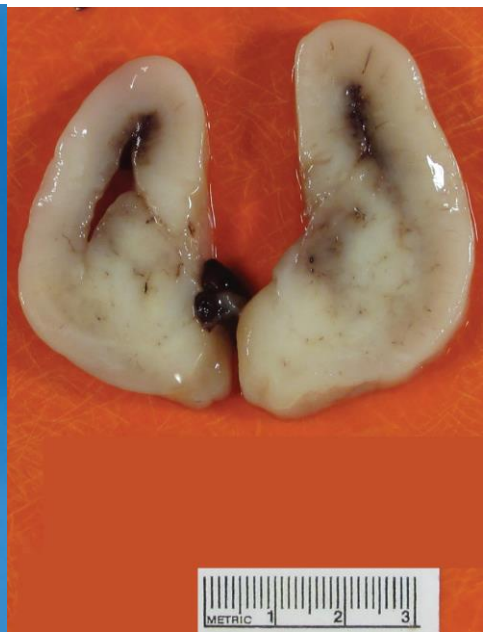


Figure 2

Points for Discussion:
1. Neurological manifestations of this syndrome
2. Pathogenesis and genetic pathway

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-7

Submitted By:
Woon Chow, MD, PhD, and Ty Abel, MD, PhD
Vanderbilt University Medical Center
Division of Neuropathology
1161 21st Ave S
Medical Center North C-2318
Nashville TN 37232-0011

Clinical History:
At the time of presentation in 2014, the patient was a 71-year-old female with a history of fibromyalgia, gastrointestinal bleeds, hypertension, and migraines who presented with confusion, lethargy, and worsening headaches. Social history included a 50-pack-year history of tobacco use. Initial non-contrast head CT imaging showed a 2.2-cm right temporal intraparenchymal hemorrhage with surrounding edema and mild mass effect. Further imaging evaluation revealed also numerous predominantly subcortical foci of microhemorrhages clustered in the right middle cerebral and left posterior cerebral artery distributions. The patient was not on anticoagulant or antiplatelet medications and laboratory studies were unremarkable. The platelet count was 258,000/µL and the INR was 1.0. A brain biopsy was performed.

Material Submitted:
H&E section of temporal lobe biopsy

Points for Discussion:
1. Differential considerations
2. Final diagnosis and clinical follow-up of patient

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-8

Submitted By:
Angela Wu, MD[1], Mark H. Lipson, MD[2], and Steven A. Moore, MD, PhD[1]
1 – The University of Iowa, Department of Pathology, Iowa City, IA 52242
2 – Medical Genetics, Kaiser Permanente, 1650 Response Road, Sacramento, CA 95864

Clinical History:
This infant boy was born by C-section at 41 weeks' gestation after a normal pregnancy, and his 2 month well child exam was normal.

However, by 3 months of age, his parents had observed him moving less and having difficulty holding his head up. On exam, he had significant hypotonia and weakness, greater proximally than distally. An MRI showed diffuse, mildly increased T2 signal in his muscles. Electromyography (EMG) showed fibrillations and low amplitude motor units with polyphasic waves consistent with myopathy. Nerve conduction and neuromuscular junction studies were normal. His weakness progressed, leading to difficulty with eating and respiration; he had episodes of aspiration. His serum creatinine kinase levels ranged from 3170 to 4944 U/L. There is no family history of neuromuscular disease. He was hospitalized multiple times, became dependent on tube feedings and respiratory support, and passed away at 6 months of age.

A right quadriceps muscle biopsy was performed at 4 months of age.

Material Submitted:
H&E stained cryosection of muscle

Points for Discussion:
1. Differential diagnosis
2. Approach to diagnostic testing

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-9

Submitted By:
Sanda Alexandrescu, MD; Sara O. Vargas, MD
Department of Pathology, Boston Children's Hospital

Clinical History:
The patient is a 3-year-year old previously healthy girl, who presented to an outside emergency department with a complaint of 2-3 weeks of intermittent right-sided headaches.

Magnetic resonance imaging (MRI) demonstrated a right superior temporal gyrus tumor involving the cortex and leptomeninges. The patient was transferred to Boston Children's Hospital, where the tumor was resected. During the surgery, it was noted that the surface of the tumor was richly vascular, and the tumor abutted, but did not attach to the dura. Gross examination revealed red-brown, soft fragments of tissue (3.5 x 2.1 x1.5 cm).

