

AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

June 7 - 10, 2018

Hyatt Regency Louisville

Louisville, Kentucky

This activity is provided by the American Association of Neuropathologists.



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American Association of Neuropathologists



The American Association of Neuropathologists

8156-E S. Wadsworth Blvd., Suite 197 Littleton, Colorado 80128 Phone: 720-372-0888; Fax: 303-568-0406

Email: aanp@aoeconsulting.com

Dear Colleagues:

As the President of the American Association of Neuropathologists (AANP), it gives me great pleasure to welcome you to Louisville, Kentucky for the AANP's 94th Annual Meeting.

The AANP has a proud history of being the *only* association exclusively focused on the clinical and scientific practice of neuropathology in the United States. Since the 1920s, the AANP has been working to advance the science, teaching and training of the diseases of the nervous system and the practice of neuropathology. Most recently, we have entered the era of genomics and precision diagnostics. In an effort to stay relevant in this rapidly evolving field, the 94th Annual Meeting serves to provide the latest insights into cutting-edge science and groundbreaking research.

This year's meeting includes a one-day Special Course on Thursday dedicated to the *Neuropathology* of Aging and an *Update* on *Molecular Analysis* of *Brain Tumors*. During the two days of scientific platform and poster sessions, Friday and Saturday, you have the opportunity to match your needs and interests with a variety of topics. Saturday evening will include the Diagnostic Slide Session, and Sunday concludes with a half-day Presidential Symposium focusing on *Emerging Technologies in Neuropathology*.

As a community that genuinely enjoys time together, you will have ample opportunity to socialize with old friends and forge new neuropathology connections with colleagues from around the world. I also want to thank our exhibitors. Their attendance at this meeting is deeply appreciated. Please visit the exhibit space and explore a variety of high-quality products and services.

As I approach the last days in my term as President, I want to thank each of you for attending the AANP's 94th Annual Meeting and for bringing your expertise, experience, questions and comments to this event. I hope you leave this conference with innovative ideas, strengthened connections and a renewed spirit.

Sincerely,

Elizabeth J. Cochran, MD

President

The American Association of Neuropathologists

Elizabeth of Cochran

Save the Date!



American Association of Neuropathologists

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

AANP OFFICE

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OFFICIAL JOURNAL

Journal of Neuropathology and Experimental Neurology
John (Jack) Lee, MD, PhD, Editor-in-Chief
Eileen S. Healy, Managing Editor

E-mail: <u>inen@pathology.wisc.edu</u>
Home page: <u>http://www.jneuropath.com</u>

DIAGNOSTIC SLIDE SESSION

Caterina Giannini, MD, PhD, Mayo Clinic, Moderator Rebecca D. Folkerth, MD, New York City Office of the Chief Medical Examiner, Manager

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*Junior Member

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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:

- 1. Describe new advancements in mechanisms and etiologies of neurologic diseases.
- 2. Discuss recent advances in neurologic disease research.
- 3. Evaluate new methodologic and diagnostic knowledge that can improve patient care in 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 25.25 $AMA\ PRA\ Category\ 1\ Credits^{TM}$. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

MAINTENANCE OF CERTIFICATION

The 94th Annual Meeting of the American Association of Neuropathologists will offer both Continuing Medical Education (CME) and American Board of Pathology (ABPath) Continuous Certification, formerly Maintenance of Certification (MOC), Part II: Lifelong Learning and Self-Assessment. Participation in the live activity and successful completion of the corresponding evaluation component for each eligible session enables participants to earn up to a maximum of 16.25 SAM credits.

CME INFORMATION (Continued)

CME AND SAM CREDIT

Instructions to Receive CME 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/aanp2018.

Instructions to Receive SAM Credit

Shortly after the 94th Annual Meeting, the evaluation component for SAM credit will launch at www.neuropath-education.org. Participants will need to use their Dayspring website log-in to gain access to each evaluation component and must have attended the live session held at the 2018 Annual Meeting in Louisville, KY. Each SAM costs \$25.00 unless you previously purchased the SAMs bundle.

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.

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 accredited for *AMA PRA Category 1 Credit*TM at this year's Annual Meeting. The chart also outlines the SAM credit available for each session.

Activity	CME Credit Hours	SAM Credit Hours
Special Course	6.75	6.75
Scientific Sessions	8.0	0
Korey Lecture	1.0	1.0
DeArmond Lecture	1.0	1.0
Parisi Lecture	1.0	1.0
Moore Lecture	0.75	0.75
What Every Neuropathologist Needs to Know	1.0	0
Diagnostic Slide Session	3.0	3.0
Presidential Symposium	2.75	2.75
Total	25.25	16.25

CONTACT INFORMATION

For any questions regarding the accreditation of this meeting, please contact AANP's CME Coordinator, Sarah Porter, via e-mail at: sporter@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 R13 Grant:

Funding for this conference was made possible, in part by 1R13AG059336-01 from National Institute on Aging. The views expressed in written conference materials or publications and by speakers and moderators do not necessarily reflect the official policies of the Department of Health and Human Services; nor does mention by trade names, commercial practices, or organizations imply endorsement by the U.S. Government.

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 all relevant financial relationships with commercial interests 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.

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

Adekunle Adesina, Douglas Anthony, Jennifer Baccon, Eileen Bigio, Daniel Brat, Rati Chkheidze, Elizabeth Cochran, Sonika Dahiya, Ivana Delalle, Sean Ferris, Rebecca Folkerth, Matthew Frosch, Caterina Giannini, Miguel Guzman, Brent Harris, Kimmo Hatanpaa, Anne Hiniker, Julie Kofler, Nancy Kois, Jesse Kresak, Edward Lee, Giselle Lopez, Michelle Madden Felicella, Qinwen Mao, Thomas Montine, Brent Orr, Angelica Oviedo, Richard Perrin, Arie Perry, Sarah Porter, Suzanne Powell, R. Ross Reichard, Charles Specht, Anat Stemmer-Rachamimov, Rachel Vaubel, Cynthia (Cindy) Welsh, Charles White, Qian Wu, Gabrielle Yeaney

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	Advisory Board - Audentes Therapeutics, Solid Biosciences, Ichorion	
	Therapuetics	
	Grant/Research Support: Audentes Therapeutics, Solid Biosciences,	
	Ichorion Therapeutics	
Han Lee	Stock Shareholder (Spouse/Partner): Gilead	
	Employee (Spouse/Partner): Theravance	
John (Jack) Lee	Consultant/Independent Contractor: Holds joint patents on a drug for	
	treatment of Alzheimer's disease/senile dementia and anxiety disorders	
	with others and Cornelli Consulting, Milan, Italy (No royalties associated –	
	patent tied to research in early stages)	

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GENERAL INFORMATION

LOCATION

Hyatt Regency Louisville 311 S. 4th Street Louisville, KY 40202

All meeting sessions will be held at the Hyatt Regency Louisville.

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 the **Regency Ballroom** and **Regency South Ballroom** of the hotel on the second floor.

All poster sessions will be held in **Gulfstream-Hialeah** and **Keeneland** on the second floor.

REGISTRATION DESK

Top of the Escalators	
Wednesday, June 6	4:00 pm – 8:00 pm
Thursday, June 7	7:00 am – 5:00 pm
Friday, June 8	7:00 am – 5:00 pm
Saturday, June 9	7:00 am – 5:00 pm
Sunday, June 10	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 2018 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 at the **Top of the Escalators**, on the second floor. 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 Annual Reception.

MICROSCOPE VIEWING ROOM

Multi-headed microscopes will be available in **Downs** on the second floor of the hotel.

Location: Downs	
Thursday, June 7	7:00 am – 7:30 pm
Friday, June 8	7:00 am – 7:30 pm
Saturday, June 9	7:00 am – 7:30 pm
Sunday, June 10	7:00 am – 10:30 am

GENERAL INFORMATION (Continued)

SPECIAL MEETINGS BY INVITATION

Day/Date	Meeting	Time/Location
Wednesday,	Neuropathology Fellowship Program Directors	4:30 pm – 6:30 pm
June 6	Committee Meeting	Gulfstream-Hialeah, 2 nd Floor
	Education Committee Meeting	6:30 pm – 9:30 pm
		Kentucky Suite, 2 nd Floor
Thursday,	Awards Committee Meeting #1	5:30 pm – 6:00 pm
June 7		Oaklawn, 2 nd Floor
	Executive Council Meeting	7:00 pm – 10:00 pm
		Pimlico, 1 st Floor
Friday,	Trainee Luncheon and Networking Event*	11:45 am – 2:00 pm
June 8	*Open to all Trainees and Travel Award Winners	Park Suite/Kentucky Suite, 2 nd Floor
	Website Committee Meeting	12:30 pm – 1:30 pm
		Aqueduct, 1 st Floor
	Awards Committee Meeting #2	5:30 pm – 6:30 pm
		Oaks, 2 nd Floor
	Professional Affairs Committee Meeting	5:30 pm – 7:00 pm
		Saratoga, 1 st Floor
Saturday,	JNEN Editorial Board Meeting	7:00 am – 8:00 am
June 9		Kentucky Suite, 2 nd Floor
	Awards Committee Meeting #3	6:00 pm – 8:00 pm
		Belmont, 1 st Floor
	Presidential Reception	6:00 pm – 8:00 pm
		The Spire
Sunday,	DSS Founders Breakfast	7:00 am – 8:00 am
June 10		Aqueduct, 1st Floor

ANNUAL RECEPTION

The annual reception will be held from 5:30 pm to 7:30 pm, Thursday, June 7 in the **Kentucky Suite/Park Suite** on the second 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.

Kentucky Suite/Park Suite	
Thursday, June 7, 2018	5:30 pm – 7:30 pm

TRAINEE LUNCHEON

Trainees and Travel Award winners are invited to attend the 2018 Trainee Luncheon and Networking Event on Friday, June 8 in **Park Suite/Kentucky Suite** (2nd Floor), hosted by Dr. Suzanne Powell. Lunch will be provided, followed by dessert. The agenda is posted below.

2018 Trainee Luncheon Agenda

Room: Park Suite

- I. 11:45 am 12:15 pm: Welcome & Lunch
- II. 12:15 pm 12:45 pm: Travel Awards Recognition

Room: Kentucky Suite

- III. 12:45 pm 2:00 pm: Networking Event & Dessert
- IV. 1:30 pm 2:00 pm: Mingle with Executive Council

EXHIBITORS & SPONSORS

Thank you to our 2018 exhibitors and sponsors! Please visit the exhibit booths in the Regency Ballroom Foyer.

Location: Regency Ballroom Foyer	
Thursday, June 7	7:00 am - 5:30 pm
Friday, June 8	7:00 am - 5:30 pm
Saturday, June 9	7:00 am - 5:30 pm

EXHIBITORS

OXFORD UNIVERSITY PRESS	Oxford University Press is the proud publisher of the <i>Journal of Neuropathology & Experimental Neurology (JNEN)</i> . OUP also publishes some of the most renowned and respected medicine books and journals in the world. Visit our stand for promotional items and more information about your AANP member benefits to publishing in <i>JNEN</i> . Note: Copies of the June Edition of the <i>JNEN</i> have been provided by Oxford University Press.	Booth #1
Booth #2	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, deliver better care, and sometimes make groundbreaking discoveries that advance the boundaries of knowledge and human progress. Elsevier provides web-based, digital solutions — among them ScienceDirect, Scopus, Elsevier Research Intelligence and ClinicalKey — and publishes over 2,500 journals, including The Lancet and Cell, and more than 33,000 book titles, including a number of iconic reference works. Elsevier is part of RELX Group plc, a world-leading provider of information solutions for professional customers across industries.	ELSEVIER
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Booth #4	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 .	autism o BRAIN NET

Roche	Roche provides innovative research and clinical diagnostics solutions for laboratories to be more productive and for healthcare providers to make faster, more confident therapy decisions for patients. Our VENTANA portfolio for slide-based tissue diagnosis of cancer and infectious disease includes comprehensive solutions for digital pathology and workflow, companion diagnostics, high medical value assays, automated slide staining and state-of-the-art sample tracking. These solutions help anatomical pathology labs improve workflow efficiency, deliver enhanced medical value and ensure safety for the user and the patient. Roche Diagnostics Corporation 9115 Hague Road Indianapolis, IN 46256 United States 800-428-5074	Booth #5
	www.usdiagnostics.roche.com www.ventana.com	
Booth #6	EntroGen is a Los Angeles-based biotechnology company with a primary focus on molecular diagnostics in the areas of hematology and oncology. EntroGen has a growing commercial portfolio of real-time PCR and NGS based tests, with many of its products being used to guide and monitor targeted therapies for various malignancies. Through a network of distributors, EntroGen's footprint reaches every corner of the globe to deliver reliable and innovative tools to researchers and clinicians.	EntroGen° Predictive • Preventive • Pesconatzed
FOUNDATIONONE CDx™	Foundation Medicine is a world-leading molecular insights company, connecting physicians and their patients to the latest cancer treatment approaches and making precision medicine a reality for thousands. Our portfolio of genomic tests is the leading platform for comprehensive genomic profiling. We interrogate more than 300 genes across all four classes of genomic alterations to help understand the genomic makeup of a patient's tumor.	Booth #7

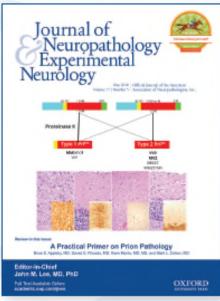
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Journal of Neuropathology Experimental Neurology

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
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- Special features that highlight AANP members e.g. autobiographies
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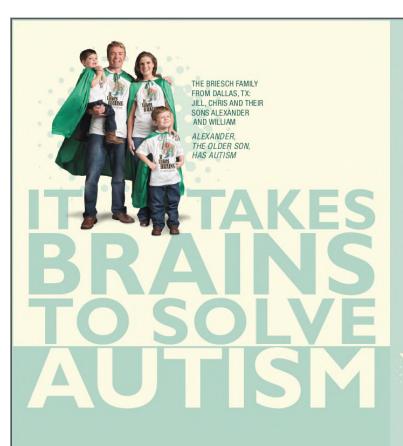
Explore a new virtual issue featuring leading JNEN research on the topic of Alzheimer disease.

Freely available here: bit.ly/JNEN_AD

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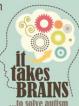
The Briesch family never saw themselves as a "super" family, yet when they bonded together to support their son Alexander, they were indeed super. And now the Briesches and families like them all over **Texas** and around the world are being looked upon as superheroes. Why?

They have all been united by one brave and heroic act ... pledging to donate the brain tissue of their sons and daughters 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.

Join forces with the Briesch family and other superhero families to help ensure a brighter future for all. Because it takes brains to solve autism.

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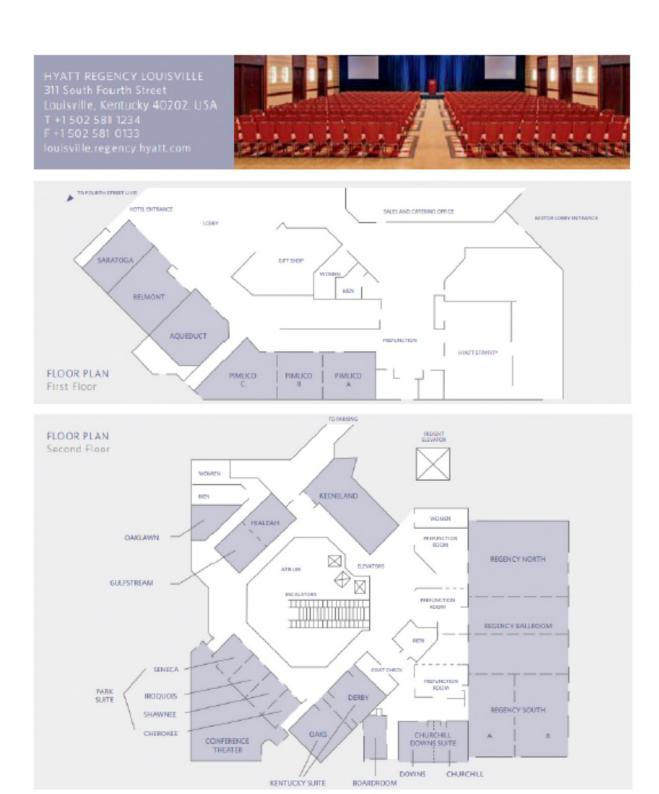
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THE HYATT REGENCY FLOOR PLANS



2018 SPECIAL COURSE

Neuropathology of Aging & Update on Molecular Analysis of Brain Tumors Co-Directors: Elizabeth J. Cochran, MD & Daniel J. Brat, MD, PhD

7:00 am – 8:00 am CONTINENTAL BREAK Neu 8:00 am – 8:15 am Welcome and CME Pro PET Imaging of Amylo	Regency Ballroom GFAST — Regency Ballroom Foyer ropathology of Aging e-Test iid and Tau in Human Aging Elizabeth Mormino, PhD Stanford University, Stanford, CA	
7:00 am – 8:00 am CONTINENTAL BREAK Neu 8:00 am – 8:15 am Welcome and CME Pro PET Imaging of Amylo	IFAST – Regency Ballroom Foyer ropathology of Aging e-Test iid and Tau in Human Aging Elizabeth Mormino, PhD	
8:00 am – 8:15 am Welcome and CME Properties PET Imaging of Amylo	ropathology of Aging e-Test id and Tau in Human Aging Elizabeth Mormino, PhD	
8:00 am – 8:15 am Welcome and CME Pro PET Imaging of Amylo	e-Test id and Tau in Human Aging Elizabeth Mormino, PhD	
8:00 am – 8:15 am PET Imaging of Amylo	id and Tau in Human Aging Elizabeth Mormino, PhD	
	Elizabeth Mormino, PhD	
0.45 0.00		
8:15 am – 9:00 am	Stanford University, Stanford, CA	
Diversity Deleted	To another (DART) Community No. 1991 to the Advance O	
9:00 am – 9:45 am	Tauopathy (PART): Concepts, Neuropathology, Advances &	
3.43 um	John F. Crary, MD, PhD	
	Icahn School of Medicine at Mount Sinai, New York, NY	
9:45 am – 10:30 am Hippocampal Sclerosis Sclerosis (CARTS)	s of Aging (HS-Aging) and <u>C</u> erebral <u>A</u> ge- <u>R</u> elated <u>T</u> DP-43 with	
3.43 um 10.30 um	Peter T. Nelson, MD, PhD	
	University of Kentucky, Lexington, KY	
10:30 am – 11:00 am REFRESHMENT BREAI	REFRESHMENT BREAK – Regency Ballroom Foyer	
Neuropathology in Ag	ed: Perspectives from Rush Cohorts	
11:00 am – 11:45 am	Julie A. Schneider, MD, MS	
	Rush University Medical Center, Chicago, IL	
11:45 am – 12:30 pm Neuropathology of Ag	ling: Panel Discussion	
12:30 pm – 1:30 pm LUNCH ON OWN	LUNCH ON OWN	
Update on Mo	lecular Analysis of Brain Tumors	
Molecular Analysis of		
1:30 pm – 2:15 pm	Kenneth Aldape, MD	
	er for Cancer Research, National Cancer Institute, Bethesda, MD	
IDH-Mutant Gliomas	Devial I Duet MD DID	
2:15 pm – 3:00 pm	Daniel J. Brat, MD, PhD Northwestern University, Chicago, IL	
3:00 pm – 3:30 pm REFRESHMENT BREAI	REFRESHMENT BREAK – Regency Ballroom Foyer	
Diffuse Gliomas of Chi	ildhood	
3:30 pm – 4:15 pm	Cynthia Hawkins, MD, PhD, FRCPC	
	University of Toronto, Toronto, ON, Canada	
Meningioma - Molecu	lar Pathogenesis and Current Biomarkers	
4:15 pm – 5:00 pm	Felix Sahm, MD	
	University of Heidelberg, Heidelberg, Germany	
5:00 pm – 5:10 pm Closing Remarks and C	CME Post-Test	
5:30 pm – 7:30 pm ANNUAL RECEPTION - All Attendees Welcom	– Kentucky Suite & Park Suite e	

2018 ABSTRACTS AND NAMED LECTURES, DAY 1

Director: Elizabeth J. Cochran, MD

	Friday, Jur	ne 8, 2018		
CONTINENTAL BREAKFAST		L BREAKFAST	Gulfstream-Hialeah &	
7:00 am – 8:00 am	Regency Bal	Regency Ballroom Foyer		
	Regency South Ballroom	Regency Ballroom		
	PLATFORM 1	PLATFORM 2		
8:00 am – 10:00 am	Developmental/Pediatric	Tumors: Adult Diffuse		
	Abstracts #1-8	Gliomas		
		Abstracts #9-16		
10:00 am – 10:30 am		REFRESHMENT BREAK		
10.00 um 10.50 um		Regency Ballroom Foyer		
		Ballroom		
		ECTURE		
		cture of Aging-Related		
10:30 am – 11:30 am	Neuropathologies: S	potlight on Microglia		
		Dhilim I Do Ingar MAD DhD		
	Columbia University N	Philip L. De Jager, MD, PhD		
		Columbia University Medical Center, New York, NY MERITORIOUS AWARD		
11:30 am – 11:45 am	Honoring Hannah C. Kinney,			
11.50 am - 11.45 am	Presented by Rebecca D. Folkerth, MD		Friday, June 8 8:00 am – 5:00 pm	
		BUSINESS MEETING I		
11:45 am – 12:45 pm	All Members Welcome			
12:45 pm – 2:00 pm		ON OWN		
	Regency South Ballroom	Regency Ballroom		
	PLATFORM 3	PLATFORM 4		
	Neurodegenerative:	Tumors: Other Glial,		
2:00 pm – 4:00 pm	Alzheimer	Neuroepithelial and		
	Abstracts #17-24	Embryonal		
		Abstracts #25-32		
4:00 pm – 4:45 pm	POSTER VIEWING & REFRESHMENT BREAK			
р		and; Regency Ballroom Foyer		
		Ballroom		
	DEARMOND LECTURE			
	Dysregulated Metabolism in the Pathogenesis of			
4:45 pm – 5:45 pm				
Suzanne M. de la Monte, MD, MPH Warren Alpert Medical School of Brown University,				
	warren Alpere Wedica	Providence, RI		
		i Tovidence, M		

2018 ABSTRACTS AND NAMED LECTURES, DAY 2

Director: Elizabeth J. Cochran, MD

	Saturday, Jur	ne 9, 2018		
7:00 am – 8:00 am	CONTINENT	CONTINENTAL BREAKFAST Regency Ballroom Foyer		
7:00 am = 8:00 am	Regency B			
	Regency Ballroom	Regency South Ballroom		
8:00 am – 10:00 am	PLATFORM 5	PLATFORM 6		
	CTE/Trauma	Muscle/Other		
	Abstracts #108-115	Abstracts #116-123		
10:00 am – 10:30 am	REFRESHI			
Regency Ballroom Foyer				
		y Ballroom		
		LECTURE		
10:20 11:20		Reading Tea Leaves:		
10:30 am – 11:30 am	Patterns of injury in the	Pediatric Nervous System		
	NIVC Office of Chief MA	Rebecca D. Folkerth, MD		
		edical Examiner, New York, NY		
11:30 am – 11:45 am		MERITORIOUS AWARD		
11.30 alli – 11.45 alli		Honoring Brian N. Harding, MA, DPhil, BM, BCh, FRCPath Presented by David W. Ellison, MD, PhD		
		Presented by David W. Ellison, MD, PhD BUSINESS MEETING II		
11:45 am – 12:45 pm		All Members Welcome		
12:45 pm – 2:00 pm	LUNCH	LUNCH ON OWN		
	Regency Ballroom	Regency South Ballroom		
	PLATFORM 7	PLATFORM 8		
2:00 pm – 4:00 pm	Neurodegenerative:	Tumors: Other	Posters #140-218	
2.00 pm = 4.00 pm	FTLD/Lewy body/Prion	Abstracts #132-139	Saturday, June 9	
	Abstracts #124-131	Abstracts #124-131		
4:00 pm – 4:45 pm		POSTER VIEWING & REFRESHMENT BREAK		
		Gulfstream-Hialeah & Keeneland; Regency Ballroom Foyer		
		y Ballroom		
		What Every Neuropathologist Needs to Know:		
4:45 pm – 5:15 pm	-	What Practical Information Every Neuropathologist		
	Needs to Know Abo			
	Mayo Clinic, Rochester, MN			
5:15 pm – 5:45 pm	What Every Neuropathologist Needs to Know:			
	Update on c-IMPACT-NOW Daniel J. Brat, MD, PhD			
	North			
		western University, Chicago, IL y Ballroom		
		SLIDE SESSION		
8:00 pm – 11:00 pm	11 Cases Moderated by Caterina Giannini, MD, PhD			
0.00 pm 11.00 pm	and Rebecca D. Folkerth, MD			
	and nebeccu	= : : 3 .		

2018 PRESIDENTIAL SYMPOSIUM

Emerging Technologies in Neuropathology

Co-Directors: Elizabeth J. Cochran, MD & Daniel J. Brat, MD, PhD

Sunday, June 10, 2018			
Time:	Regency Ballroom		
7:00 am – 8:00 am	CONTINENTAL BREAKFAST		
7.00 am = 8.00 am	Regency Ballroom Foyer		
8:00 am – 8:05 am	Welcome and CME Pre-Test		
	Elizabeth J. Cochran, MD		
	Medical College of Wisconsin, Milwaukee, WI		
	Evolution of Diagnostic Techniques in Pathology: Moving Beyond the Microscope		
8:05 am – 8:30 am	Elizabeth J. Cochran, MD		
	Medical College of Wisconsin, Milwaukee, WI		
	MOORE LECTURE		
8:30 am – 9:15 am	Deciphering Single-Cell Regulatory Programs in Adult and Pediatric Gliomas		
	Mario L. Suvà, MD, PhD		
	Harvard Medical School, Boston, MA		
9:15 am – 10:00 am	Teleneuropathology for Intraoperative Consultation		
	Clayton A. Wiley, MD, PhD		
10:00 am 10:20 am	University of Pittsburgh, Pittsburgh, PA AANP AWARDS PRESENTATIONS		
10:00 am – 10:30 am			
10:30 am – 10:45 am	REFRESHMENT BREAK		
	Regency Ballroom Foyer		
10:45 am – 11:30 am	Liquid Biopsy in Cancers of the Central Nervous System Matthias Holdhoff, MD, PhD		
	John Hopkins University School of Medicine, Baltimore, MD		
	Deep Learning Neural Networks		
11:30 am – 12:15 pm	Lee Cooper, PhD, MS		
11.50 am – 12.15 pm	Emory University School of Medicine, Atlanta, GA		
12:15 pm – 12:30 pm	INSTALLATION OF NEW OFFICERS AND ADJOURNMENT		
12.13 hiii – 12.30 hiii	INSTALLATION OF NEW OFFICERS AND ADJOURNMENT		



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Special Course

Thursday, June 7, 2018

Learning Objectives:

- 1. Summarize recent advances in neurodegenerative disease neuropathology.
- 2. Articulate advancements in the molecular analysis of brain tumors.

SPECIAL COURSE

PET Imaging of Amyloid and Tau in Human Aging

Time: 8:15 am - 9:00 am

Elizabeth Mormino, PhD, Assistant Professor, Department of Neurology, Stanford

I. Learning Objectives

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

- 1. Describe the prevalence of Alzheimer disease (AD) neuropathology (amyloid and tau) among individuals without clinical symptoms.
- 2. Explain how molecular imaging can be used to investigate AD pathophysiology.
- 3. Summarize imaging predictors of early cognitive decline.

II. Abstract & Relevant References

The accumulation of beta-amyloid (A β) begins at least a decade before the clinical onset of Alzheimer's disease (AD) dementia, providing an opportunity to understand early disease processes in clinically normal (CN) older individuals. Work across multiple research groups has shown that CN older individuals with abnormal levels of A β show significant cognitive decline and risk of progression to clinical impairment compared to A β - CN. The ability to measure the brain accumulation of the other pathological hallmark of AD, the aggregation of Tau, has recently become available and integrated into studies of aging. Importantly, Tau PET elevation in a set of restricted brain regions--the medial temporal lobe and inferior temporal cortex—have been found in A β + CN and are associated with short term memory decline. The opportunity to investigate the pathophysiological processes of AD in clinically asymptomatic individuals provides an ideal opportunity to test whether disease-modifying treatments applied during the preclinical stage of AD will prevent the clinical manifestation of AD dementia.

References:

1. Sperling, Mormino, Johnson, 2014. PMID: 25442939

2. Jack et al, 2018. PMID: 29538647

Donohue et al, 2017. PMID: 28609533
 Nelson et al, 2012. PMID: 22487856

5. Jansen et al, 2017. PMID: 25988462

III. Faculty Biography

Dr. Beth Mormino is a neuroscientist that applies imaging techniques in clinical populations to understand disease progression and the neural correlates of behavioral and cognitive changes that occur in disease. Her primary research focus is on the intersection between Alzheimer's disease (AD) and human aging, and her research program is focused on the use of multimodality imaging to improve the ability to monitor disease progression, detect pathological changes as early as possible, and to understand mechanisms underlying the behavior manifestations of neurodegenerative diseases.

Dr. Mormino completed a PhD in Neuroscience at UC Berkeley in the laboratory of Dr. William Jagust, where she performed some of the initial studies applying Amyloid PET with the tracer PIB to clinically normal older individuals. This initial work provided evidence that the pathophysiological processes of Alzheimer's disease begin years before clinical symptoms and are associated with subtle changes to brain regions critical for memory. During her postdoctoral fellowship with Drs. Reisa Sperling and Keith Johnson at Massachusetts General Hospital she used multimodal imaging techniques to understand longitudinal cognitive changes among individuals classified as preclinical AD. In 2017, Dr. Mormino joined the faculty at Stanford University in the department of Neurology and Neurological Sciences. Her recent work has focused on the quickly developing field of Tau PET imaging, a critical technology for understanding the earliest accumulation of this hallmark pathological feature of AD.

SPECIAL COURSE

Primary Age-Related Tauopathy (PART): Concepts, Neuropathology, Advances and Questions

Time: 9:00 am - 9:45 am

John F. Crary, MD, PhD, Associate Professor, Department of Pathology, Fishberg Department of Neuroscience, Friedman Brain Institute, Ronald M. Loeb Center for Alzheimer's Disease, Icahn School of Medicine at Mount Sinai

I. Learning Objectives

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

- 1. Summarize the key neuropathological features of primary age-related tauopathy (PART).
- 2. Distinguish primary age-related tauopathy from Alzheimer disease and other tauopathies.
- 3. Assess the clinical implications of primary age-related tauopathy in patients.

II. Abstract & Relevant References

Recent consensus criteria have been advanced by a large group of neuropathologists defining a new category of Alzheimer disease-type (AD) neuropathologic change. Individuals with this disorder, now termed primary agerelated tauopathy (PART), develop AD-type neurofibrillary tangles (NFT) in the absence of amyloid-beta peptide containing-plaques. Patients with PART may have normal cognition, amnestic mild cognitive impairment (aMCI), or an amnestic dementia. The prevalence of PART dementia is unclear, with estimates ranging from 1-7%. The objective of this lecture is to review the clinical, neuropathological, molecular and genetic features that distinguish PART from other tauopathies. The lecture will also explore the controversial relationship between PART and AD.

References:

- Crary JF, Trojanowski JQ, Schneider JA, Abisambra JF, Abner EL, Alafuzoff I, Arnold SE, Attems J, Beach TG, Bigio EH, Cairns NJ, Dickson DW, Gearing M, Grinberg LT, Hof PR, Hyman BT, Jellinger K, Jicha GA, Kovacs GG, Knopman DS, Kofler J, Kukull WA, Mackenzie IR, Masliah E, McKee A, Montine TJ, Murray ME, Neltner JH, Santa-Maria I, Seeley WW, Serrano-Pozo A, Shelanski ML, Stein T, Takao M, Thal DR, Toledo JB, Troncoso JC, Vonsattel JP, White CL, 3rd, Wisniewski T, Woltjer RL, Yamada M, Nelson PT. Primary age-related tauopathy (PART): a common pathology associated with human aging. Acta neuropathologica. 2014;128(6):755-66. Epub 2014/10/29. doi: 10.1007/s00401-014-1349-0. PubMed PMID: 25348064; PMCID: 4257842.
- 2. Crary JF. Primary age-related tauopathy and the amyloid cascade hypothesis: the exception that proves the rule? J Neurol Neuromedicine. 2016;1(6):53-7. PubMed PMID: 27819070; PMCID: PMC5094182.
- 3. Besser LM, Crary JF, Mock C, Kukull WA. Comparison of symptomatic and asymptomatic persons with primary age-related tauopathy. Neurology. 2017. doi: 10.1212/WNL.000000000004521. PubMed PMID: 28916532.
- 4. Kovacs GG, Ferrer I, Grinberg LT, Alafuzoff I, Attems J, Budka H, Cairns NJ, Crary JF, Duyckaerts C, Ghetti B, Halliday GM, Ironside JW, Love S, Mackenzie IR, Munoz DG, Murray EM, Nelson PT, Takahashi RH, Trojanowski JQ, Ansorge O, Arzberger T, Baborie A, Beach TG, Bieniek KF, Bigio EH, Bodi I, Dugger BN, Feany M, Gelpi E, Gentleman S, Giaccone G, Hatanpaa KJ, Heale R, Hof PR, Hofer M, Hortobagyi T, Jellinger K, Jicha GA, Ince P, Kofler J, Kövari E, Kril JJ, Mann DM, Matej R, McKee AC, McLean C, Milenkovic I, Montine TJ, Murayama S, Lee EB, Rahimi J, Rodriguez RD, Rozemuller A, Schneider JA, Schultz C, Seeley W, Seilhean D, Smith C, Tagliavini F, Takao M, Thal DR, Toledo JB, Tolnay M, Troncoso JC, Vinters HV, Weis S, Wharton SB, White CLI, Wisniewski T, Woulfe JM, Yamada M, Dickson DW. Aging-related tau astrogliopathy (ARTAG): harmonized evaluation strategy. Acta neuropathologica. 2015;DOI 10.1007/s00401-015-1509-x.
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- tauopathy, different from classical sporadic Alzheimer disease. Acta neuropathologica. 2015;129(5):757-62. Epub 2015/03/18. doi: 10.1007/s00401-015-1407-2. PubMed PMID: 25778618.
- 6. Nelson PT, Trojanowski JQ, Abner EL, Al-Janabi OM, Jicha GA, Schmitt FA, Smith CD, Fardo DW, Wang WX, Kryscio RJ, Neltner JH, Kukull WA, Cykowski MD, Van Eldik LJ, Ighodaro ET. "New Old Pathologies": AD, PART, and Cerebral Age-Related TDP-43 With Sclerosis (CARTS). Journal of neuropathology and experimental neurology. 2016;75(6):482-98. Epub 2016/05/23. doi: 10.1093/jnen/nlw033. PubMed PMID: 27209644.

III. Faculty Biography

Dr. John F. Crary received his MD-PhD training at SUNY Downstate Medical Center, Brooklyn, NY. He trained in anatomic and neuropathology at Columbia University Medical Center, New York, NY. He is now a practicing neuropathologist and associate professor at the Icahn School of Medicine at Mount Sinai, New York, NY, where he directs an NIH-funded translational research program focused on tauopathies. Dr. Crary has published widely on neurodegenerative disease, but is best known for his work on primary age-related tauopathy. He is a member of the Rainwater Charitable Foundation's Tau Consortium. He has received a number of awards, including the SUNY Chancellor's Award for Student Excellence, American Academy of Neurology Medical Student Prize for Excellence in Neurology. He is an Alexander Saint-Amand Scholar and Louis V. Gerstner Jr. Scholar. He serves on the editorial board of Acta Neuropathologica, is a member of the Chronic Effects of Neurotrauma Consortium Data Monitoring Committee and is a frequent ad hoc member of the Cellular and Molecular Neurodegeneration study section.

SPECIAL COURSE

Hippocampal Sclerosis of Aging (HS-Aging) and Cerebral Age-Related TDP-43 with Sclerosis (CARTS)

Time: 9:45 am - 10:30 am

Peter T. Nelson, MD, PhD, Professor of Pathology, University of Kentucky

I. Learning Objectives

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

- 1. Describe neuropathologic features of HS-Aging/CARTS.
- 2. Discuss the overlap of features between HS-Aging/CARTS and other neuropathologic disorders and how to differentiate between these entities.
- 3. Outline evidence for TDP-43 as a biomarker for HS-Aging/CARTS.

II. Abstract & Relevant References

Hippocampal sclerosis of aging (HS-Aging), which we refer to using the terminology Cerebral Age-Related TDP-43 with Sclerosis (CARTS), is a subtype of dementia-related disease. The published literature on HS-Aging/CARTS provides strong evidence of a prevalent, high-morbidity, and under-appreciated brain disease of aging. A full understanding of this pathology-defined disease requires an appreciation of the broader field of clinicalpathological studies in advanced age, a field which has seen dramatic changes in recent years. The current definition of HS-Aging are as follows: neuronal loss and gliosis in the hippocampal formation that is out of proportion to Alzheimer's disease (AD)-type, or any other type, of hippocampal pathology. HS-Aging is also strongly associated with TDP-43 pathology. HS-Aging pathology appears to be most prevalent in the oldest-old: autopsy series indicate that 5-30% of nonagenarians' brains harbor HS-Aging pathology. Among prior studies, differences in study design have contributed to the study-to-study variability in reported disease prevalence. The presence of HS-Aging pathology correlates with significant cognitive impairment which is often misdiagnosed as AD clinically. The antemortem diagnosis is further confounded by other diseases linked to hippocampal atrophy (and which often have TDP-43 pathology), including frontotemporal lobar degeneration and AD. Recent advances characterizing the neurocognitive profile of HS-Aging patients have begun to provide clues that may help identify living individuals with HS-Aging pathology. Structural brain imaging studies of research subjects followed to autopsy reveal hippocampal atrophy that is substantially greater in people with eventual HS-Aging pathology, compared to those with AD pathology alone. An overview of genetics is presented. We also help convey strategies for differential diagnosis in the context of brain aging, which is often complicated by "mixed" pathology subtypes. We conclude with a discussion of why an overhaul of terminology is required, and why CARTS may serve as an improvement over any terminology that includes "hippocampal sclerosis".