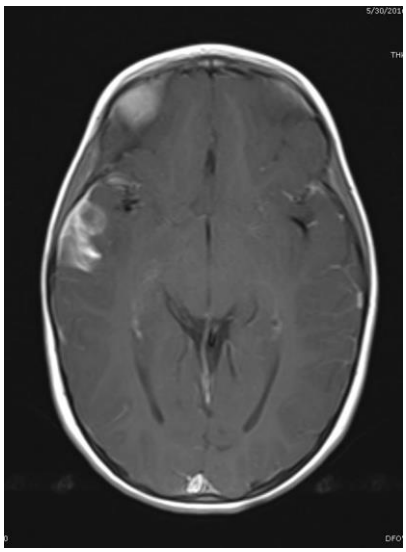


Figure 1: T1 hyperintensw Right temporal lobe lesion

Material Submitted:
One virtual H&E slide

Points for Discussion:
1. Differential diagnosis
2. Ancillary studies
3. Integration of molecular test results, and their clinical implications

58th ANNUAL DIAGNOSTIC SLIDE SESSION 2017
CASE 2017-10

Submitted By:
Andrew R. Guajardo, MD and Fausto Rodriguez, MD.
Division of Neuropathology, Johns Hopkins University Hospital, 720 Rutland Avenue, Ross Bldg., Room 558, Baltimore, MD 21205

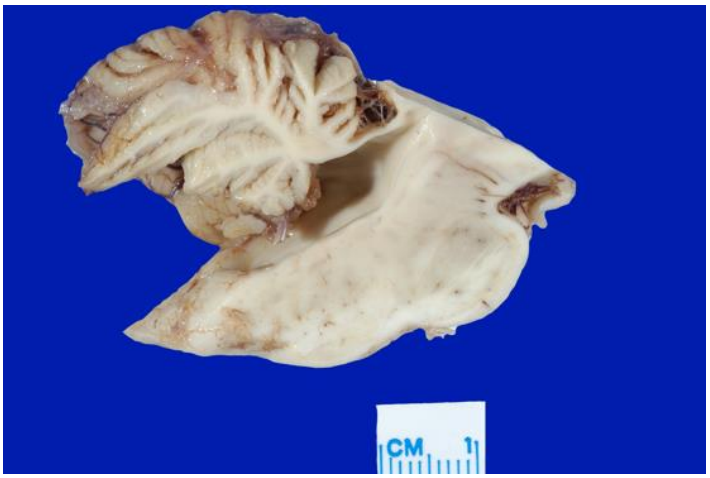
Clinical History:
14-year-old boy with a history of myelodysplastic syndrome (MDS) status post bone marrow transplant in 2014. There was a strong family history of neoplastic disease including a paternal aunt who died at 26 years old due to lymphoma, a paternal grandmother and great-grandmother with breast cancer, and a maternal great aunt with ovarian cancer. Other medical history included posterior reversible encephalopathy syndrome (PRES) complicated by seizures, developmental delay, iron overload secondary to transfusions, gastrointestinal graft vs host disease, and repeated infections including CMV colitis, enterococcus sepsis, and pneumocystis pneumonia. A month prior to death, the patient was admitted to the hospital with a gastrointestinal hemorrhage, acute respiratory distress, renal failure, and disseminated intravascular coagulopathy. His family elected for comfort care. A complete autopsy was performed.

Autopsy Findings:
In addition to extensive evidence of medical therapy and terminal medical complications, the general autopsy showed an irregular scale-like pigmentation of the skin with a yellowish cast overall. The eyelashes and eyebrows appeared diminished. The fingernails and toenails were abnormal. Internal exam demonstrated focal greenish discoloration of the left ventricular papillary muscle of the heart as well as patchy hemorrhage and peripheral nodularity of the lungs.

On neuropathological examination, the fresh brain weighed 770 g. The cerebellum was diffusely hypoplastic with significant vermian atrophy. Coronal sections revealed that the neocortex was unremarkable for prominent vasculature, hemorrhages, or mass lesions. There were multiple distinct cavitary lesions filled with firm, yellow-tan material within the thalami. There was a cavum septum pellucidum. The midbrain, basis pontis, and medulla were grossly unremarkable. Gross examination of the eyes showed no significant abnormalities.