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- "New Old Pathologies": AD, PART, and Cerebral Age-Related TDP-43 With Sclerosis (CARTS).
 Nelson PT, Trojanowski JQ, Abner EL, Al-Janabi OM, Jicha GA, Schmitt FA, Smith CD, Fardo DW, Wang WX, Kryscio RJ, Neltner JH, Kukull WA, Cykowski MD, Van Eldik LJ, Ighodaro ET.
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III. Faculty Biography

PMID: 23864344

PMID: 24899141

Pete Nelson is the Director of the Neuropathology Division of the Pathology Department, University of Kentucky Medical Center, and he also directs the brain bank and performs brain autopsies for the Neuropathology Core of the University of Kentucky Alzheimer's Disease Center. Pete is an experimental neuropathologist interested in neurodegenerative diseases. Following a PhD in Dr. Clif Saper's lab focusing on animal models of neurodegeneration, Pete was trained in neurodegenerative disease neuropathology by Dr. John Trojanowski and in molecular biology by Dr. Zissimos Mourelatos at the University of Pennsylvania. Pete's work at the University of Kentucky has provided insights into the associative impact of pathology in the aged brain, and how genetics may play a role in neurodegenerative diseases. Pete contributed to papers on primary age-related tauopathy (PART), cerebral age-related TDP-43 with sclerosis (CARTS), and brain arteriolosclerosis. Pete also helped produce scholarship defining the neuropathologic features underlying diabetes (it's not Alzheimer's disease!), and the disease substrates that underlie subjective memory complaints and MCI (also often not Alzheimer's disease!). Teasing apart the features that delineate each AD mimicking disease provides an exciting opportunity to obtain new disease-modifying or disease-prevention strategies. In addition to duties as a neuropathologist, Pete is an experimental researcher focusing on the molecular neurochemistry of the human brain — in health and in neurodegenerative disease — particularly in the context of RNA biology. Pete's career is motivated partly by his own grandmother, Sylvia "Tib" Becker, who died with Alzheimer's disease.

SPECIAL COURSE

Neuropathology in Aged: Perspectives from Rush Cohorts

Time: 11:00 am - 11:45 am

Julie A. Schneider, MD, MS, The Deborah R. And Edgar D. Jannotta Presidential Professor of Pathology and Neurological Sciences, Associate Director, Rush Alzheimer's Disease Center, Rush University Medical Center

I. Learning Objectives

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

- 1. List the common mixed and single pathologies that underlie Alzheimer's dementia in older persons.
- 2. Describe clinical and pathologic factors associated with resistance and resilience to AD dementia in aging.
- 3. Discuss implications of pathologic heterogeneity underlying Alzheimer's dementia for public health and treatment of dementia in aging.

II. Abstract & Relevant References

"Alzheimer's disease", considered the most common cause of dementia in aging, is a term often used to encompass both a clinical syndrome as well as the pathologic basis of this syndrome (amyloid plaques and tau neurofibrillary tangles); however new guidelines propose nomenclature that separates the pathologic disease from the clinical syndrome, the latter of which may be caused by non-AD or multiple brain pathologies. Alzheimer's disease now refers to the specific pathologies of plaques and tangles, whereas the clinical syndrome of slowly progressive memory loss evolving into impairment of other cognitive domains is referred to as Alzheimer's dementia. The underlying pathologic basis of the clinical syndrome of Alzheimer's dementia especially in the old – is often confirmed to be Alzheimer's disease, however it is quite commonly the result of mixed or multiple comorbid pathologies. This is recognized in current criteria for the pathologic diagnosis of AD.² Moreover, it is well known that Alzheimer's disease can be present in older persons without dementia or even any cognitive impairment. Thus the aging brain may be one without significant pathology, or have mild, moderate, severe pathology, or single or many pathologies and the person may or may not exhibit symptoms from these pathologies. Prospective longitudinal studies have shown that older persons who die with Alzheimer's dementia are often confirmed to have a pathologic diagnosis of Alzheimer's disease.^{3,4} In an important minority, the dementia can be attributed to vascular or other degenerative diseases. In addition, in those confirmed to have a pathologic diagnosis of AD, studies show that less than half will have only AD pathology underlying their dementia The most common co-morbidities are vascular(macroinfarcts, microinfarcts, atherosclerosis, arteriolosclerosis, and cerebral amyloid angiopathy), TDP-43 pathology, hippocampal sclerosis, and Lewy bodies. These pathologies each add to the likelihood of a dementia diagnosis during life. While some have unique clinical signs and symptoms when presenting in isolation; these may overlap and/or be overshadowed by those caused by the AD pathology, making it difficult to determine which pathologies are contributing to the dementia. Moreover, all of these pathologies can impair episodic memory, the clinical hallmark of Alzheimer's disease. The aging brain is rarely without any pathologies³ and in about a third of older persons there is a pathologic diagnosis of AD^{3,5} in spite of normal cognition. This is often referred to as "reserve". Older persons often have not only Alzheimer;s Disease pathology but also "subclinical" vascular pathology, TDP-43 pathology, hippocampal sclerosis, and Lewy bodies. The more severe pathology and the greater numbers of pathologies in a brain, the more likely a person will exhibit dementia. Other life style, behavioral, medical and genetic factors also are likely to play a role not only in the level of pathology (resistance) but your resilence to that pathology.⁶

References:

1. Jack CR Jr., Bennett DA, Blennow K, Carrillo MC, Dunn B, Haeberlein SB, Holtzman DM, Jagust W, Jessen F, Karlawish J, Liu E, Molinuevo JL, Montine T, Phelps C, Rankin KP, Rowe CC, Scheltens P, Siemers E, Snyder HM, Sperling R; Contributors. NIA-AA Research Framework: Toward a biological definition of Alzheimer's disease. Alzheimers Dement. 2018 Apr;14(4):535-562.

- 2. Montine TJ, Phelps CH, Beach TG, et al. National Institute on Aging-Alzheimer's Association guidelines for the neuropathologic assessment of Alzheimer's disease: a practical approach. Acta neuropathologica 2012;123:1-11.
- 3. Kapasi A, DeCarli C, Schneider JA. Impact of multiple pathologies on the threshold for clinically overt dementia. Acta Neuropathol. 2017 Aug;134(2):171-186.
- 4. Rahimi J, Kovacs GG. Prevalence of mixed pathologies in the aging brain. Alzheimers Res Ther. 2014 Nov 21;6(9):82. doi: 10.1186/s13195-014-0082-1. eCollection 2014.
- 5. Petrie EC1, Cross DJ, Galasko D, et al. Preclinical evidence of Alzheimer changes: convergent cerebrospinal fluid biomarker and fluorodeoxyglucose positron emission tomography findings. Arch Neurol. 2009 May;66(5):632-7. doi: 10.1001/archneurol.2009.59.
- 6. Arenaza-Urquijo EM, Vemuri P. Resistance vs resilience to Alzheimer disease: Clarifying terminology for preclinical studies. Neurology. 2018 Mar 28.

III. Faculty Biography

Dr. Julie A. Schneider is the The Deborah R. And Edgar D. Jannotta Presidential Professor of Pathology (Neuropathology) and Neurological Sciences, and Associate Director at the Rush Alzheimer's Disease Center, at Rush University Medical Center. She completed her Neurology residency at the University of Chicago and Neuropathology fellowship at Emory University in Atlanta and is board certified in both specialties. Dr. Schneider is certified in Geriatric Neurology, and has a Master's Degree in Clinical Research with a focus in Epidemiology. She is the Neuropathology Core Leader of the Rush Alzheimer's Disease Center and the senior neuropathologist for multiple studies including the Religious Orders Study, Rush Memory and Aging Project, and Rush Minority Aging Research Study, Rush Latino Core, and NCRAD (National Cell Repository for Alzheimer's disease). Schneider has provided peer review for over 25 journals; has been invited to multiple journal editorial boards; and has provided numerous grant peer reviews for the National Institutes of Health, Alzheimer's Association, and over 5 other funding agencies. She has served on numerous scientific and external national and international advisory boards for academia and industry; and has presented findings from her research both nationally and internationally. Dr. Schneider has extensive experience with clinical-pathologic epidemiologic studies of aging and dementia and has over 250 peer-reviewed publications and 4 book chapters. She also has extensive experience collaborating with researchers, participating in multicenter grants and initiatives, and partnerships with industry to advance science. The foundation of Dr. Schneider's research is the exploration of pathologic factors in the clinical expression of cognitive and motor decline in aging. Her work focuses on Alzheimer's and mixed pathologies, vascular pathologies, and their role in cognitive decline, and transitions from normality to MCI, clinical AD and other dementias. More recently, Dr. Schneider has been investigating TDP-43 and hippocampal sclerosis pathologies in aging and dementia, and linking pathology with neuroimaging to inform on disease pathogenesis and potential biomarkers. Finally, she collaborates with numerous investigators in studies linking risk factors (e.g. genetic, diet and life style, neurobehavioral, and health related), neuroimaging and peripheral biomarkers, and other biochemical/molecular factors with neuropathology to ascertain mechanisms of susceptibility, resistance and resilience to age-related cognitive impairment.

SPECIAL COURSE

Molecular Analysis of Ependymoma

Time: 1:30 pm - 2:15 pm

Kenneth Aldape, MD, Center for Cancer Research, National Cancer Institute, Bethesda, MD

I. Learning Objectives

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

- 1. Explain the role of integrated molecular and histological criteria in the classification of ependymoma.
- 2. Identify the major subgroups of supratentorial and posterior fossa ependymoma.
- 3. Describe recently defined molecular platforms and immunohistochemical tests that can be used to delineate ependymoma subgroups.

II. Abstract & Relevant References

Ependymoma is a histologically defined intrinsic tumor that involves the three major anatomic compartments (supratentorial brain, posterior fossa, and spinal cord) of the central nervous system and affects both children and adults. The current standard for diagnosis is primarily histological, with the recent addition of one molecular subgroup within supratentorial ependymomas. The consensus on the prognostic ability of histopathological grading criteria to risk-stratify patients has not been reached, and the use of molecular or tumor-specific immunohistochemical markers to subgroup tumors is only now emerging. Recent advances in the biological characterization of ependymal tumors have demonstrated the existence of multiple clinically, demographically, and molecularly distinct entities, with distinct subgroups within each anatomic compartment, identified by specific genomic/epigenomic events. These findings offer new opportunities to create a precise, reliable, and objective platform for stratification of ependymoma patients, and the potential for altering therapeutic decisions based on molecular features. This talk will discuss the current consensus on the molecular subgroups of intracranial ependymoma (WHO Grade II/III) in children and adults, to place recent molecular findings into a diagnostic context.

References:

- 1. Godfraind C, Kaczmarska JM, Kocak M, Dalton J, Wright KD, Sanford RA, Boop FA, Gajjar A, Merchant TE, Ellison DW. Distinct disease-risk groups in pediatric supratentorial and posterior fossa ependymomas. Acta Neuropathol. 2012;124:247–257
- 2. Johnson RA, Wright KD, Poppleton H, Mohankumar KM, Finkelstein D, Pounds SB, Rand V, Leary SE, White E, Eden C, et al. Cross-species genomics matches driver mutations and cell compartments to model ependymoma. Nature. 2010;466:632–636.
- 3. Mack SC, Witt H, Piro RM, Gu L, Zuyderduyn S, Stutz AM, Wang X, Gallo M, Garzia L, Zayne K, et al. Epigenomic alterations define lethal CIMP-positive ependymomas of infancy. Nature. 2014
- 4. Pajtler KW, Witt H, Sill M, Jones DT, Hovestadt V, Kratochwil F, Wani K, Tatevossian R, Punchihewa C, Johann P, et al. Molecular classification of ependymal tumors across all CNS compartments, histopathological grades, and age groups. Cancer Cell. 2015;27:728–743.
- 5. Bayliss J, Mukherjee P, Lu C, Jain SU, Chung C, Martinez D, Sabari B, Margol AS, Panwalkar P, Parolia A, Pekmezci M, McEachin RC, Cieslik M, Tamrazi B, Garcia BA, La Rocca G, Santi M, Lewis PW, Hawkins C, Melnick A, David Allis C, Thompson CB, Chinnaiyan AM, Judkins AR, Venneti S. Lowered H3K27me3 and DNA hypomethylation define poorly prognostic pediatric posterior fossa ependymomas. Sci Transl Med. 2016 Nov 23;8(366).

III. Faculty Biography

Kenneth Aldape is currently the Chief of the Laboratory of Pathology at the Natiuonal Cancer Institute. Previously he held faculty positions at the University of California, San Francisco, and MD Anderson Cancer Center and Princess Margaret Cancer Centre. He focusses on genomics and molecular diagnostics of primary brain tumors. As a neuropathologist, is involved in the integration of biologic tumor signatures into clinical use for brain tumor classification.

SPECIAL COURSE

IDH-Mutant Gliomas

Time: 2:15 pm - 3:00 pm

Daniel J. Brat, MD, PhD, Magerstadt Professor and Chair, Department of Pathology, Northwestern Feinberg School of Medicine

I. Learning Objectives

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

- 1. Describe the markers that are currently used to establish the diagnosis of IDH-mutant diffusely infiltrating astrocytomas.
- 2. Describe the markers that are currently used to establish the diagnosis of oligodendroglioma.
- 3. Explain the gaps that remain in our grading scheme following the reclassification of diffuse gliomas in the WHO 2016.

II. Abstract & Relevant References

Molecular-genetic analysis is now an integral component of contemporary surgical neuropathology. . A critical conceptual shift that materialized with the publication of the revised 4th edition of the WHO classification of CNS tumors was the incorporation of molecular alterations into the diagnosis of specific neoplastic entities, in distinction to the prior practice of recognizing them as mere association. In this new era, diagnostic impressions from microscopic examination of H&E stained slides are interpreted in the setting of molecular-genetic testing. The classification of gliomas has been re-structured with the discovery of *isocitrate dehydrogenase* (*IDH*) 1/2 mutations in the vast majority of lower grade infiltrating gliomas and secondary glioblastomas (GBM), with *IDH*-mutant astrocytomas further characterized by *TP53* and *ATRX* mutations. Whole-arm 1p/19q co-deletion in conjunction with *IDH* mutations now define oligodendrogliomas, which are also enriched for *CIC*, *FUBP1*, *PI3K*, *NOTCH1*, and *TERT-p* mutations. This lecture will summarize the current understanding of IDH-mutant gliomas.

References:

- 1. Cancer Genome Atlas Research N, Brat DJ, Verhaak RG, et al. Comprehensive, Integrative Genomic Analysis of Diffuse Lower-Grade Gliomas. N Engl J Med. 2015;372(26):2481-2498.
- 2. Eckel-Passow JE, Lachance DH, Molinaro AM, et al. Glioma Groups Based on 1p/19q, IDH, and TERT Promoter Mutations in Tumors. N Engl J Med. 2015;372(26):2499-2508.
- 3. Ceccarelli M, Barthel FP, Malta TM, et al. Molecular Profiling Reveals Biologically Discrete Subsets and Pathways of Progression in Diffuse Glioma. Cell. 2016;164(3):550-563.
- 4. Louis DN, Perry A, Reifenberger G, et al. The 2016 World Health Organization Classification of Tumors of the Central Nervous System: a summary. Acta Neuropathol. 2016;131(6):803-820.
- 5. Aoki K, Nakamura H, Suzuki H, et al. Prognostic relevance of genetic alterations in diffuse lower-grade gliomas. Neuro Oncol. 2018;20(1):66-77.
- 6. Weller M, Weber RG, Willscher E, et al. Molecular classification of diffuse cerebral WHO grade II/III gliomas using genome- and transcriptome-wide profiling improves stratification of prognostically distinct patient groups. Acta Neuropathol. 2015;129(5):679-693.
- 7. Cimino PJ, Zager M, McFerrin L, Wirsching HG, Bolouri H, Hentschel B, von Deimling A, Jones D, Reifenberger G, Weller M, Holland EC. Multidimensional scaling of diffuse gliomas: application to the 2016 World Health Organization classification system with prognostically relevant molecular subtype discovery. Acta Neuropathol Commun. 2017;5(1):39

III. Faculty Biography

Dr. Brat received his MD and PhD from the Mayo Medical and Graduate Schools and completed Residency in Anatomic Pathology and a Fellowship in Neuropathology at Johns Hopkins Hospital. Dr. Brat's is a practicing surgical neuropathologist with special expertise in neoplastic diseases. He also directs an NIH-funded basic and translational research lab that investigates mechanisms of glioma progression, including the contributions of hypoxia, genetics, tumor microenvironment and stem cells, using *Drosophila* and mice to model the human disease. He also has an interest in the *in silico* investigation of brain tumors and has used large-scale clinical and molecular databases, such as The Cancer Genome Atlas (TCGA), to address fundamental questions in human glioma behavior. He has over 18 years of experience in brain tumor research and has written more than 200 peer-reviewed manuscripts and reviews. Dr. Brat has served in leadership positions that oversee clinical practice and investigation in Oncology and Pathology, including the TCGA Glioblastoma and Lower Grade Gliomas Working Groups; the College of American Pathologists (CAP) Glioma Guidelines Committee; the Executive Council of the American Association of Neuropathologists; the Board of Directors for the Society of Neuro-oncology; the WHO Committee for Classification of Brain Tumors; and the AJCC Expert Panel. He is a member of the American Society for Clinical Investigation.

SPECIAL COURSE

Diffuse Gliomas of Childhood

Time: 3:30 pm - 4:15 pm

Cynthia Hawkins, MD, PhD, FRCPC, University of Toronto, Toronto, ON, Canada

I. Learning Objectives

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

- 1. Approach the diagnosis of pediatric gliomas using a combination of morphologic and molecular diagnostic techniques.
- 2. Describe the spectrum and relative frequency of molecular alterations in pediatric glioma.
- 3. Explain the difference between pediatric-type and adult type low grade gliomas and its significance in the management of pediatric gliomas.

II. Abstract & Relevant References

This lecture will provide an update on the molecular alterations found in low and high grade pediatric diffuse gliomas including: 1) the association with particular morphologies; 2) the prognostic implications; 3) the relative frequencies of these alterations; and 4) the differences from adult diffuse gliomas. It will also provide an approach to molecular investigation based on clinical features and morphologic appearance.

References:

- 1. Mackay et al. Integrated Molecular Meta-Analysis of 1,000 Pediatric High-Grade and Diffuse Intrinsic Pontine Glioma. Cancer Cell. 32(4):520-537. 2017
- 2. Lassaletta et al. Therapeutic and Prognostic Implications of BRAF V600E in Pediatric Low-Grade Gliomas. J Clin Oncol. 35(25):2934-2941, 2017
- 3. Lassaletta et al. An integrative molecular and genomic analysis of pediatric hemispheric low-grade gliomas: an update. Child's nervous system. 32(10): 1789-97, 2016
- 4. Ryall et al. Targeted detection of genetic alterations reveal the prognostic impact of H3K27M and MAPK pathway aberrations in paediatric thalamic glioma. Acta neuropathologica communications. 4(1): 93, 2016
- 5. Solomon et al. Diffuse Midline Gliomas with Histone H3-K27M Mutation: A Series of 47 Cases Assessing the Spectrum of Morphologic Variation and Associated Genetic Alterations. Brain Pathology. 26:569-80, 2016
- 6. Pratt et al. Circumscribed/non-diffuse histology confers a better prognosis in H3K27M-mutant gliomas. Acta Neuropath. 135(2): 299-301, 2018
- 7. Huse et al. Polymorphous low grade neuroepithelial tumor of the young (PLNTY): an epileptogenic neoplasm with oligodendroglioma like components, aberrant CD34 expression, and genetic alterations involving the MAP kinase pathway. Acta Neuropath. 133:417-29, 2017
- 8. Qaddoumi et al. Genetic alterations in uncommon low grade neuroepithelial tumors: BRAF, FGFR1, and MYB mutations occur at high frequency and align with morphology. Acta Neuropath 131:833-45, 2016

III. Faculty Biography

Dr. Cynthia Hawkins is a Neuropathologist and Senior Scientist at the Hospital for Sick Children and a Professor of Laboratory Medicine and Pathobiology at the University of Toronto, in Toronto, Canada. Her research focuses on molecular pathogenesis and therapeutics for pediatric gliomas as well as identification and clinical implementation of novel prognostic and therapeutic markers for pediatric brain tumors.

SPECIAL COURSE

Meningioma - Molecular Pathogenesis and Current Biomarkers

Time: 4:15 pm - 5:00 pm

Felix Sahm, MD, University of Heidelberg, Heidelberg, Germany

I. Learning Objectives

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

- 1. Recall the most frequently mutated genes in meningiomas and their association with histological subtypes.
- 2. Explain the concept of accumulation of molecular aberrations during meningioma progression.
- 3. Summarize the limitations of mutational and cytogenetic profiling as potential adjuncts in grading.

II. Abstract & Relevant References

Molecular markers are not yet included in the diagnostic approach to meningioma. However, there are associations between molecular aberrations and histological subtypes, WHO grade, and risk of recurrence. These emerging findings may result in an "integrated" approach to meningioma diagnostics.

The talk will first review the molecular concept of meningioma progression: Accumulation of molecular aberrations, including deletions of chromosomal arm 1p, chromosome 10, homozygous deletion of CDKN2A, TERT promoter mutations, and others are accompanied by increasing aggressiveness [1-4].

Then, the correlation of molecular findings and histological presentation will be summarized: AKT1/TRAF7 and SMO mutations in meningothelial meningioma, KLF4/TRAF7 in secretory meningioma, SMARCE1 in clear cell meningioma, BAP1 in rhabdoid meningioma, gain of chromosome 5 in angiomatous meningioma [5-9]. This part will also review the genomic features characteristic for meningiomas induced by radiation (NF2 fusions, double strand breaks) and progestin-associated meningioma (PIK3CA) [10, 11].

Subsequently, the potential and limitations of these molecular aberrations to assist in grading and identifying novel treatment targets will be discussed [12, 13].

Finally, more recent developments on the role of DNA methylation in identifying subgroups of meningioma with distinct risk of recurrence will be presented [14, 15].

References:

- 1. Perry, A., D.H. Gutmann, and G. Reifenberger, *Molecular pathogenesis of meningiomas*. J Neurooncol, 2004. **70**(2): p. 183-202.
- 2. Mawrin, C. and A. Perry, *Pathological classification and molecular genetics of meningiomas.* J Neurooncol, 2010. **99**(3): p. 379-91.
- 3. Louis, D.N., et al., *The 2016 World Health Organization Classification of Tumors of the Central Nervous System: a summary.* Acta Neuropathol, 2016. **131**(6): p. 803-20.
- 4. Peyre, M., et al., *De novo and secondary anaplastic meningiomas: a study of clinical and histomolecular prognostic factors.* Neuro Oncol, 2017.
- 5. Clark, V.E., et al., *Genomic analysis of non-NF2 meningiomas reveals mutations in TRAF7, KLF4, AKT1, and SMO*. Science, 2013. **339**(6123): p. 1077-80.
- 6. Brastianos, P.K., et al., *Genomic sequencing of meningiomas identifies oncogenic SMO and AKT1 mutations.* Nat Genet, 2013. **45**(3): p. 285-9.
- 7. Shankar, G.M., et al., *Germline and somatic BAP1 mutations in high-grade rhabdoid meningiomas*. Neuro Oncol, 2017. **19**(4): p. 535-545.

- 8. Smith, M.J., et al., Loss-of-function mutations in SMARCE1 cause an inherited disorder of multiple spinal meningiomas. Nat Genet, 2013. **45**(3): p. 295-8.
- 9. Abedalthagafi, M.S., et al., *Angiomatous meningiomas have a distinct genetic profile with multiple chromosomal polysomies including polysomy of chromosome 5.* Oncotarget, 2014. **5**(21): p. 10596-606.
- 10. Peyre, M., et al., Progestin-associated shift of meningioma mutational landscape. Ann Oncol, 2017.
- 11. Agnihotri, S., et al., *Therapeutic radiation for childhood cancer drives structural aberrations of NF2 in meningiomas.* Nat Commun, 2017. **8**(1): p. 186.
- 12. Sahm, F., et al., *TERT Promoter Mutations and Risk of Recurrence in Meningioma.* J Natl Cancer Inst, 2016. **108**(5).
- 13. Yuzawa, S., et al., *Clinical impact of targeted amplicon sequencing for meningioma as a practical clinical-sequencing system.* Mod Pathol, 2016. **29**(7): p. 708-16.
- 14. Olar, A., et al., Global epigenetic profiling identifies methylation subgroups associated with recurrence-free survival in meningioma. Acta Neuropathol, 2017. **133**(3): p. 431-444.
- 15. Sahm, F., et al., *DNA methylation-based classification and grading system for meningioma: a multicentre, retrospective analysis.* Lancet Oncol, 2017. **18**(5): p. 682-694.

III. Faculty Biography

Dr. Sahm graduated from University Heidelberg Medical School in 2009 after studying in Heidelberg, Boston, and London. He conducted the research for his MD thesis in the Department of Neurooncology Heidelberg (Prof Wolfgang Wick) in the group of Prof Michael Platten, working on glioma-promoting effects of tryptophan degradation (Opitz, Litzenburger, Sahm et al., Nature 2011). He subsequently started his residency in the Department of Neuropathology led by Prof Andres von Deimling at the University Hospital Heidelberg, being also affiliated with the Clinical Cooperation Unit Neuropathology at the German Cancer Research Center (DKFZ) as postdoc researcher. In 2016, he was board-certified in Neuropathology, which is an independent discipline in Germany and requires a minimum residency of six years. In parallel, he pursued his Habilitation under the mentorship of Prof Andreas von Deimling. This was completed by being awarded the venia legendi in Clinical Neuropathology by the Medical Faculty Heidelberg in 2017. He currently serves as consultant and head of molecular diagnostics at the Dept. of Neuropathology Heidelberg, providing molecular testing by panel sequencing and DNA methylation array for diagnostic routine and several clinical trials with collaboration partners Profs Wolfgang Wick, Michael Platten, and Stefan Pfister. His research focus is molecular pathology of glioma and meningioma, with special interest in identifying novel clinically meaningful subgroups, their respective markers and developing innovative diagnostic tools.



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Overview: Scientific Sessions

Friday, June 8, 2018 & Saturday, June 9, 2018

All abstracts of the papers presented in this program are published in the June 2018 issue of the *Journal of Neuropathology and Experimental Neurology.*

FRIDAY PLATFORMS 1 & 2

	Platform Session 1: Developmental/Pediatric Regency South Ballroom Moderators: David Nauen, MD, PhD; Brian Harding, DPhil, FRCPath	Platform Session 2: Tumors: Adult Diffuse Gliomas Regency Ballroom Moderators: Joanna Phillips, MD, PhD; Maria Martinez- Lage, MD
8:00 am – 8:15 am	1 GABAergic Neuronal Deficiency Patterns and Type 2 Potassium-Chloride Co-transporter immaturity in Human Focal Cortical Dysplasia Pengcheng Han, Cynthia Welsh, Michael Smith, Robert Schmidt, Steven Carroll	Prospective genomic testing in newly diagnosed glioblastoma facilitates trial analysis and biomarker discovery David Meredith, Mehdi Touat, Adrian Dubuc, Sarah Gaffey, Jack Geduldig, Shakti Ramkissoon, John de Groot, Evanthia Galanis, Mary Welch, Burt Nabors, Isabel Arrilaga-Romany, E. Antonio Chiocca, Sandro Santagata, David Schiff, Manmeet Ahluwalia, Howard Colman, Jan Drappatz, Brian Alexander, Patrick Wen, Keith Ligon
8:15 am – 8:30 am	Procused ion beam – scanning electron microscopy reveals distinct structural features and putative contacts of CA1 pyramidal neuron primary cilia Shu-Hsien Sheu, Pichet Adstamongkonkul, Amalia Pasolli, Song Pang, Justin Houser, Tomas Kirchhausen, Harald Hess, David Clapham	Capture-Based 1,200 Gene NGS Panel as a Comprehensive Tool for Classifying Gliomas on FFPE Tissue or Intraoperative Smear Preparations Megan Parilla, John Collins, Sabah Kadri, Sushant Patil, Jeremy Segal, Carrie Fitzpatrick, Peter Pytel
8:30 am – 8:45 am	Altered Gene Expression by Dentate Gyrus Granule Cells in the Development of Epilepsy David Nauen, Begum Choudhury, Andrew Wilson, Anne Li, Alaleh Azhir, Ben Langmead	Protein-based molecular profiling of gliomas using mass spectrometry Ugljesa Djuric, Jennifer Kao, Mike Papaioannou, Ihor Batruch, Almos Klekner, Ken Aldape, Phedias Diamandis
8:45 am – 9:00 am	4 In situ visualization of protein interactions reveals Cdc42's coordination of cytoskeletal pathways during dendrite morphogenesis Nima Sharifai, Daichi Kamiyama, Akira Chiba	DNA methylation of circulating tumor educated leukocytes as a biomarker of IDH1/2 mutation in diffuse gliomas Andreas Kloetgen, Jonathan Serrano, Seema Patel, Christopher Bowman, Guomiao Shen, David Zagzag, Matthias Karajannis, John Golfinos, Dimitris Placantonakis, Aristotelis Tsirigos, Andrew Chi, Matija Snuderl
9:00 am – 9:15 am	5 Blockade of potassium channel KCa3.1 in the post- treatment of neonatal hypoxic-ischemic brain injury Mirna Lechpammer, Verónica Martínez Cerdeño, Hilary Gonzales, Yen Tran, Philip Huebner, Heike Wulff, Robert Berman	Recurrent Unusual Patterns in Clinical Molecular Profiling of Adult Diffuse Gliomas Cristiane Ida, Cinthya Zepeda-Mendoza, Corinne Praska, Jessica Balcom, Kirsten Swanson, Emily Barr Fritcher, Joseph Parisi, Mark Jentoft, Katherine Geiersbach, Aditya Raghunathan, Caterina Giannini, Benjamin Kipp, Robert Jenkins
9:15 am – 9:30 am	6 Genetic mapping of diversity among developing brainstem motor neuron subtypes at single cell resolution Matthew Rose, Alon Gelber, Max Tischfield, Alan Tenney, Alicia Nugent, Phillip Ang, Sarah Izen, Matthew Bauer, Wentao Huang, Rahul Satija, Orit Rozenblatt-Rosen, Aviv Regev, Elizabeth Engle	Methylation-dependent suppression of Tissue Factor is a key contributor to the reduced malignancy of IDH1 mutant gliomas Dusten Unruh, Snezana Mirkov, Denise Scholtens, Jann Sarkaria, C. James, Craig Horbinski
9:30 am – 9:45 am	7 Complex Ependymal Remnants In Early-onset Tethered Cord Syndrome Mercia Bezerra Gondim, Laurie Ackerman, Daniel Fulkerson, Nucharin Supakul, Natasha Gibson, Alexander Vortmeyer	Increased HOXA5 expression provides a selective advantage for gain of whole chromosome 7 in proneural glioblastoma P.J. Cimino, Youngmi Kim, Hua-Jun Wu, Jes Alexander, Hans-Georg Wirsching, Frank Szulzewsky, Ken Pitter, Tatsuya Ozawa, Jiguang Wang, Julio Vazquez, Sonali Arora, Raul Rabadan, Ross Levine, Franziska Michor, Eric Holland
9:45 am – 10:00 am	8 Anti-Myelin Proteolipid Protein Antibodies Bind Cell Surface Proteins on Developing Neurons and Inhibit Their Differentiation Raymond Sobel, Mary Jane Eaton, Julian Hinojoza	16 Neuroradiology Concordance With Neuropathology In Predicting 1p/19q-Codeletion In IDH-Mutant Lower-Grade Gliomas Edward Kelly Mrachek, Prem Batchala, Sohil Patel, Camilo Fadul, David Schiff, Eli Williams, Maria-Beatriz Lopes

FRIDAY PLATFORMS 3 & 4

	Platform Session 3 Neurodegenerative: Alzheimer	Platform Session 4 Tumors: Other Glial, Neuroepithelial and Embryonal
	Regency South Ballroom Moderators: Ivana Delalle, MD, PhD; Janna Neltner, MD	Regency Ballroom Moderators: Jason Chiang, MD, PhD; Cynthia Hawkins, MD, PhD
2:00 pm – 2:15 pm	Faster Cognitive Decline in Alzheimer's Disease with Clinically-Unsuspected Lewy Body Disease Thomas Beach, Michael Malek-Ahmadi, Zamrini Edward, Marwan Sabbagh, Holly Shill, Charles Adler, Richard Caselli, Bryan Woodruff, Steven Rapscak, Geoff Ahern, Jiong Shi, John Caviness, Erika Driver-Dunckley, Shyamal Mehta, David Shprecher, Bryan Spann, Christine Belden, Kathryn Davis, Kathy Long, Lisa Nicholson, Anthony Intorcia, Michael Glass, Jessica Walker, Michael Callan, Jasmine Curry, Brett Cutler, Javon Oliver, Richard Arce, Geidy Serrano, Lucia Sue, Eric Reiman	Methylation Based Classification of Pleomorphic Xanthoastrocytoma Rachael Vaubel, Paul Decker, Alissa Caron, Jeanette Eckel Passow, Amulya NageswaraRao, Dan Lachance, Ian Parney, Fausto Rodriguez, Robert Jenkins, Caterina Giannini
2:15 pm – 2:30 pm	18 Clinico-neuropathological diagnostic differences across Hispanics, Blacks, and Non-Hispanic Whites with dementia Brittany Dugger, Teresa Filshtein, Lee-Way Jin, John Olichney, Sarah Farias, Luis Carvajal-Carmona, Paul Lott, Dan Mungas, Bruce Reed, Laurel Beckett, Charles DeCarli	The genomic landscape of anaplastic pleomorphic xanthoastrocytoma Joanna Phillips, Henry Gong, Katharine Chen, Nancy Joseph, Jessica Van Ziffle, Boris Bastian, Theodore Nicolaides, Tarik Tihan, Andrew Bollen, Perry Arie, Joseph Shieh, David Solomon
2:30 pm – 2:45 pm	19 Differential Involvement of the Nucleus Basalis of Meynert across Alzheimer's Disease Subtypes Fadi Hanna Al-Shaikh, Neill Graff-Radford, Amanda Liesinger, Kelly Ross, Nilufer Ertekin-Taner, Tanis Ferman, Owen Ross, Otto Pedraza, Ranjan Duara, Dennis Dickson, Melissa Murray	27 NF1-Associated SEGA-Like Tumor (NASLT): a neoplasm with a distinct phenotype and variable genetic features Doreen Palsgrove, Jacqueline Brosnan-Cashman, Caterina Giannini, Aditya Raghunathan, Mark Jentoft, Chetan Bettegowda, Murat Gokden, Ming-Tseh Lin, Christopher Heaphy, Fausto Rodriguez
2:45 pm – 3:00 pm	Quantification and distribution of neuropathologic changes in primary age-related tauopathy Jamie Walker, Timothy Richardson, Kurt Farrell, John Crary, Eileen Bigio, Edward Lee, Chan Foong, Ping Shang, Charles White	28 Astroblastoma Is A Molecularly Heterogeneous Tumor Comprised Of At Least Three Major Genetic Subgroups Norman Lehman, Eyas Hattab, Aisulu Usubalieva, Kirsteen Maclean, Joseph McElroy, Amy Hite, Paolo Fadda, Bret Mobley, Roger McLendon, Matthew Schneiderjan, Ralph Cooke, Tom Liu, Cynthia Timmers, Werner Paulus, Maria-Magdalena Georgescu, Marta Couce, Mohanpal Dulai, Jack Raisanen, Cheryl Palmer, Sariah Allen, Tong Lin, Brent Orr
3:00 pm – 3:15 pm	21 Methionine Sulfoxide Reductase-B3 Risk Allele Implicated In Alzheimer's Disease Affects Odds For Small Covert Brain Infarcts Laurent Benayoun, Jayandra Himali, Sarah Conner, Stephanie Adams, Sudha Seshadri, Ivana Delalle	Pediatric treatment-induced high-grade gliomas are enriched for a specific methylation subgroup and recurrent genomic abnormalities John Lucas, John DeSisto, Andrew Donson, Bridget Sanford, Gang Wu, Gregory Armstrong, Michael Arnold, Smita Bhatia, Patrick Flannery, Rakeb Lemma, Lakotah Hardie, Lindsey Hoffman, Kathleen Dorris, Arthur Liu, Nicholas Foreman, Rajeev Vibhakar, Kenneth Jones, Sariah Allen, Thomas Merchant, Adam Green, Brent Orr
3:15 pm – 3:30 pm	Brain Insulin Resistance as a Mediator of Vascular and White Matter Degeneration in AD: Role of ApoE4 Dose Suzanne de la Monte, Ming Tong, Gina Gallucci, Emine Yalcin, Edward Stopa, Miles Miller, Jared Kay	The Genomic Landscape of Ganglioglioma Melike Pekmezci, Javier Villaneuva-Meyer, Benjamin Goode, Jessica Van Ziffle, James Grenert, Boris Bastian, BK DeMasters, David Samuel, Sabine Mueller, Anu Banerjee, Jennifer Clarke, Tabitha Cooney, Joseph Torkildson, Nalin Gupta, Philip Theodosopoulos, Edward Chang, Mitchel Berger, Andrew Bollen, Arie Perry, Tarik Tihan, David Solomon
3:30 pm – 3:45 pm	A Novel <i>PSEN1</i> Mutation and Small Case Series Highlighting the Breadth of Neuropathology Encountered in Familial Dementia Melissa Blessing, Daniel Drubach, Teresa Kruisselbrink, Ralitza Gavrilova, Melissa Murray, R. Ross Reichard	Multinodular and vacuolating neuronal tumor of the cerebrum is defined by genetic alterations activating the MAP kinase signaling pathway Melike Pekmezci, Meredith Stevers, Joanna Phillips, Jessica Van Ziffle, Boris Bastian, Nadejda Tsankova, Bette Kleinschmidt-DeMasters, Mark Rosenblum, Tarik Tihan, Arie Perry, David Solomon
3:45 pm – 4:00 pm	24 Amyloid Precursor Protein Is Required To Permit And To Limit Cortical Plasticity In Vivo Christopher William, Matthew Stern, Xuewei Pei, Lubna Saqran, Matthew Frosch, Bradley Hyman	Deep sequencing of WNT-activated medulloblastomas reveals secondary SHH pathway activation Bryan lorgulescu, Jessica Van Ziffle, Meredith Stevers, James Grenert, Boris Bastian, Lukas Chavez, Damian Stichel, Ivo Buchhalter, David Samuel, Theodore Nicolaides, Anu Banerjee, Sabine Mueller, Nalin Gupta, Tarik Tihan, Andrew Bollen, Pau Northcott, Marcel Kool, Stefan Pfister, Andrey Korshunov, Arie Perry, David Solomon