Material Submitted:
1. Gross photographs of the brainstem and cerebellum
2. H&E sections of cerebellum

Points for Discussion:
1. Diagnosis and subtyping
2. Underlying pathogenesis



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Presidential Symposium

Sunday, June 11, 2017

Learning Objectives:

1. *Summarize recent advances in the field of neuropathology.*
2. *Discuss innovative approaches to neuropathology teaching.*
3. *Cite ways to apply new research data and methods to daily practice.*

PRESIDENTIAL SYMPOSIUM

Zika Virus Encephalomyelopathy

Time: 9:00 am – 9:45 am

Leila Chimelli, MD, PhD, *State Institute of Brain and Federal University of Rio de Janeiro, Rio de Janeiro, Brazil*

I. Learning Objectives:

At the end of this activity, learners should be able to:

1. Explain the epidemiology, risk of infection and main symptoms of Zika virus infection.
2. Recognize the morphological patterns of neurodevelopmental disorders associated with Zika virus infection and the differential diagnosis with other congenital infections.
3. Describe the pathogenesis of the lesions of Zika virus enceophalopathy and their relationship with the time of infection.
4. Correlate the radiological and clinical presentations of congenital Zika virus infection with the pathological changes.

II. Abstract and Relevant Resources

Zika virus (ZIKV), a flavivirus transmitted by *Aedes **aegypti***, recently arrived in Brazil and spread to many states. Human infection varies from mild fever, arthralgia, rash, headache, and myalgia, but may be asymptomatic. A major concern associated with ZIKV infection was the increased incidence of microcephaly with frequent calcifications in infants born from mothers who were infected during the 1st trimester of pregnancy. Those who were followed intrauterus with ultrasound/CT were reported to have cerebral maturation and growth, drastically affected. Many were born with arthrogryposis. Microcephaly was not always observed, sometimes due to a compensation of cephalic perimeter by ventriculomegaly. A comprehensive neuropathological study was made in ten newborn babies infected with ZIKV during pregnancy, including the spinal cords and dorsal root ganglia, and also muscle, eye, systemic organs and placentas. Most were born at term. Using *in situ* hybridization and electron microscopy, we investigated the role of direct viral infection in the pathogenesis of the lesions. We defined three patterns of CNS lesions, with a mixture of destruction, calcification, hypoplasia and migration disturbances. Hydrocephalus was severe in the first pattern (5 cases) due to midbrain damage with aqueduct stenosis/distortion. The second pattern had small brains and mild/moderate (ex-vacuo) ventriculomegaly (4 cases). The third pattern, a well-formed brain with mild calcification, coincided with late infection. Absence of descending fibers resulted in hypoplastic basis pontis, pyramids and cortico-spinal tracts. Spinal motor cell loss explained the intrauterine akinesia, arthrogryposis and neurogenic muscle atrophy. DRG, dorsal nerve roots and columns were normal. Inflammation was mild. ISH showed meningeal, germinal matrix and neocortical infection, consistent with neural progenitors death leading to proliferation and migration disorders. A secondary ischemic process may also explain the destructive lesions. We characterized the destructive and malformative consequences of ZIKV in the nervous system, as reflected in the topography and severity of lesions, anatomic localization of the virus, and timing of infection during gestation, indicating a developmental vulnerability of the immature CNS.

References:

1. Araujo AQ, Silva MT, Araujo AP (2016) Zika virus-associated neurological disorders: A review. Brain 139:2122–2130. doi: 10.1093/brain/aww158
2. Chimelli L, Melo AS, Avvad-Portari E, et al (2017) The spectrum of neuropathological changes associated with congenital Zika virus infection. Acta Neuropathol. Mar 22. doi: 10.1007/s00401-017-1699-5. [Epub ahead of print]
3. Klase ZA, Khakhina S, Schneider ADB, et al (2016) Zika Fetal Neuropathogenesis: Etiology of a Viral Syndrome. PLoS Negl Trop Dis 10:1–32. doi: 10.1371/journal.pntd.0004877.
4. Melo AS, Aguiar RS, Amorim MMR, et al (2016) Congenital Zika Virus Infection. Beyond Neonatal

Microcephaly. JAMA Neurol 73:1407-1416. doi:10.1001/jamaneurol.2016.3720.
5. Štrafela P, Vizjak A, Mraz J, Mlakar J, Pižem J, Tul N, Županc TA, Popović M (2016) Zika Virus–Associated Micrencephaly: A Thorough Description of Neuropathologic Findings in the Fetal Central Nervous System. Arch Pathol Lab Med 141:73-81. doi: 10.5858/arpa.2016-0341.
6. Tang H, Hammack C, Ogden SC et al (2016) Zika Virus Infects Human Cortical Neural Progenitors and Attenuates Their Growth. Stem Cell 18:587-90. doi: 10.1016/j.stem.2016.02.016.

III. Faculty Biography:

Dr. Leila Chimelli has graduated in Medicine in 1976 and took postgraduate training as Resident in Pathology in Rio de Janeiro, Brazil. Her neuropathology training and PhD were obtained at the Institute of Neurology Queen Square, London, UK (1982-1985) and the post Doctorate, at Henri Mondor Hospital, Créteil, University of Paris XII, France (1989). Assistant and Associate Professor at Universities in Rio and São Paulo since 1980, she has been Professor of Pathology at Federal University of Rio de Janeiro since 1997 and coordinator of the Neuropathology Laboratory at State Institute of Brain since 2013. With experience in surgical neuropathology, muscle and nerve biopsies and autopsies, she has published several articles and book chapters on infectious diseases, neuropathies and brain tumors. She has been member of the American Association of Neuropathologists for more than 20 years, served as arquivist and vice-president of the International Society of Neuropathology, and in 2014 was the President of the XVIII International Congress of Neuropathology in Rio. Since the beginning of 2016 she has been involved with the neuropathology of congenital Zika.

PRESIDENTIAL SYMPOSIUM

Perinatal Neuropathology – Current Concepts

Time: 10:30 am – 11:15 am

Mark R. Del Bigio, MD, PhD, FRCPC, Professor - *Department of Pathology, University of Manitoba, Winnipeg, MB, Canada*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Define the perinatal period with respect to neuropathology
2. Describe human brain cell generation in the 2nd half of gestation and the negative impacts of premature birth.
3. Summarize different modes of cell death that can occur in brain cells
4. Describe the general mechanism through which viral infections damage the fetal brain.

II. Abstract & Relevant References

Perinatal neuropathology is understandable in the context of developmental neurobiology. Animal studies have led to significant insights about brain development; markers of immature brain cells and mechanisms of migration have been identified and successfully applied to human brain tissue. For administrative purposes the perinatal period has been defined as the period between 22 weeks' gestation and 1 week after birth. Perinatal neuropathology, if it requires a definition, should be considered more broadly and in biologic terms. The period of copious neuron and glial precursor proliferation begins roughly mid gestation and extends to approximately 29-30 weeks' gestation (including postnatal development in premature infants), with some neocortical and basal nuclei neurons generated as late as 35 weeks. Immature cell migration and early maturation continue into the first year of life, in particular in the cerebellum. Destructive lesions during these periods can have primary and secondary effects that differ from those in the mature brain and may include malformative events. Modes of cell death differ from those in the mature brain and are not restricted to necrosis and apoptosis. This is not a critical point for the practicing neuropathologist, but is important for the conduct of preclinical therapeutic research. In utero arterial ischemic lesions can result in a range of destructive patterns according to the anatomic distribution; residual damage such as polymicrogyria reflects ongoing aberrant development at the edge of these lesions. In utero infections can also result in a range of lesions; the anatomic pattern of damage and age of susceptibility are in accordance with the temporal expression of cellular receptors used by the viruses to gain entrance. In utero toxic lesions result in a range of damage patterns; some such as retinoic acid interfere with specific signaling pathways, others like ethanol have broad but less specific effects including epigenetic modifications. Complications of premature birth can broadly be categorized as hemorrhagic or hypoxic-ischemic. Hemorrhagic lesions in the periventricular germinal tissue can interfere with subsequent cell proliferation and ultimately with generation of inhibitory interneurons. Pre-oligodendrocytes, axons, and immature neurons are susceptible to hypoxia-ischemia. Inflammatory reactions to the insults can be used as markers of regional injury and might represent targets for therapy. Mechanical trauma to the head can cause a range of hemorrhagic and distortional injuries to the brain. These may to some extent overlap with postnatal trauma (including abusive injury) in the infant brain. Despite the fact that animal research has been critical for discovery of damage mechanism, the preclinical animal models of developmental brain disease remain limited in their ability to identify successful interventions. Interaction between clinical neuropathologists and basic researchers remains an important enterprise.