FRIDAY POSTERS #33-#51

		Friday, June 8, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am -	33	Prevalence of Constitutional Mismatch Repair Deficiency in Saudi Arabian HGG cohort and
5:00 pm		successful use of Nivolumab in an affected child
		Malak Abedalthagafi, Musa AlHarbi, Nahla Mobark
	34	Malignant Glioneuronal Neoplasm with Divergent Differentiation and Histone H3 K27M Mutation:
		Case Report
		Jiancong Liang, Devang Pastakia, John Wellons, Christine Fuller
	35	Sporadic Pilocytic Astrocytoma with Novel Neurotrophic Receptor Tyrosine Kinase 2 (NTRK2)
		Alteration
		Rebecca Yoda, Siobhan Pattwell, Sonali Aurora, Eric Konnick, P.J. Cimino
	36	ATRX, IDH, p53 and H3F3A-G34 Integrated Analysis in Hemispheric Glioblastomas in Young
		Patients
		Elena Martinez-Saez, Jessica Camacho, Teresa Moliné, Rosa Somoza, Regina Mayor, Francisco Ramon
		Martinez-Ricarte, Joan Seoane, Javier Hernandez-Losa, Santiago Ramon y Cajal
	37	Supratentorial Clear Cell Ependymoma Associated with RELA Fusion and Long Term Survival
		Ernest Nelson, MacLean Nasrallah, Angela Viaene, Phyllis Faust, Cristina Antonescu, Marilyn Li, Lea
		Surrey, Minjie Luo, Eun-Sook Cho, Leroy Sharer
	38	Primary diffuse leptomeningeal gliomatosis initially diagnosed as T.B meningitis
		Mariam Youssef, KO Riley, Marcus Bredel, Mina Lobbous, DJ Israel, JR Hackney
	39	Expression of PRAME is increased in K27M mutant gliomas: Identification of a potential target for
		immunotherapy
		Marissa Spino, James Stafford, Luis Chiriboga, Briana Zeck, Vladislav Sviderskiy, Andrew Chi, Richard
		Possemato, Matija Snuderl
	40	Cerebellar glioblastoma: a clinicopathologic series of 16 cases
		Andrew Gao, Maryam Abdollahi, Hidehiro Okura, Cynthia Hawkins, Michael Cusimano, David Munoz
	41	Thyroid Transcription Factor-1 (TTF-1) Expression in Glioblastoma with Primitive Neuronal
		Component (GB-PN)
		Mustafa Goksel, Manoj Kumar, Murat Gokden
	42	Rosette-Forming Glioneuronal Tumor (RFGT) of the Temporal Lobe
		Murat Gokden, Amy Joiner, Brandon Evans, Kremer Nicholas
	43	High-grade Diffuse Astrocytoma with Epithelioid Features, BRAF V600E and IDH-1 (R132H)
		Mutations
		Urooba Nadeem, Rohan Samant, Hazem Ahmed, Murat Gokden
	44	Immunohistochemical Assessment of Tumor-Associated Microglia/Macrophages and T Cells in CNS
		Neoplasms
		M. Adelita Vizcaino, Charles G. Eberhart
	45	Differential microRNA expression in NF1-associated high- and low-grade glioma
		James Nix, Eddie Imada, Heather Ames, Luigi Marchionni, Laura Smithson, David Gutmann, Fausto
		Rodriguez
	46	Novel insight into mechanisms of tumorigenesis of glioblastoma on the background of Ollier
		disease: A case report
		Maliha Khara, Julian Spears, Lyne Noel de Tilly, David Munoz
	47	The AURKA Inhibitor Alisertib Is Synergistic With Irinotecan And Carboplatin Against Glioblastoma
		Cells
		Cory Zumbar, Paul King, Muge Sak, Aisulu Usubalieva, Xiaohui Li, Caroline Mifsud, Joseph McElroy,
		Eric Burton, Norman Lehman
	48	Histone H3F3A G34R-mutant Infiltrating Glioma with Rapid Development of Diffuse Extracranial
		Bony Metastases
		Ashley Holloman, Kevin Fisher, Hao Wu, Vidya Mehta, Sergio Islas, Suzanne Bull, Xuli Wu, Nilesh
		Desai, Guillermo Aldave Orzaiz, Holly Lindsay, Timothy Lotze, Adekunle Adesina, Carrie Mohila
	49	INSM1 over-expression in CDK4-amplified glioblastoma
		Heather Ames, Fausto Rodriguez, Charles Eberhart, Jody Hooper, John Laterra
	50	IDH-Mutant Or IDH-Wildtype? Unusual Subclonal IDH Mutation In A Glioblastoma
		Maria Martinez-Lage, Deborah Forst, Pamela Jones, Dora Dias-Santagata, Jochen Lennerz
	51	A case of adult intraventricular low-grade glial neoplasm with epithelioid features and TERT and
	32	BRAFV600E mutations
		Amir Banihashemi, Mellissa Umphlett, Clare Bryce, Mary Fowkes
		Sammasherin, memosa Ompriece, siare Styce, mary rownes

FRIDAY POSTERS #52-#70

		Friday, June 8, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am -	52	Infantile High-grade Glioma with Novel Translocation Recurring as Ganglion Cell Tumor
5:00 pm	3 2	Martin Powers, Christina Di Loreto, Lawrence Hansen, Denise Malicki
5.00 p	53	Against the odds: genome-wide analysis of glioblastomas with ultra-long survival
	55	Timothy Richardson, Matija Snuderl, Jonathan Serrano, Dwight Oliver, Charles White, Jack Raisanen, Bruce
	Ε4	Mickey, Kimmo Hatanpaa
	54	An unusual case of recurrent epilepsy associated rosette-forming glioneuronal tumor with anaplastic
		transformation in the absence of therapy
		Aaron Halfpenny, Sean Ferris, Marjorie Grafe, Randy Woltjer, Nathan Selden, Kellie Nazemi, Arie Perry,
		David Solomon, Stephen Moore, Helen Lawce, Lora Lucas, Chris Corless, Matthew Wood
	55	Prognostic Implications of MAPT Expression in Primary Brain Tumors
		Meaghan Morris, Antionette Price, Charles Eberhart
	56	Polymorphous low-grade neuroepithelial tumor of the young: How much is PLNTY?
		Jason Gregory, Steven Roper, Anthony Yachnis, Marie Rivera-Zengotita
	57	A high-grade glioneuronal tumor with SOS1 amplification
		Barbara Vidal, Christine Bryke, Nancy Hsu, Ronnie Alterman, Hemant Varma
	58	Dysembryoplastic Neuroepithelial Tumor-Like Neoplasm (DNET) of Septum Pellucidum: Molecular
		Characterization of Primary & Recurrent Tumors
		Mohammad Khan, Kevin Kawachi, Jean Lopategui, Serguei Bannykh, Warren Tourtellotte, Xuemo Fan
	59	Histologic, Immunohistochemical, and Molecular Features of Pituicytomas and Atypical Pituicytomas
	33	Angela Viaene, Edward Lee, Jason Rosenbaum, MacLean Nasrallah
	60	Next-Generation Sequencing Uncovers Novel Glioma Stem Cell-Associated Mutations
	00	Igor Katsyv, Jessica Tome-Garcia, Ethan Ellis, Maya Strahl, Wissam Hamou, Jane Houldsworth, Raymund
		Yong, Bin Zhang, Robert Sebra, Nadejda Tsankova
	C1	5 7
	61	Next Generation Sequencing in Clinical Practice: A Prospective Study of 316 Diffuse Astrocytomas. Does
		it Replace Other Ancillary Studies?
		Thomas Pearce, Christopher Suciu, Ronald Hamilton
	62	False negative rate of ATRX immunohistochemistry in gliomas
		Wesley Samore, Maria Martinez-Lage
	63	Utilizing Histology to Define a Digital Signature for Cell Infiltration in Glioblastoma
		Melissa Umphlett, Vu Nguyen, Rajarsi Gupta, Dimitris Samaras, Heather Bell, Joel Saltz, Nadejda Tsankova
	64	A Case of Widely Metastatic Glioblastoma with Multiple Genetic Mutations
		Melissa Umphlett, Stephanie Shea, Amy Chan, Adilia Hormigo, Jane Houldsworth, Mary Fowkes, Nadejda
		Tsankova, Raymund Yong
	65	Relationship between Ki67 and clinical outcome in PFA pediatric ependymomas
		Nishant Tiwari, Debra Hawes, Sriram Venneti, Alexander Judkins
	66	High-grade Infiltrating Gliomas of the Spinal Cord in Adults: Clinico-pathological and Genomic Evaluation
		of 16 cases
		Mohammed Ali Alvi, Michael A. Paolini, Cristiane Ida, Panagiotis Kerezoudis, Sandy Goncalves, Mohamad
		Bydon, Aditya Raghunathan
	67	BRAF activating mutations involving the β3-αC loop in V600E-negative anaplastic pleomorphic
	07	xanthoastrocytoma
		•
		Drew Pratt, Sandra Camelo-Piragua, Kathryn McFadden, Denise Leung, Rajen Mody, Arul Chinnaiyan, Carl
		Koschmann, Sriram Venneti
	68	A Novel Targetable RAF1-QKI Fusion in Adult Spinal Pleomorphic Xanthoastrocytoma without BRAF
		V600E Mutation
		Elena Daoud, Timothy Richardson, Divya Mella, Edward Pan, Anna Schwarzbach, Charles White, III, Kimmo
		Hatanpaa
	69	Pre-treatment Monocyte-to-Lymphocyte Ratio in Pediatric Gliomas with Histone H3 K27M Mutation
		Seema Patel, Shiyang Wang, Jonathan Serrano, Sharon Gardner, Matija Snuderl
	70	In vivo PiB-PET neuroimaging correlates with autopsy β-amyloidosis in both late-onset and autosomal
		dominant Alzheimer disease
		Namita Sinha, Aihong Zhou, Erin Franklin, Chengjie Xiong, John Morris, Randall Bateman, Tammie
		Benzinger, Nigel Cairns

FRIDAY POSTERS #71-#88

		Friday, June 8, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am -	71	White matter small vessel pathology in Alzheimer's disease
5:00 pm		Sara Nasrabady, James Goldman, Adam Brickman
•	72	A 3D High Resolution Model of Brain Connectivity
		Clio Gonzalez Zacarias, Celia Williams, Michael Bienkowski, Hong Wei Dong, Kristi Clark, Carol Ann
		Miller
	73	Patient-derived induced pluripotent stem cells from brain donors for the study of Alzheimer's
		disease and other neurodegenerative disorders
		Derek Oakley, Naomi Klickstein, Bradley Hyman, Matthew Frosch
	74	Associations of APOE in the Honolulu Asia Aging Study
		Margaret Flanagan, Steven Edland, Laura Hemmy, Brenna Cholerton, Thomas Montine, Lon White
	75	Neuronal identity of the dentate gyrus in AD: ex vivo neuroimaging and histology
		Farshid Sepehrband, Linda Szymanski, Nishant Tiwari, Kristi Clark, Carol Miller
	76	Hereditary, early-onset, and rapidly progressive Alzheimer Disease, associated with the
	70	p.Leu381Phe mutation in the <i>Presenilin</i> 1 gene
		Jose Bonnin, Bernardino Ghetti, Holly Garringer, Francine Epperson, Rose Marie Richardson, Gerald
		Campbell, Benjamin Gelman, Jill Mokry, Brendan Lee, Paolo Moretti
	77	Suppression of Non-Specific Bielschowsky Silver Staining by Pretreatment with Potassium
	//	Permanganate and Oxalic Acid
		Anthony Intorcia, Geidy Serrano, Lucia Sue, Thomas Beach
	70	
	78	Neuropathologic Associations of <i>APOE</i> in the Nun Study
		Margaret Flanagan, Steven Edland, Laura Hemmy, Joyce Meints, Brenna Cholerton, Kelvin Lim,
		Thomas Montine, Lon White
	79	Early-Onset Alzheimer Disease in a Puerto Rican Man with the G206A Mutation in Presenilin 1
		(PSEN1)
		Jose Bonnin, Holly Garringer, Francine Epperson, Rose Marie Richardson, Bernardino Ghetti
	80	Increasing the Value of Autopsies in Patients with Brain Tumors in the Molecular Era
		Jared Ahrendsen, Mariella Filbin, Susan Chi, Peter Manley, Karen Wright, Pratiti Bandopadhayay,
		Mark Kieran, Keith Ligon, Sanda Alexandrescu
	81	Novel BRAF Alteration in Desmoplastic Infantile Ganglioglioma
		Melissa Blessing, Patrick Blackburn, Jessica Balcom, Chandra Krishnan, Michael Zimmermann, Rory
		Jackson, Asha Nair, Christopher Zysk, Kevin Halling, Benjamin Kipp, Cristiane Ida
	82	Two Cases of Pineal Anlage Tumor
		Kathryn Scherpelz, Nicholas Vitanza, Sarah Leary, Ralph Ermoian, Bonnie Cole
	83	Dentate Gyrus Dysplasia: Prevalence in Unselected Autopsy Populations
		Douglas Miller, Kristen Scheitler, Deiter Duff, Jeffery Holloway, C Stacy
	84	Inhibitory Interneurons in Hemimegalencephaly: A Survey of 9 Cases
		Seth Lummus, Jimena Andersen, Sergiu Pasca, Bette Kleinschmidt-DeMasters, Hannes Vogel
	85	Temporal and anatomical expression of Rab5 subfamily of GTPases in the central nervous system
		Kwok-Ling Kam, Paige Parrack, Camille Milton, Marcellus Banworth, Louisa Williams, Sheeja
		Aravindan, Guangpu Li, Kar-ming Fung
	86	Neuropathology of CHARGE Syndrome: Report of Two Cases
		Christopher Borck, Kristen Landi, Nori Williams, Rebecca Folkerth
	87	Eosinophilic Cytoplasmic Astrocytic Inclusions in Pediatric Patients with Epilepsy: A Report of 2
		Cases
		Elmira Vaziri Fard, Manish Shah, Gretchen von Allmen, Rajan Patel, Leomar Ballester, Meenakshi
		Bhattacharjee
	88	Mice Expressing <i>c-MYC</i> in Neural Precursors Develop Choroid Plexus and Ciliary Body Tumors
	00	Morgan Shannon, Ryann Fame, Kevin Chau, Neil Dani, Monica Calicchio, Hart Lidov, Sanda
		Alexandrescu, Maria Lehtinen
		Alexandresed, Iviana Lentinen

FRIDAY POSTERS #89-#107

		Friday, June 8, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am -	89	Neuropathological characterization of a long term survivor with RCDP type I
5:00 pm		Josh Klonoski, Valarie McMurtry, Joshua Sonnen, Jessica Comstock, Theodore Wilson
	90	Acquired neuropathological lesions in infants dying from congenital heart disease from 2000-2017
		Leigh Rettenmaier, Patricia Kirby, Marco Hefti
	91	Immune Myopathy with Large-Histiocyte-Related Myofiber Necrosis
		Namita Sinha, Ziad Alhumayd, Cindy Ly, Robert Bucelli, Robert Schmidt, Alan Pestronk
	92	Detection of Mitochondrial Adaptations to Exercise in a Muscle Biopsy
		David Priemer, Alexander Vortmeyer
	93	A Case of Sarcoid Associated Necrotizing Myopathy With Immune Mediated Features
		Yasir Al-Khalili, Osama Elkadi
	94	Brachial Plexopathy Caused by Endoneurial IgM Deposition in a Patient with Waldenstrom's
		Macroglobulinemia
		Sean Ferris, Giselle López, Marta Margeta
	95	GRANULOMATOUS MYOSITIS AS SOLE MANIFESTATION OF GRAFT VERSUS HOST DISEASE
		Ewa Borys, Stefan Pambuccian
	96	Mitochondrial DNA Depletion Syndrome 12A: A Cause of Mitochondrial Disease with Severe
		Neurological Symptoms
		Heidi Reinhard, Debra Byler, Roger Ladda, Charles Specht
	97	Novel RYR1 Mutation in Congenital Muscular Dystrophy
		Christina Di Loreto, Martin Powers, Lawrence Hansen, Denise Malicki
	98	Plectinopathy Resulting In Epidermolysis Bullosa Simplex With Muscular Dystrophy And Pyloric
	30	Stenosis
		Lindsey Lowder, Jose Velazquez Vega, Pia Mendoza, Stewart Neill, Matthew Schniederjan
	99	Single-cell transcriptome analysis of medulloblastoma
	33	John DeWitt, Volker Hovestadt, Mariella Filbin, Laure Bihannic, Mckenzie Shaw, Andrew Groves, Kyle
		Smith, Jennifer Hadley, Amar Gajjar, Giles Robinson, Lisa Mayr, Irene Slavc, Liliana Goumnerova, Keith
		Ligon, Mario Suva, Paul Northcott, Bradley Bernstein
	100	A Case of Reversible Infantile Respiratory Chain Deficiency Presenting with hypotonia,
		hyperammonemia and failure to thrive
		Jessenia Guerrero, Helio Pedro, Sarah Parisotto, Ada Baisre
	101	Adult orbital xanthogranulomatous disease: A rare histiocytic disease of the orbit with variable
	101	systemic manifestations
		Rajnish Bharadwaj, Kathryn Scherpelz, C. Keene, Joanne Ho, Arash Amadi, Gordana Juric-Sekhar, Luis
		Gonzalez-Cuyar
	102	Endogenous Aspergillus Endophthalmitis: A Case Study of Inpatient Phthisis Bulbi With Enucleation
	102	Due To Fungal Infection
		Edward Kelly Mrachek, Steven Newman, James Mandell
	103	Unusual Overexpression of CD34 in Mature Orbital Adipose Tissue May Pose Challenge in the
	103	Diagnosis of Orbital Spindle Cell/Pleomorphic Lipomas
		Rati Chkheidze, Kyle Molberg, Elena Lucas, Sunati Sahoo, Charles White, Robert Hogan, Bret Evers
	104	Bilateral <i>Acanthamoeba</i> Panophthalmitis: a Rare and Unique Case
	104	Rati Chkheidze, Dominick Cavuoti, Robert Hogan, Bret Evers
	105	PRES: Review of Histological Features
	103	Justin Honce, Nicholas Willard
	106	Segmental Arterial Mediolysis as a Cause of Intracranial Dissecting Aneurysm: Pathological Findings
	100	in Two Surgical Cases
		Hajime Miyata, Junko Kawamura, Junta Moroi, Tatsuya Ishikawa, Toshibumi Kinoshita, Yasuji Yoshida
	107	An Experimental White Matter Infarct Model: Persistent Motor Paralysis by a Laser
	107	Photothrombotic Pyramidotomy
		Min-Cheol Lee, Kyung-Hwa Lee, Hanlim Song, Hyoung-Ihl Kim
		iviiii-Cheoi Lee, Kyung-Awa Lee, Aaniim Song, Ayoung-ini Kim

SATURDAY PLATFORMS 5 & 6

	Platform Session 5 CTE/Trauma Regency Ballroom	Platform Session 6 Muscle/Other Regency South Ballroom
	Moderators: R. Ross Reichard, MD; Edward Lee, MD, PhD	Moderators: Sandra Camelo-Piragua, MD; Rupal Mehta, MD
8:00 am – 8:15 am	108 Distinguishing features that improve specificity and sensitivity in the diagnosis of chronic traumatic encephalopathy (CTE) Travis Danielsen, R. Reichard, Ping Shang, Charles White	116 Neurogenic features in mitochondrial myopathies Jian-Qiang Lu, Adnan Mubaraki, Chuanzhu Yan, John Provias, Mark Tarnopolsky
8:15 am – 8:30 am	109 TMEM106b Risk Variant is Associated with Pathological Outcome in CTE Jonathan Cherry, Jesse Mez, Victor Alvarez, Bertrand Huber, Ann McKee, Thor Stein	117 Dystrophinopathy female carriers diagnosed by muscle biopsy: a 20-year case review Steven Moore
8:30 am – 8:45 am	110 Chronic traumatic encephalopathy is not a predominant pathology in concussed brains Lili-Naz Hazrati	118 Use of Abnormal Protein Expression to Identify Subclasses of Nemaline Myopathy Jennifer Tinklenberg, Emily Siebers, Rebecca Slick, Hui Meng, Samuel Ayres, Mark VandenAvond, Kristen Nowak, Henk Granzier, Edna Hardeman, Federica Montanaro, Michael Lawlor
8:45 am – 9:00 am	111 Alteration of astrocytic networks in chronic traumatic encephalopathy Bertrand Huber, Katherine Babcock, Audrey Hildebrandt, Jonathan Cherry, Victor Alvarez, Thor Stein, Ann McKee	Histopathologic correlates of familial hemophagocytic lymphohistiocytosis isolated to the central nervous system Isaac Solomon, Hojun Li, Leslie Benson, Lauren Henderson, Barbara Degar, Mark Gorman, Christine Duncan, Hart Lidov, Sandra Alexandrescu
9:00 am – 9:15 am	Disease specific nature of microglial neuroinflammation in TBI and CTE Missia Kohler, Stephanie Powers, Ponni Arunkumar, Travis Danielsen, Charles White, Marsel Mesulam, Eileen Bigio, Qinwen Mao	120 Developmental Mechanisms of Apneic Presentation in Congenital Central Hypoventilation Syndrome Jilian Liu, Diego Alzate, Hasnaa Mostafa, Behiye Kaya, Michele Alves, Catherine Czeisler, Jose Otero
9:15 am – 9:30 am	113 Screening for Chronic Traumatic Encephalopathy (CTE) in a Forensic Setting Christopher Borck, Jacqueline Nunez, Hannah Jarvis, Rebecca Folkerth	121 Visualization of deep learning data structures and AI-based classification in histopathology using dimensionality reduction Kevin Faust, Quin Xie, Ugljesa Djuric, Phedias Diamandis
9:30 am – 9:45 am	114 Frontotemporal lobar degeneration Type E and chronic traumatic encephalopathy: connecting the dots? Kevin Bieniek, Melissa Blessing, David Jones, David Knopman, Dennis Dickson, Joseph Parisi	122 Development of a deep learning decision support tool for intraoperative histopathologic image analysis Andrew Gao, Kevin Faust, Quin Xie, Ugljesa Djuric, Phedias Diamandis
9:45 am – 10:00 am	115 Thoracic and lumbar nerve root hemorrhage in pediatric non-accidental head trauma Missia Kohler, Stephanie Powers, Ponni Arunkumar, Christina Appin, Eileen Bigio, Qinwen Mao	123 Print-culture as bridge between anatomic pathology and microbiology: A case series and comparison to standard culture techniques Phillip McMullen, II, Vera Tesic, Peter Pytel

SATURDAY PLATFORMS 7 & 8

	Platform Session 7 Neurodegenerative: FTLD/Lewy body/Prion Regency Ballroom Moderators: John Crary, MD, PhD; Richard Perrin, MD, PhD	Platform Session 8 Tumors: Other Regency South Ballroom Moderators: Craig Horbinski, MD, PhD; Matija Snuderl, MD
2:00 pm – 2:15 pm	Molecular phenotyping of human neurons with TDP-43 pathology reveals decondensation of transposable elements Elaine Liu, Jenny Russ, Alexandre Amlie-Wolf, Edward Lee	Metabolomic Analysis of CSF Differentiates Primary and Metastatic CNS Tumors from Non-Neoplastic Disease Leomar Ballester, Lu Gurangong, Soheil Zorofchian, Venkata Vantaku, Vasanta Putluri, Yuanqing Yan, Octavio Arevalo-Espejo, Roy Riascos-Castaneda, Arun Sreekumar, Yoshua Esquenazi, Nagireddy Putluri, Jay-Jiguang Zhu
2:15 pm – 2:30 pm	Antisense RNA foci and poly-GR dipeptide repeat proteins are unique in repeat-expanded C9orf72 ALS/FTD Shahram Saberi, Jennifer Stauffer, Jie Jiang, Sandra Diaz Garcia, Amy Taylor, Derek Schulte, Takuya Ohkubo, Cheyenne Schloffman, Marcus Maldonado, Michael Baughn, Maria Rodriguez, Don Pizzo, Don Cleveland, John Ravits	Whole genome DNA methylation signatures for diagnosis of brain metastases Olga Krasnozhen-Ratush, Jonathan Serrano, Matija Snuderl
2:30 pm – 2:45 pm	126 GRN Mutations Cause Unique CA1 Pathology Qinwen Mao, Missia Kohler, Tamar Gefen, Jayson Wilson, Zachary Parton, Haibin Xia, Rosa Rademakers, Emily Rogalski, Sandra Weintraub, Marek-Marsel Mesulam, Eileen Bigio	CSF cytology in the evaluation of primary and metastatic CNS malignancies Tatiana Belousova, Vanya Jaitly, Octavio Arevalo-Espejo, Roy Riascos-Castaneda, Jing Liu, Yoshua Esquenazi, Meenakshi Bhattacharjee, Leomar Ballester
2:45 pm – 3:00 pm	127 Neuropathology of Progressive Supranuclear Palsy – Experience from 20-years of Brain Banking for CurePSP Dennis Dickson	Integrated Liquid Biopsy Analysis for Pediatric Brain Tumor Patients Using Detection of ctDNA and Circulating Tumor Cells Kaicen Zhu, Katherine Barnett, Guomiao Shen, Hussein Mohammed, Tania Panicucci-Roma, Jonathan Serrano, David Harter, Jeffrey Wisoff, Amanda Yaun, Shiyang Wang, Sharon Gardner, Matija Snuderl
3:00 pm – 3:15 pm	128 Evidence of Novel Pathologic Protein Misfolding in the Human Amygdala a Proteomics Study Peter Nelson, Haining Zhu, Jozsef Gal	Telomere alterations in NF1-associated solid tumors Fausto Rodriguez, Mindy Graham, Jacqueline Brosnan-Cashman, Christine Davis, M. Adelita Vizcaino, Doreen Palsgrove, Caterina Giannini, Murat Gokden, Jaishri Blakeley, Allan Belzberg, Christopher Heaphy
3:15 pm – 3:30 pm	Gain-of-function of the ATXN1-CIC co-repressor complex drives the cerebellar pathology in spinocerebellar ataxia type 1 (SCA1) Hsiang-Chih Lu, Maxime Rousseaux, Tyler Tschumperlin, Elizabeth Lackey, Vitaliy Bondar, Ying-Wooi Wan, Qiumin Tan, Carolyn Adamski, Jillian Friedrich, Kirk Twaroski, Weili Chen, Jakub Tolar, Christine Henzler, Ajay Sharma, Aleksandar Bajić, Tao Lin, Lisa Duvick, Roy Sillitoe, Huda Zoghbi, Harry Orr	Whole Exome Sequencing of Clinically Aggressive Meningiomas Reveals Mutational Signatures Associated with DNA Mismatch Repair and Aging Benjamin Liechty, Sylvia Eisele, Stephen Kelly, Varshini Vasudevaraja, Ramona Bledea, Peter Wu, Jonathan Serrano, Leah Katz, Joshua Silverman, Donato Pacione, Stephen Russell, Chandra Sen, John Golfinos, Andrew Chi, Matija Snuderl
3:30 pm – 3:45 pm	130 Dominantly Inherited prion protein cerebral amyloidosis: a reappraisal Bernardino Ghetti, Pedro Piccardo, Gianluigi Zanusso	138 Multiplexed Immunofluorescence Reveals Potential PD-1/PD-L1 Pathway Vulnerabilities in Craniopharyngioma Shannon Coy, Rumana Rashid, Jia-Ren Lin, Ziming Du, Andrew Donson, Todd Hankinson, Nicholas Foreman, Peter Manley, Mark Kieran, David Reardon, Peter Sorger, Sandro Santagata
3:45 pm – 4:00 pm	131 Automated digital solutions for quantitative neuropathology Kangway Chuang, Ziqi Tang, Michael Keiser, Charles DeCarli, Laurel Beckett, Lee-Way Jin, Brittany Dugger	139 LEF1 Immunohistochemistry in the Evaluation of Medulloblastoma Subgroups Diana Thomas, Geoffrey Murdoch, Ronald Hamilton, Brent Orr

SATURDAY POSTERS #140-#160

		Saturday, June 9, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am –	140	Pediatric Primary CNS NTRK-Associated Fibroblastic Neoplasm: A Case Report
5:00 pm		Matthew Torre, Nicholas Jessup, Jason Hornick, Sanda Alexandrescu
•	141	Dural tail in meningioma – Is it a radiological guide for curative resection?
		Dewa Pakshage Chula Kanishka Ananda Lal
	142	CNS involvement in Peripheral T-cell lymphoma, a rare presentation.
	<u>_</u>	Mariam Youssef, Deniz Peker, JR Hackney
	143	Evaluating Circulating Tumor DNA from the Cerebrospinal Fluid of Patients with Melanoma and
	113	Leptomeningeal Disease
		Leomar Ballester, Isabella Glitza Oliva, Dzifa Douse, Melissa Chen, Chieh Lan, Lauren Haydu, Jason
		Huse, Sinchita Roy-Chowdhuri, Rajalakshmi Luthra, Ignacio Wistuba, Michael Davies
	144	Malignant progression in extraventricular neurocytoma arising from the VIIIth cranial nerve: A
	144	case report, literature review, and debate
		•
	4.45	Yasuo Sugita, Takuya Furuta, Satoru Komaki, Koichi Ohshima, Kiyohiko Sakata, Motohiro Morioka
	145	Corpus Callosum Chordoma
		David Priemer, Lorenzo Rinaldo, Terence Burns, Aaron Cohen-Gadol, Daniel Brat, Caterina Giannini,
		Alexander Vortmeyer
	146	ATRX Mutation in Pineal Parenchymal Tumor of Intermediate Differentiation
		Michelle Nagurney, Christopher Farrell, Zi-Xuan Wang, Mark Curtis
	147	Spindle Cell Oncocytoma with Papillary Features
		Kymberly Gyure, Rahul Singh, Joseph Voelker, M. Beatriz Lopes
	148	Choroid Plexus Papilloma (CPP) with Neuropil-like (NPL) Differentiation
		John Block, Christine Fuller, Rongsheng Cai, Rahgu Ramakrishnaiah, Murat Gokden
	149	Pituitary Adenoma with Ganglion Cell Differentiation (PA-G)
		Michael Occidental, Jennifer McCarty, John Day, Murat Gokden
	150	Calcifying pseudoneoplasm of the neuraxis: a histologic characterization of 38 cases
		Michael Paolini, Matthew Ball, Mai Lan Ho, Hannah Monahan, Aditya Raghunathan
	151	Primary Intracranial Nerve Sheath Myxoma within the Cavernous Sinus
		Nichole Allen, Gary Pearl, Aaron Wagner, Naren Ramakrishna
	152	Clinicopathologic and Genetic Features of a Meningioma with Unusual Morphology
		Jared Woods, Matthew Torre, Alexandra Golby, Eudocia Lee, Shyam Tanguturi, William Denison,
		Zachary Spigelman, Umberto De Girolami
	153	Intravascular Large B-cell Lymphoma (IVL) with Endocrine Organ Involvement
	155	Anna Tart, Raga Palagiri, Jeanette Ramos, Vikas Koppurapu, Jennifer Forsyth, Anusha Jillella,
		Harmeen Goraya, Murat Goraya
	154	, , ,
	134	Clinical and Immunohistochemical Analysis of Clinically Non-functional Pituitary Neuroendocrine Tumors
		Jonathan Lavezo, Merav Frankel, Brunilda Balliu, James Pan, Andrew Hoffman, Robert Dodd,
	455	Griffith Harsh, Laurence Katznelson, Hannes Vogel
	155	A Case of STAT6 Positive Intraventricular Hemangiopericytoma In An Adult
	4	Colin Kanach, Osama Elkadi
	156	Molecular Alterations in Primary and Metastatic Central Nervous System Lymphomas
		Soheil Zorofchian, Hanadi El-Achi, Yoshua Esquenazi, Leomar Ballester
	157	CNS High-Grade Neuroepithelial Tumor with BCOR Alteration: Heterogeneous Histologic Features
		Expand the Morphologic Spectrum
		José Velázquez Vega, Matthew Schniederjan, Arie Perry, David Solomon
	158	Epstein-Barr Virus-Associated Smooth Muscle Tumor: A Case with Unusual Histology
		José Velázquez Vega, Lindsey Lowder, Matthew Schniederjan
	159	Papillary Tumor of The Pineal Region: A Case Report Including Detailed IHC and Molecular
		Evaluation
		Astin Powers, Martha Quezado, Cara Monroe, Edjah Nduom, Abhik Ray-Chaudhury, Mark Gilbert,
		Mark Raffeld, Terri Armstrong, Sonja Crandon, Elizabeth Vera
	160	Synaptophysin-positive Solitary Fibrous Tumor/Hemangiopericytoma Occurring in the
	100	Parasellar/Cavernous Region
		Shalini Mukhi, Emilie Rouah
		Shainii iviukiii, Ellillie kuudii

SATURDAY POSTERS #161-#181

		Saturday, June 9, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am –	161	Adenocarcinoma arising from a recurrent intracranial teratoma
5:00 pm		Daniel Shepherd, Stefan Pambuccian, Ewa Borys
	162	Clinicopathological and molecular analysis of multinodular and vacuolating neuronal tumors
		Seong-Ik Kim, Euno Choi, Jae Kyung Won, Chun Kee Chung, Sung-Hye Park
	163	A case report of necrotizing myopathy, likely paraneoplastic in origin, arising in the setting of high
		grade serous carcinoma of the ovary.
		Nathan Clement, Anat Stemmer-Rachamimov
	164	Lymphomatoid granulomatosis, grade 3
		Josh Klonoski, Valarie McMurtry, Frederic Clayton, Cheryl Palmer
	165	SOX-10 Expression Aids in the Differential Diagnosis of Endolymphatic Sac Tumors
		Anne Shepler, Julia Kofler
	166	Where Is My Hemangioblastoma – Case Report: Gliosis Radiographically Mimicking A Paraspinous
		Neoplasm
		Kanish Mirchia, Joseph Fullmer
	167	Loss of H3K27me3 Expression Distinguishes MPNST from ANNUBP but Lacks Prognostic
	107	Significance
		Erik Williams, Ann Belan Larque, Tareq Juratli, Vikram Deshpande, Ivan Chebib, G. Petur Nielsen,
		Anat Stemmer-Rachamimov
	168	Medulloepithelioma: a case report with mosaic loss of chromosomes 19 and 2
	100	·
	1.00	Suash Sharma, Ravindra Kolhe, Ian Heger, Amyn Rojiani
	169	Polymorphous low-grade neuroepithelial tumor of the young: a case report with genomic finding
	170	Suash Sharma, Ravindra Kolhe, Cole Giller
	170	Ventricle-Predominant Primary CNS Lymphomas: A Series of 5 Cases
		Matthew Ball, Adam Wood, Jonathan Morris, Aditya Raghunathan
	171	Metastatic urothelial carcinoma to the brain and infiltrating into the overlying dura and invading
		the skull
		Kristyn Galbraith, Joseph Fullmer
	172	Detection of the MYD88 p.L265P mutation in the CSF of a Patient with Central Nervous System
		Lymphoma
		Soheil Zorofchian, Lu Guangrong, Jay-Jiguang Zhu, Dzifa Y. Duose, Chieh Lan, Yoshua Esquenazi,
		Leomar Y. Ballester
	173	Plurihormonal pituitary neuroendocrine tumor with Pit1 and SF-1 coexpression: A novel entity
		Lavezo Jonathan, Erna Forgo, Larry Katznelson, Griffith Harsh, Hannes Vogel
	174	An Unusual Location For An Uncommon Adult CNS Tumor
		Nishant Tiwari, Kyle Hurth, Anna Mathew
	175	Histiocytic Neoplasm Harboring a MAP2K1 Gene Mutation Presenting as a Dural Mass. A Case
		Presentation
		Diana Castro, Eli Diamond, Christine Moung, Marc Rosenblum, James Liu, Ada Baisre
	176	An aggressive primary melanocytic neoplasm of the spinal cord with molecular characterization.
	2.0	Rajnish Bharadwaj, Daniel Gallego, Ashley Eckel, Lincoln Pac, Jing Zhang, Eric Konnick, Desiree
		Marshall
	177	Molecular and histological findings in Multinodular and Vacuolating Glioneuronal Tumor (MVNT)
	1//	suggest the entity is a clonal neoplasm
		Aivi Nguyen, Jason Rosenbaum, Suyash Mohan, MacLean Nasrallah
	178	
	1/6	Application of Raman Techniques for the Diagnosis of Skull Base Tumors
	470	Gordana Juric-Sekhar, Kseniya Shin, Andrew Francis, Jason Barber, Laligam Sekhar, Dan Fu
	179	Spinal Myoepithelial Carcinoma with T12 Spine Fracture and Spinal Cord Compression: A Case
		Report
		Zhiyan Fu, Jiang Qian
	180	Tau Inclusions in Pick Disease Associated with DeltaK280 MAPT Mutation
		Kathy Newell, Manuel Schweighauser, Masami Masuda-Suzukake, Therése Klingstedt, Holly
		Garringer, Peter Nilsson, Bernardino Ghetti, Michel Goedert
	181	CNS Synucleinopathy in two siblings with Action Myoclonus Renal Failure Syndrome (AMRF)
		Graham Lobel, Camilo Toro, Nahid Tayebi, Edwards Nancy, Virginia Kimonis, Abhik Ray Chaudhury