***References*:**

1. Del Bigio MR. Cell proliferation in human ganglionic eminence and suppression after prematurity-associated haemorrhage. Brain. 2011 134:1344-61
2. Malik S et al. Neurogenesis continues in the third trimester of pregnancy and is suppressed by premature birth. J Neurosci. 2013 33:411-23

3. Arshad A et al. Extended production of cortical interneurons into the third trimester of human gestation. Cereb Cortex. 2016 26:2242-56
4. Nikoletopoulou V et al. Crosstalk between apoptosis, necrosis and autophagy. Biochim Biophys Acta. 2013 1833:3448-59.
5. Chimelli L et al. The spectrum of neuropathological changes associated with congenital Zika virus infection. Acta Neuropathol. 2017 Mar 22.
6. Nowakowski TJ et al. Expression analysis highlights AXL as a candidate Zika virus entry receptor in neural stem cells. Cell Stem Cell. 2016 18:591-6
7. Ballabh P. Pathogenesis and prevention of intraventricular hemorrhage. Clin Perinatol. 2014 41:47-67
8. Lea CL et al. Protecting the premature brain: current evidence-based strategies for minimising perinatal brain injury in preterm infants. Arch Dis Child Fetal Neonatal Ed. 2017 102:F176-F182.
9. Penn AA et al. Controversies in preterm brain injury. Neurobiol Dis. 2016 92:90-101.

III. Faculty Biography:

Dr. Del Bigio obtained his MD in 1982 and PhD in 1987 from the University of Manitoba. He started residency in neurosurgery, but reevaluated his career goals and switched to neuropathology, training at the University of Toronto from 1990 to 1993. After a postdoctoral research period, he was recruited back to the University of Manitoba, where he has worked as a neuropathologist of the Health Sciences Centre since 1994. His particular clinical interests are in pediatric neuropathology and forensic neuropathology. He teaches in the Department of Pathology, where he is currently tenured Professor (since 2002). He served nationally as chair of the Specialty Committee in Neuropathology for the Royal College of Physicians and Surgeons of Canada (2008-14).

As a clinician-scientist, Dr. Del Bigio has had a long interest in premature birth, brain hemorrhage, and hydrocephalus. More recent work involves investigating how the physical properties of brain determine its response to injury, and the fetal alcohol spectrum disorder. He holds the Tier 1 Canada Research Chair in Developmental Neuropathology (2004-2017). His research mainly involves studies of preclinical animal models and corresponding autopsy material. He has also developed an interest in education and training of neuropathologists.

PRESIDENTIAL SYMPOSIUM

Innovative Approaches to Neuropathology Teacher – Panel Discussion: Music, Social Media and Online Resources

Time: 11:15 am – 12:00 pm

Music

Arie Perry, MD, *University of California San Francisco School of Medicine, San Francisco, CA*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Describe the associations between music and memory.
2. Identify the benefits of using music in neuropathology education.

II. Abstract & Relevant References

Although not entirely understood from a mechanistic perspective, it has long been recognized that musical memories are often more powerful and long-lasting than rote memorization, likely due to its emotional overlay. As such, the presenter has spent many years exploiting this phenomenon by reinforcing lecture topics with "neuropathology songs" to aid medical students and other medical professionals in learning key facts about neurological disorders. Eventually, this led to a commercial CD recording of 16 such songs in 2010, with well over 1000 copies to date distributed to medical students and others as an educational supplement, as well as a more recent YouTube video entitled "Brain Tumor Rhapsody" that has garnered over 8400 views to date. To a lesser extent, these resources have also been utilized by other neuropathologists as an educational aid within their own courses and lectures. Nevertheless, the presenter hopes that the AANP audience will be able to simply sit back and enjoy this presentation, at least as much as they learn from it.