SATURDAY POSTERS #182-#200

		Saturday, June 9, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am –	182	Broadening the Clinical and Pathological Spectrum of FTLD-FUS
5:00 pm		Mari Perez-Rosendahl, Veronica Hirsch-Reinshagen, Freddi Segal-Gidan, Ian Mackenzie, Harry Vinters
	183	Are Lewy bodies associated with sympathetic pathology in dementia subjects?
		Geidy Serrano, David Shprecher, Michael Callan, Brett Cutler, Charles Adler, Holly Shill, John Caviness,
		Marwan Sabbagh, Christine Belden, Erika Driver-Dunckley, Shyamal Mehta, Lucia Sue, Kathryn Davis,
		Edward Zamrini, Thomas Beach
	184	Parkinsonism and Late-Onset Seizures: Adult Polyglucosan Body Disease (APBD) and
		Dysembryoplastic Neuroepithelial Tumor (DNT)
		Raymond Sobel, Shannon Kilgore, Sherry Xue He
	185	Neuropathology of ataxia and myoclonus associated with celiac disease
		Jie Chen, Robert Bucelli, Gabriela de Bruin, Richard Perrin
	186	Glial heterogeneity in Huntington's disease
	100	Osama Al Dalahmah, Istvan Adorjan, James Goldman
	187	PrPres deposition in the retina of sporadic, familial and iatrogenic Creutzfeldt-Jakob diseases (CJD)
	107	Masaki Takao, Hiroaki Kimura, Ban Mihara
	100	
	188	Three Generations of Spinocerebellar Ataxia with Cognitive Decline: Neuropathology and Potential
		Genetics
		Caitlin Latimer, Dong-Hui Chen, Luis Gonzalez-Cuyar, Suman Jayadev, Wendy Raskind, Thomas Bird,
		C. Keene
	189	Neuropathology of Spastic paraplegia-15 (SPG15) mimics that of Parkinson's Disease
		Hisatomo Kowa, Tohko Miyawaki, Maki Mitani, Naonobu Futamura, Itaru Funakawa, Kenji Jinnai
	190	The first Neuropathological Description of a CHCHD10 case affected by ALS
		Julia Keith, Andrew Gao, Emily Swinkin, Janice Robertson, Lorne Zinman, Eric Shoubridge, Ekaterina
		Rogaeva
	191	Suspect Melarsomine Dihydrochloride Related Myelopathy in a German Shepherd Dog
		Tiffany Peterson, Andrea Dedeaux, Nancy Welborn, Kirk Ryan, Fabio Del Piero
	192	Neuropathologic correlates of tau by [18F]flortaucipir PET in Gerstmann-Sträussler-Scheinker
		disease with the PRNP F198S mutation
		Shannon Risacher, Martin Farlow, Francine Epperson, Eileen Tallman, Rose Richardson, Jill Murrell,
		Jose Bonnin, Bernardino Ghetti, Andrew Saykin
	193	Chronic Traumatic Encephalopathy with Clinical and Neuropathologic Features of Motor Neuron
		Disease
		Kathy Newell, Bernardino Ghetti
	194	A case of Marfan syndrome with superficial siderosis.
		Rajnish Bharadwaj, Annie Samraj, Rebecca Yoda, C. Keene
	195	A Case of Adult-Onset Leukoencephalopathy with Axonal Spheroids and Pigmented Glia (ALSP)
	155	with Novel Mutation in CSF1R gene.
		Vaibhav Chumbalkar, Mohammed Shiekhmohammed, Earl Zimmerman, Peter Bauer, Arnulf
		Koeppen, Jiang Qian
	196	The spectrum of tau pathology in Huntington's disease
	190	
	107	Swikrity Upadhyay Baskota, Oscar Lopez, Julia Kofler
	197	Neurofibrillary tau pathology and PrP amyloidosis are associated with the PRNP Q160X nonsense
		mutation
		Bernardino Ghetti, Jose Bonnin, Holly Garringer, Rose Richardson, Francine Epperson, Jamie Fong,
		Julio Rojas, Andrea Legati, Anna Karydas, Giovanni Coppola, Michael Geschwind
	198	Asymmetry in Primary Progressive Aphasia, Probable Alzheimer Disease and Frontotemporal
		Dementia
		Missia Kohler, Qinwen Mao, Christina Appin, Emily Rogalski, Tamar Gefen, Sandra Weintraub, Alfred
		Rademaker, Marsel Mesulam, Eileen Bigio
	199	Reactive astrocytes mimicking diffuse astrocytoma in the setting of Erdheim-Chester Syndrome: A
		case report
		David Ullman, Richard Koenig, David Dorn, Danielle Fasciano, James Hackney
	200	Acute Disseminated Encephalomyelitis in an Adult Patient with Human Immunodeficiency Virus
		Infection and Recent Influenza Vaccination
		Yang Liu, Arslan Ahmad, Bo Lin, Karrah St.laurent-ariot, Jenny Libien, Jianying Zeng

SATURDAY POSTERS #201-#218

		Saturday, June 9, 2018
Time:	Poster #:	Gulfstream-Hialeah & Keeneland
8:00 am -	201	Neuropathological Findings in a Case of Aicardi–Goutières Syndrome with IFIH-1 Mutation
5:00 pm		Ahmed Gilani, Kelley Capocelli, Eric Wartchow, Bette Kleinschmidt-DeMasters
	202	Dual Pathology in Rasmussen Encephalitis
		Ahmed Gilani, Eric Prince, Bette Kleinschmidt-DeMasters
	203	A case report of presumed adult-onset Rasmussen's encephalitis.
		Nathan Clement, Matthew Frosch
	204	Anti-GABA-A receptor antibody-associated encephalitis in an adult and pediatric case: radiologic,
		biopsy, and CSF findings
		Aivi Nguyen, Neena Cherayil, Ana Cristancho, Mariarita Santi, Eric Lancaster, MacLean Nasrallah
	205	Behcet's Disease of the Central Nervous System (CNS)
		Susan de la Monte, Edward Stopa, Galam Khan, Justin Remer, Katarina Dakay
	206	Fatal Granulomatous Amebic Encephalitis In a Heart Transplant Patient: A Correlation Between
		Clinical, Radiographic, and Autopsy Findings.
		William Harrison, Bruce Leckey, Christine Hulette
	207	Non-tubercular mycobacterial spinal cord abscesses in an HIV+ male due to M. Haemophilum
		Bette Kleinschmidt-DeMasters, Kellie Hawkins, Carlos Franco-Paredes
	208	Congenital rubella syndrome (CRS)lest we forget
		Bette Kleinschmidt-DeMasters
	209	Pseudorabies Outbreak in Two Catahoula Cur Hunting Dogs in Louisiana
		Tiffany Peterson, Fabio Del Piero
	210	Brainbow labeling in human ex vivo brain slices for high-throughput morphological analysis
		Shu-Hsien Sheu, Jonathan Ting, Pichet Adstamongkonkul, Ian Boothby, Leonie Hoyo, Ed Lein, Jeff
		Lichtman, David Clapham
	211	Trigeminal Amyloidoma: A Report of Two Cases
		Amy Swanson, Rachael Vaubel, Michael Link, Jamie Van Gompel, John Wald, Ellen McPhail, Caterina
		Giannini
	212	Validation of THK 5351 PET ligand to detect astrogliosis in vivo
		Shigeo Murayama, Renpei Sengoku, Kenji Ishii, Yuko Saito
	213	A novel in-frame BRAF deletion in a case of central nervous system Rosai-Dorfman disease
		Timothy Richardson, Megan Wachsmann, Dwight Oliver, Zahidur Abedin, Dennis Burns, Jack
		Raisanen, Benjamin Greenberg, Kimmo Hatanpaa
	214	Histopathology of the Cat Somatosensory Cortex After Chronic Electrical Stimulation
		Nishant Tiwari, Ryan Long, Carol Miller, Douglas McCreery
	215	Relapsing Multifocal Necrotizing Encephalopathy in a 60-year-old Patient
		Diana Thomas, Kiruba Dharaneeswaran, Clayton Wiley, Julia Kofler
	216	Detecting Astrocytopathy with mAb DE-R-11
		Diana Thomas, Stephanie Bissel, Geoffrey Murdoch
	217	Rare/Unsual Pathologies in Epilepsy Surgical Resections, and Their Implications for Genetic
		Disease Identification in Some Cases.
		Meenakshi Bhattacharjee, Gretchen Von Allmen, Manish Shah, David Sandberg, Elliott Friedman,
		Nitin Tandon
	218	A novel FIJI workflow demonstrates dynamic changes in postnatal respiratory nuclei innervation
		by Nkx2.2- and Olig3-derived neurons.
		Jillian Liu, Julie Stephens, Catherine Czeisler, Jose Otero
		The state of the s



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Endowed Lectureships

Friday, June 8, 2018

- I. Parisi Lecture
- II. DeArmond Lecture

Saturday, June 9, 2018

I. Saul R. Korey Lecture

Sunday, June 10, 2018

I. Matthew T. Moore Lecture

ENDOWED LECTURESHIP

PARISI LECTURE

he *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 **Philip L. De Jager** 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
2018	Philip L. De Jager	The Genomic Architecture of Aging-Related Neuropathologies: Spotlight on Microglia

PARISI LECTURE

The Genomic Architecture of Aging-Related Neuropathologies: Spotlight on Microglia

Time: 10:30 am – 11:30 am Date: Friday, June 8, 2018

Philip L. De Jager, MD PhD, Weil-Granat Professor of Neurology, Columbia University Medical Center

I. Learning Objectives

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

- 1. Explain how genetic variation contributes to the onset of aging-related neuropathologies
- 2. Describe the extent of heterogeneity in human microglial states
- 3. Outline the role of the epigenome in aging-related pathologies

II. Abstract & Relevant References

Using participants in two cohort studies of aging with prospective autopsy samples (the Religious Order Study and the Memory and Aging Project), we have generated a multi-omic atlas of the dorsolateral prefrontal cortex. The component whole genome sequence, RNA sequence, DNA methylation and Histone mark H3K9Ac sequence data on up to 1200 individuals provides a unique perspective on how the human genome contributes to and is influenced by the accumulation of neuropathologies with advancing age. In addition, new single cell data generated from these individuals is uncovering a previously unrecognized heterogeneity among microglia and identifying microglial cell states that are implicated in different pathologic conditions. Integrating these diverse data from cortical tissue and purified cells, we have established a molecular network map of the aging cortex that identifies transcriptional programs and individual proteins that drive Alzheimer's disease (AD) pathology. Further, we have used this network map to infer the relation of certain cells in the causal chain of events leading to AD dementia: for example, activated microglia appear to synergize with amyloid pathology in leading to the accumulation of Tau pathology and to the subsequent decline in cognitive function. Thus, this talk will illustrate how molecular data can be leveraged to provide new insights into the molecular events that lead to aging-related neurodegeneration.

References:

- 1. Olah et al 2018 A transcriptomic atlas of aged human microglia. Nat Comm 9:539
- 2. Mostafavi et al 2018 A molecular network of the aging brain implicates INPPL1 and PLXNB1 in Alzheimer's disease. BioRXiv 205807
- 3. Ellis et al. 2018 A cortical immune network map identifies a subset of human microglia involved in Tau pathology. BioRXiv 234351
- 4. Klein et al. 2018 Epigenome-wide study uncovers tau pathology-driven changes of chromatin organization in the aging human brain. BioRxiv 273789
- 5. White et al. 2017 Identification of genes associated with dissociation of cognitive performance and neuropathological burden: Multistep analysis of genetic, epigenetic, and transcriptional data. PLOS Med 14:e1002287
- 6. De Jager PL et al. A multi-omic atlas of the human frontal cortex for aging and Alzheimer's disease research. BioRxiv 251967

III. Faculty Biography

Dr. De Jager is the Weil-Granat Professor of Neurology in the Taub Institute for Research on Alzheimer's Disease and the Aging Brain, and the Columbia Precision Medicine Initiative. At the Department of Neurology at New York-Presbyterian/Columbia University Medical Center, he is the Director of the Center for Translational and Computational Neuroimmunology and the Director of the Multiple Sclerosis Center.

"The goal of my work as a clinician-scientist is to apply modern methods of human immunology, statistical genetics, epigenomics and systems biology to the understanding of common neurodegenerative diseases," says Dr. De Jager, who also serves as Chief of the Division of Neuroimmunology, which focuses on characterizing and targeting the neuroimmunologic component of neurodegenerative diseases.

"Our new division seeks to provide innovative, compassionate care to patients with immune dysfunction that targets the brain and spinal cord," says Dr. De Jager. "We also lead transformative, rigorous human research studies to first understand and then to target the role of the immune system in neurodegenerative diseases such as ALS, Alzheimer's, Parkinson's, and multiple sclerosis."

After graduating from Yale University with a degree in French literature and Molecular Biophysics and Biochemistry, he received a PhD in neurogenetics from The Rockefeller University and his medical degree from Weill Cornell Medicine, followed by an MMSc in clinical investigation at Harvard Medical School and MIT. He then pursued a residency in neurology and additional training in clinical neuroimmunology at the Massachusetts General Hospital and Brigham and Women's Hospital. Prior to joining Columbia, Dr. De Jager was on the faculty of Harvard Medical School.

ENDOWED LECTURESHIP

DEARMOND LECTURE

he *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 Suzanne M. de la Monte 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	
2018	Suzanne M. de la	Dysregulated Metabolism in the Pathogenesis of Alzheimer's	
	Monte	Disease: Type 3 Diabetes	

DEARMOND LECTURE

Dysregulated Metabolism in the Pathogenesis of Alzheimer's Disease: Type 3 Diabetes

Time: 4:45 pm – 5:45 pm Date: Friday, June 8, 2018

Suzanne M. de la Monte, MD, MPH, Warren Alpert Medical School of Brown University, Providence, RI

I. Learning Objectives

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

- 1. Summarize evidence demonstrating links between impaired energy metabolism in the brain and neurodegeneration, particularly Alzheimer's disease.
- 2. Explain how insulin resistance and other diabetes-type effects may contribute to major brain pathologies in AD
- 3. Consider potential cofactor roles of systemic diseases in the pathogenesis of AD and other neurodegenerative diseases.

II. Abstract & Relevant References

The metabolic basis of AD is fundamentally linked to insulin resistance and deficiency in the brain. In AD, dysregulation of glucose and lipid metabolism, impairments in insulin signaling, activation of pro-inflammatory cytokines, and compromises to mitochondrial function are all reminiscent of abnormalities that occur in systemic insulin resistance diseases including, diabetes mellitus (DM), non-alcoholic fatty liver disease (NAFLD), obesity, and metabolic syndrome (MetS). Over the past 12 years, many of AD's salient neuropathological features, including amyloid-beta (Aβ42) accumulations, tau hyperphosphorylation, impairments in neuronal plasticity, and cholinergic deficits have been correlated with perturbations in brain insulin and insulin-like growth factor signaling. Moreover, experimental data show that brain insulin resistance compromises oligodendrocyte survival and myelin maintenance, providing a mechanism for leukoaraiosis and white matter atrophy in AD. Similarly, microvascular disease, a universal consequence of insulin resistance, is associated with brain hypo-perfusion, which likely exacerbates concurrent effects of neurodegeneration, and contributes to break-down of the bloodbrain barrier due to disruption of glial-vascular networks. The findings that DM, obesity, NAFLD, and MetS increase risk for cognitive impairment and brain insulin resistance suggest that systemic insulin resistance diseases may promote neurodegeneration. Finally, recent evidence suggests that in sporadic AD unrelated to DM, MetS, NAFLD, or obesity, metabolic hormones and inflammatory indices are simultaneously altered in blood and CSF in the early stages of disease.

References:

- 1. de la Monte SM. Insulin Resistance and Neurodegeneration: Progress Towards the Development of New Therapeutics for Alzheimer's Disease. Drugs. 2017;77(1):47-65.
- 2. Steen E, Terry BM, Rivera EJ, Cannon JL, Neely TR, Tavares R, et al. Impaired insulin and insulin-like growth factor expression and signaling mechanisms in Alzheimer's disease--is this type 3 diabetes? J Alzheimers Dis. 2005;7(1):63-80.
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III. Faculty Biography

Suzanne de la Monte, MD MPH is a Professor in Pathology and Laboratory Medicine, Neurology, & Neurosurgery at the Alpert Medical School of Brown University and Rhode Island Hospital. She obtained an A.B. from Cornell University, M.D. from the Weill Medical College of Cornell University, and MPH from the Blumberg School of Public Health at Johns Hopkins. She received residency training in Anatomical and Pediatric Pathology at Hopkins and fellowship training in Neuropathology at the Massachusetts General Hospital. Dr. de la Monte launched her career as a physician-scientist while on the faculty at MGH and Harvard Medical School. In 2000, she joined the faculty at Rhode Island Hospital and Brown where she conducts basic, translational, and clinical research on mechanisms and consequences of brain insulin resistance and metabolic dysfunction, which she refers to as 'brain diabetes'. Her lab also investigates how various lifestyle exposures cause neurodegeneration associated with brain insulin resistance, and evaluates novel approaches for therapeutic targeting of brain metabolic defects. Dr. de la Monte's research is funded by the NIH. She is the recipient of a MERIT award from the NIAAA and has published over 300 peer-reviewed articles. Extra-curricular interests include golf, spin biking, theater, classical music, and freestyle Paleo cooking.

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 **Rebecca D. Folkerth** 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		
2018	Rebecca D. Folkerth	Reading Tea Leaves: Patterns of Injury in the Pediatric Nervous System		

SAUL R. KOREY LECTURE

Reading Tea Leaves: Patterns of Injury in the Pediatric Nervous System

Time: 10:30 am - 11:30 am Date: Saturday, June 9, 2018

Rebecca D. Folkerth, MD, Neuropathologist, New York City Office of the Chief Medical Examiner, Clinical Associate Professor of Forensic Medicine, NYU School of Medicine

I. Learning Objectives

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

- 1. Identify constellations of general autopsy and nervous system findings consistent with inflicted injury in children
- 2. Describe how to use nervous system autopsy prosection techniques to obtain diagnostically important specimens for complete case analysis
- 3. Identify the "gray zones" between traumatic injury and other causes of nervous system abnormalities in children
- 4. Explain pitfalls in interpretation, documentation, and testimony in pediatric medicolegal cases

II. Abstract & Relevant References

Child abuse is, sadly, a fixed feature of the human condition, having been described through the ages and across all cultures. The nervous system is frequently affected, both directly by the mechanical injury itself, and indirectly by supervening hypoxia/ischemia, the latter related to several factors, including resuscitation. Thus, neuropathologists must discern the subtle and overlapping patterns of these direct and indirect insults, as well as the features of underlying natural disease. A solid understanding of the range of non-traumatic neuropathology in the immature brain is therefore essential.

Bringing to bear her longtime interest and experience in study of the developing brain, Dr. Folkerth will review the elements of specimen analysis most helpful in child abuse cases. She will also emphasize the importance of context, collaboration, and intellectual honesty in making observations and interpretations that may eventually surface in the courtroom setting.

References:

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- 7. Snyder VS, Hansen LA. A Conceptual Overview of Axonopathy in Infants and Children with Allegedly Inflicted Head Trauma. Acad Forensic Pathol 2016;6(4):608-21

8. Folkerth RD, Nunez J, Georgievskaya Z, McGuone D. Neuropathologic Examination in Sudden Unexpected Deaths in Infancy and Childhood: Recommendations for Highest Diagnostic Yield and Cost-Effectiveness in Forensic Settings. Acad Forensic Pathol 2017;7(2):182-99

III. Faculty Biography

Rebecca D. Folkerth, MD, is a Neuropathologist with wide diagnostic experience in the brain abnormalities of infants and children. Prior to joining the New York City Office of the Chief Medical Examiner as the agency's Neuropathologist in 2016, Dr. Folkerth was Director of Neuropathology at Brigham and Women's Hospital, affiliated with Harvard Medical School, in Boston. For nearly 3 decades, she also served as a Consultant in Neuropathology at Boston Children's Hospital, participating with her mentor, Dr. Hannah C. Kinney, in a long-running NIH-funded program project analyzing the cellular basis of brain damage in prematurely born infants. She also was a member of an NIH-supported international investigation into maternal alcohol exposure and other key factors in the risk of sudden infant death and stillbirth, results of which are being compiled and published currently.

More recently, Dr. Folkerth has been part of a national consortium, also NIH-funded, studying traumatic brain injury. She serves as part of the working group that has developed the consensus criteria for the neuropathologic diagnosis of chronic traumatic encephalopathy (CTE). Her interests since joining the OCME include the neuropathology of abusive head trauma, as well as in long-term survivors of traumatic brain injury; brain abnormalities in individuals with cerebral palsy; and developmental and acquired lesions in sudden unexpected death in infancy and childhood (SUID/SUDC), and in sudden unexpected death in epilepsy (SUDEP).