References:

1. Perry A. Newborn twins of neuropathology education. Brain Pathol 2010;20:i.
2. Altenmuller E, Demorest SM, Fujioka T, et al. Introduction to The neurosciences and music IV: learning and memory. Ann N Y Acad Sci 2012;1252:1-16.
3. Innes KE, Selfe TK, Khalsa DS, Kandati S. Meditation and Music Improve Memory and Cognitive Function in Adults with Subjective Cognitive Decline: A Pilot Randomized Controlled Trial. J Alzheimers Dis 2017;56:899-916.
4. http://www.neuropathsongs.com.
5. https://www.youtube.com/watch?v=FfP4HTuu6Vs.

III. Faculty Biography:

Arie Perry is a Professor at the University of California in San Francisco, where he serves as the Director of the Neuropathology Division and the Neuropathology Fellowship program. His interests have focused mostly on classification, grading, and molecular characterization of both adult and pediatric brain tumors. In addition, he enjoys creative modes of teaching making use of his longstanding passion for music. In this respect, he has been featured in several media stories for using neuropathology songs in education, including the St. Louis Post Dispatch, the Washington University Record, KWMU Radio, NPR, Chorus America, the San Francisco Chronicle, UCSF Pulse, Monocle Magazine, and KCBS Radio.

Social Media

Brian E. Moore, MD, MEd, *Associate Professor, University of Colorado School of Medicine, Aurora, CO*

I. Learning Objectives

At the end of this activity learners should be able to:

1. Identify the principle social media websites used by neuropathologists.
2. List the official social media accounts used by the American Association of Neuropathologists.
3. Explain the basics of how to interact with other neuropathologists and the public-at-large via social media.

II. Abstract & Relevant References

There is increasing use of social media and social networking among neuropathologists in recent years. The most popular sites are Facebook and Twitter, but others include Blogspot, Tumblr, Instagram, Pinterest, Flickr, LinkedIn, and many more. The American Association of Neuropathologists has joined virtually all other professional organizations in establishing official social media accounts (Facebook and Twitter) to communicate with its members, to foster communication among members, and to educate individuals outside the field about the role neuropathologists play in science and medicine. This presentation will focus on the current state of social media in the field of neuropathology and how it enhances communication, education, and even diagnosis.

References:

1. Gonzalez RS, Amer SM, Yahia NB, *et al*. (2016) **Facebook Discussion Groups Provide a Robust Worldwide Platform for Free Pathology Education**. *Archives of Pathology & Laboratory Medicine*. In Press at time of preparation of this talk.
2. Tanveer N. **Social Media: The New Frontier for Pathologists**. *Indian J Pathol Microbiol*. 2017 Jan-Mar;60(1):143-144.
3. Cohen D, Allen TC, Balci S, et al. **#InSituPathologists: how the #USCAP2015 meeting went viral on Twitter and founded the social media movement for the United States and Canadian Academy of Pathology**. Mod Pathol 2017 Feb;30(2):160-168.

III. Faculty Biography:

Brian E. Moore, MD, MEd is an associate professor at the University of Colorado School of Medicine in Denver. He is a member of the AANP website committee. Since October 2007, Dr. Moore has operated Neuropathology Blog, which has had over 800,000 pageviews since its inception.

Online Resources

Cheryl Ann Palmer, MD, *Professor and Director of Neuropathology, University of Utah, Salt Lake City, UT*

I. Learning Objectives

At the end of this activity learners should be able to:

1. List the most common online resources available to neuropathologists for surgical neuropathology.
2. Identify the best websites for teaching neuropathology to medical students.
3. Identify the most common online resources available to neuropathologists for neuromuscular pathology.

II. Abstract & Relevant References

Neuropathology education has evolved over the years, with more residency programs feeling the need for their residents to obtain more neuropathology education. Additionally, other health science professionals desire some formal neuropathology education, such as physical therapists and dental students. Online resources can help stretch the time of busy neuropathologists by providing educational value to different trainees.

References:

1. Neuromuscular.wustl.edu
2. Neuropathology-web.org
3. Library.med.utah.edu
4. Urmc.rochester.edu
5. Virtualpathology.leeds.ac.uk

III. Faculty Biography:

Cheryl Ann Palmer, M.D. is the current Vice President of the American Association of Neuropathologists. She is a Professor of Pathology and the Director of Neuropathology at the University of Utah, and serves as the Pathology Residency Program Director.

NOTES

NOTES