ENDOWED LECTURESHIP

MATTHEW T. MOORE LECTURE

n 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 Mario L. Suvà 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 DeathMechanisms 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, 4 th Edition
2018	Mario L. Suvà	Deciphering Single-Cell Regulatory Programs in Adult and Pediatric Gliomas

MATTHEW T. MOORE LECTURE

Deciphering Single-Cell Regulatory Programs in Adult and Pediatric Gliomas

Time: 8:30 am – 9:15 pm Date: Sunday, June 10, 2018

Mario L. Suvà, MD, PhD, Assistant Professor of Pathology, Massachusetts General Hospital; Associate Member, The Broad Institute of MIT and Harvard; Co-Director, Cancer Program, Harvard Stem Cell Institute

I. Learning Objectives

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

- 1. Identify new methods to profile of single-cells.
- 2. Discuss research findings related to single-cell genomic studies in human gliomas.
- 3. Discuss the cancer stem cell model in gliomas.

II. Abstract & Relevant References

Tumor heterogeneity is a major barrier to therapy. Three dominant factors govern tumor heterogeneity: (I) genetic alterations shape cancer cells programs and their evolution; (II) developmental programs, such as programs of adult stem cell and their differentiation into specialized cell types, ascribe cancer cells with specific functional properties; (III) the composition of the tumor micro-environment and its interaction with malignant cells provides further heterogeneity to the tumor ecosystem. In this lecture, we dissect these influences in human diffuse gliomas studying patient samples with single-cell genomic technologies. We discuss comprehensive single-cell data that help redefine malignant cell lineages, cancer stem cell programs, genetic heterogeneity, and dissect the transcriptional programs of microglia, macrophages, and T cell in clinical gliomas. We will also review the toolbox available in the field of single-cell genomics and the strengths and limitations of different protocols. Overall, we will provide technological insights in the field of single-cell genomics and deep characterization of gliomas biology, with important implications for their management.

References:

- 1. Patel et al, Science 2014
- 2. Tirosh et al, Nature 2016
- 3. Venteicher et al, Science 2017
- 4. Filbin et al, Science 2018
- 5. Suvà et al, Cell 2014

III. Faculty Biography

Mario Suvà is a scientist in the Department of Pathology and the Center for Cancer Research at Massachusetts General Hospital (MGH) and at the Broad Institute. Suvà's expertise is in neuro-oncology and single-cell genomics. Suvà's laboratory focuses on diffuse gliomas in adults and children. A particular effort of the laboratory is on dissecting the heterogeneity of patient tumors and relating transcriptional and genetic programs of individual cancer cells. Suvà directed pioneering studies characterizing glioblastoma, oligodendroglioma, astrocytoma and pediatric gliomas with single-cell genomic technologies, shedding light on tumor heterogeneity, tumor classification, glioma cell lineages, cancer stem cell programs, tumor evolution and the composition of the tumor microenvironment.

Dr. Suvà obtained his M.D and Ph.D. in Lausanne, Switzerland, studying cancer stem cells in gliomas and sarcomas. He did his post-doctoral research at MGH with Brad Bernstein and David Louis, applying chromatin analysis and functional approaches to identify master regulators of glioma stem cell programs.



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Award for Meritorious Contributions to Neuropathology

Friday, June 8, 2018

11:30 am – 11:45 am

Honoring Hannah C. Kinney, MD

Presented by: Rebecca D. Folkerth, MD

Saturday, June 9, 2018

11:30 am – 11:45 am

Honoring Brian N. Harding, MA, DPhil, BM, BCh, FRCPath

Presented by: David Ellison, MD, PhD

AWARD FOR MERITORIOUS CONTRIBUTIONS TO NEUROPATHOLOGY

he 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. Hannah C. Kinney** and **Dr. Brian N. Harding**. They join the rich roster of distinguished former award recipients.

Dociniont

Paciniant

Voor

Year	Recipient	Year	Recipient
1959	Armando Ferraro	1999	Lucy B. Rorke
	Arthur Weil	2000	William R. Markesbery
1960	Joseph H. Globus	2001	John J. Kepes
	George B. Hassin		Henry de Forest Webster
1968	Abner Wolf	2002	Dikran S. Horoupian
	Paul I. Yakovlev		Fusahiro Ikuta
	Harry M. Zimmerman		Kurt A. Jellinger
1970	Webb E. Haymaker	2003	Bernardino F. Ghetti
1971	James W. Kernohan	2004	Michael N. Hart
1972	George A. Jervis	2005	E. Tessa Hedley-Whyte
1979	Raymond D. Adams		Suzanne S. Mirra
	David Cowen	2006	Joseph E. Parisi
	Matthew T. Moore		Jeannette J. Townsend
1981	Richard Lindenberg	2007	James M. Powers
1983	Orville T. Bailey		Cedric S. Raine
1984	Margaret Murray	2008	Kinuko Suzuki
1985	Kenneth M. Earle		Margaret G. Norman
	Nathan Malamud	2009	Peter C. Burger
	Leon Roizin		Pierluigi Gambetti
1986	Martin G. Netsky		Nicholas K. Gonatas
1987	No Award Presented	2010	Stephen J. DeArmond
1988	Edward P. Richardson, Jr.		Samuel K. Ludwin
	F. Stephen Vogel	2011	William W. Schlaepfer
1989	Lucien J. Rubinstein		Leroy R. Sharer
	Robert D. Terry	2012	Bernd W. Scheithauer
1991	Lysia K. S. Forno		Donald L. Price
1992	John Moossy	2013	Reid Heffner
	Gabriele M. ZuRhein		Dawna Armstrong
1993	Peter W. Lampert	2014	Floyd Gilles
	Elias E. Manuelidis		Françoise Gray
1994	Murray B. Bornstein	2015	John Q. Trojanowski
	Samuel P. Hicks		Bette K. Kleinschmidt-DeMasters
	Lowell W. Lapham	2016	Dennis W. Dickson
1995	Amico Bignami		Barbara J. Crain
	Asao Hirano	2017	Ronald C. Kim
1997	Henryk M. Wisniewski		Harry V. Vinters
1998	Richard L. Davis	2018	Hannah C. Kinney
	Wolfgang Zeman		Brian N. Harding

AWARD FOR MERITORIOUS CONTRIBUTIONS TO NEUROPATHOLOGY

Biography: Hannah C. Kinney, MD



Dr. Kinney is Emeritus (2018) Professor of Pathology at Harvard Medical School, Boston, MA, and a pediatric neuropathologist at Children's Hospital Boston. Her laboratory performs research in developmental disorders of the human brain in early life, with a focus upon brain disorders leading to sudden death in fetuses (stillbirth), infants, and children. Her research includes studies of the brainstem and hippocampus in the sudden infant death syndrome (SIDS), the hippocampus and temporal lobe in sudden unexplained death in childhood (SUDC), and the cerebral cortex, white matter (periventricular leukomalacia), thalamus, and brainstem in premature infants, among other disorders of the developing human brain.

In 1970 Dr. Kinney graduated from Duke University, Durham, NC, with a BA degree (major in history), and from Case Western Reserve University School of Medicine, Cleveland, OH, in 1974.

She subsequently performed a pediatric residency at the Children's Hospital of Philadelphia, PA, and an anatomic pathology residency at Duke University Medical School, Durham, NC. Dr. Kinney is board-certified in pediatrics, anatomic pathology, and neuropathology. She came to Children's Hospital Boston and Harvard Medical School, Boston, MA, for specialized training in pediatric neuropathology and developmental neuroscience in 1981, and subsequently rose through the academic ranks to Professor of Pathology in 2003.

In her career, Dr. Kinney served as the Director of the Christopher James Murphy SIDS Laboratory and oversaw research into SIDS brainstems in search of underlying neural mechanisms of sudden death, as well as potential biomarkers of brainstem pathology in living infants at risk for SIDS. She is served as the Director of a NICHD-funded program project to study the role of the brainstem in SIDS though integrated and multidisciplinary studies of human brainstem pathology and models. This program project involved ~50 investigators and post-graduate level students at Harvard, Dartmouth, University of Iowa, Rady Children's Hospital, San Diego, CA, and the medical examiner's system of San Diego County. Dr. Kinney served as the Director of the Developmental Biology and Pathology Center of the Safe Passage Study which oversees alcohol-related research in the developing human brain and placenta. This NICHD/NIAAA-funded study addressed the question of the potential links among prenatal exposure to maternal drinking and smoking, SIDS, stillbirth, and fetal alcohol spectrum disorders (FASD) in a prospective analysis of 12,000 pregnancies, with infants followed to the end of the first postnatal year, the period of SIDS risk. In the last five years of her career, Dr. Kinney served as the Director and then Associate Director of the comprehensive Program in Sudden and Unexpected Death in Pediatrics (SUDP) that she co-founded with the pediatrician and palliative care physician, Dr. Richard D. Goldstein at Boston Children's Hospital in 2012. This program provides comprehensive care and pathology case review to families who have lost an infant under 3 years to sudden and unexpected death due to natural causes.

Dr. Kinney's publication record includes 148 original articles, 37 reviews, 18 chapters, and 4 tributes. She has received multiple awards for her contributions to SIDS and developmental brain research, as well as invitations for honorary SIDS lectureships. Her awards include a MERIT Award from the NICHD (2000-2010), and Moore Award (1987) and Weil Award (Honorable Mention, 1986) from the American Association of Neuropathologists, Award for Excellence in Giving, CJ Foundation for SIDS (2014), Award for "SIDS Researcher of the Year", American SIDS Institute, (2014), and Distinguished Researcher, International Society of Perinatal and Infant Deaths for Outstanding Contributions to SIDS Research (2016). The SIDS Symposium at the 50th Annual Congress of the Federation of South African Societies of Pathology (FSASP) in Somerset West, South Africa, in September 2010, was dedicated to her. Dr. Kinney has mentored many medical students, neuropathology residents and doctoral and post-doctoral fellows in developmental neuropathology and neuroscience over her professional career.

Dr. Hannah C. Kinney "Contributions to the Field", written by Rebecca D. Folkerth, MD

Dr. Hannah Chase Kinney, best known as the originator of the Serotonin Hypothesis in the Sudden Infant Death Syndrome (SIDS), was born in Cleveland, Ohio, and went to college at Duke University, majoring in History. She then returned to Cleveland to the Case Western Reserve University School of Medicine, graduating in 1974. Following a full residency in Pediatrics at the Children's Hospital of Philadelphia, she returned to Duke for Anatomic Pathology and Neuropathology training, the latter with Dr. Peter Burger. She came to Children's Hospital in Boston in 1981, for additional clinical training with Dr. Floyd Gilles, and research with Dr. Richard Sidman. Since her appointment to the faculty at Children's and Harvard Medical School in 1984, her trajectory has been one of continued excellence in the field of developmental neuropathology, having been continuously funded by the NIH. She was promoted to Professor of Pathology in 2003, in recognition of her furtherance of the field of research into SIDS and its biologic underpinnings directly in the human brain, as well as for her role in training a generation of neuroscientists and neuropathologists. In 2012, she spearheaded the creation of Robert's Program at Children's Hospital Boston, for comprehensive clinical, pathologic, and genetic investigation of sudden unexpected death in pediatrics.

Additional interests of Professor Kinney include the neuroanatomic substrates of coma and persistent vegetative state; the spectrum of gray and white matter abnormalities in preterm infants; neuropathology of stillbirth; genomics and proteomics of developmental brain disease; and most recently, imaging-neuropathologic correlation in pursuit of in vivo biomarkers for SIDS and hippocampal dysgenesis.

Professor Kinney has been a dedicated member of the Association since 1983, serving on the Awards Committee, including 2 years as its Chair, and on the Professional Affairs Committee, and as an AANP Councilor to the International Society of Neuropathology. She has presented a great deal of her research at the Annual Meeting, and her laboratory has received the Moore Award (5 times, of which 4 were honorable mentions), and the Weil Award (2 honorable mentions). Hannah has been an editorial board member and reviewer for the *Journal of Neuropathology and Experimental Neurology* her entire career. In fact, she has no fewer than 28 original articles and 3 review articles published in the Journal! Nearly every one of these papers involved trainees, generously welcomed to her lab at all levels, from college students on summer break, to medical students, to post-docs in PhD and MD programs. Hannah is a real "pied piper" in the sense of getting individuals to follow her into our field!

When not marveling at the brain, Hannah likes to write poetry, paint, and spend time with her husband, son and daughter-in-law, and grandson on the Chesapeake River.

Biography: Brian N. Harding, MA, DPhil, BM, BCh, FRCPath



Although both sides of my family hale from various eastern European countries, I am like my parents a Londoner. I was borne at St George's Hospital at Hyde Park Corner (now the Lanesborough Hotel!). My father had been a medical student there, then surgical houseman (intern) during the Blitz. I was also a medical student and intern there, and a trainee pathologist.

Before my clinical training I spent over six years in Oxford, first as an undergraduate at Worcester College earning a BA in Animal Physiology in 1969, and then, after winning a research scholarship, 3 years as a postgraduate in the Anatomy department. It was my particular good fortune to be supervised by TPS (Tom) Powell, one of the most illustrious neuroanatomists of his day – in his early career he had supplied the anatomic substrate for Vernon Mountcastle's groundbreaking discovery of the columnar organization of the cortex.

This turned out to be pivotal to my career. Tom was an inspiring presence, with a fierce objectivity, piercing intelligence and scientific and moral integrity, once remarking to me that "there was more

science fiction than science fact in the literature". His quiet yet coruscating wit did not suffer fools gladly, which did not commend him to some of his contemporaries. Perhaps this was partly the reason he was never given a Professorship in Oxford, despite being FRS. But the output from his group was prodigious. In that period, electron-microscopy was in vogue and I was tasked with examining the ultrastructure and connexions of primate thalamic nuclei, comparing the intralaminar centre median nucleus of Luys with the ventrolateral relay nucleus. Furnished with long ribbons of ultrathin serial sections (up to 200 thanks to the virtuosity of our techs), I was able to reconstruct complex formations of axo- and dendro-dendritic and reciprocal synapses and uncover their triadic nature, which formed the basis of my DPhil thesis (1976). There was enough material for 4 papers, but Tom's Welsh parsimony inclined towards a monograph, unpopular with most journals, but it formed a single issue of Phil Trans Roy Soc.

Following my clinical studies, it was Tom Powell who suggested a career in neuropathology, and a return to Oxford to join Dr Oppenheimer's department; but I preferred to remain in London. Despite the initial enthusiasm at St George's for my request to learn neuropathology, there was little practical support. I had become acquainted with William Blackwood, erstwhile editor of Greenfield's Neuropathology, recently retired from the chair at the Institute of Neurology, Queen Square, and he introduced me to his successor. Leo Duchen had migrated from the Institute of Psychiatry to Queen Square with his assistant, Francesco Scaravilli, and they took on 2 trainee neuropathologists, myself and Seth Love. Both Leo and Francesco were great mentors and superb clinician scientists, but I increasingly gravitated towards the pediatric material presided over by a feisty Hungarian/Jewish holocaust survivor, Magda Erdohazi, who worked both at Queen Square and at the adjacent Great Ormond Street Hospital for Children (GOSH). Following her retirement, I was appointed to the joint senior lectureship which I held until 1994, when because of the increasing work load it was recast into a fulltime position at GOSH.

The 1980s was an exciting time for the neuropathologist at GOSH, a tertiary referral center and the leading children's hospital in the UK. Imaging was just beginning to make an impact on diagnosis, metabolic studies were becoming increasingly sophisticated, but the explosion in genetics had not quite begun; there was still a considerable clinical need and opportunity for morphologic studies and diagnosis. With an increasing consultation practice across Europe and further afield I was exposed to a huge range of both systemic and neurologic disease, as evidenced by the extensive catalogue of illustrations in the pediatric sections of "Neuropathology, a reference text" (eds Ellison and Love) almost entirely furnished from my own material. It was a superb teaching archive, which attracted many UK neuropathology trainees to spend short periods in my lab, and it was extensively mined for the lectures and seminars I gave at the Royal College of Pathologists, the British Neuropathological Society, EuroCNS and IPPA, among others. It was also invaluable in writing for Greenfield's Neuropathology, with which I have had a long association, contributing 8 chapters over 5 editions.

During much of my tenure at GOSH I ran the pediatric neuropathology service single-handed, encompassing surgical biopsy, neuromuscular and epilepsy surgery and autopsy and my research interests emerged from clinical experience mostly with metabolic degenerative and developmental disorders and epilepsy, rather than neoplasia. Published reports of metabolic errors ran the gamut from urea cycle through leukodystrophies to glycosylation disorders, but the large series of Leigh syndrome and Alpers' disease were perhaps the most informative, clarifying the clinico-pathologic phenotypes particularly for Alpers' where there had previously been much confusion regarding this hepato-cerebral mitochondrial disorder. My interest in Alpers' was sparked when Dr Erdohazi made a naked eye diagnosis at brain cutting, a feat I was lucky enough to repeat 10 years later in a rare example with adult onset.

A pediatric neuropathologist encounters their fair share of rarities, Fowler's and Fazio-Londe disease, zoster embryopathy, pseudo-Torch and migrational disorders, to name a few of mine. Some others never made it to the journals, but reside in book chapters, for example my collection of dentato-olivary anomalies, although one group became a published case series and later proved familial. And some remained on the shelf when I migrated across the "pond".

In the last part of my London career I was fortunate to recruit a very talented trainee, who later became my colleague: Tom Jacques who now runs the GOSH service and has recently become the editor of Neuropathology and applied Neurobiology. Thus, I did not feel too guilty when at the suggestion of Lucy Rorke and Jeff Golden, I jumped ship and landed up at CHOP where I have been extremely happy for nearly 10 years now, and privileged to have both of them, Alex Judkins and Mariarita Santi as wonderful colleagues, but especially fortunate that Mariarita Santi has been my friend and close collaborator throughout this whole period. And it has allowed me to continue my interest in SMA and rare brainstem malformations, and orphan diseases such as Amish cerebro-renal disease, now underpinned by genetic diagnosis. And it has enabled me to solve an old mystery, a micro-lissencephaly with multinucleated neurons, which had sat on the shelf for nearly 20 years; DNA analysis by my collaborator Stephanie Bielas at Ann Arbor, found a deletion of citron kinase, a central actor in cytokinesis.

Philadelphia with its exuberant artistic and culinary culture has been good to me, not least because I have been able to indulge twin passions for music and collecting ceramics. There are many musicians in my family, and for as long as I can remember music has been an essential part of my life. So with both the world famous Philadelphia orchestra and the superb Philadelphia Chamber Music Society on my doorstep, and frequent visits to the Metropolitan opera, I do not miss the London scene too much. My interest in ceramics dates back nearly 40 years. There is a vibrant crafts community in Philadelphia with which I have become closely involved, while my mainly British collection has considerably enlarged and now includes some American examples. In Philadelphia, fortunately, I have the space to house them.

Dr. Brian N. Harding "Contributions to the Field", written by David W. Ellison, MD, PhD

Dr. Brian Harding was born and grew up in London, England, although both sides of his family came originally from Eastern Europe. He went to Oxford University to study medicine, and as is usual for those trained at Oxford or Cambridge took an intercalated degree in a medical science subject – animal physiology in Brian's case. Unusually for British medics at that time, Brian then spent a further three years studying for a DPhil degree before beginning the clinical element of his medical undergraduate training. His postgraduate studies in the Oxford Anatomy department were mentored by Dr. Tom Powell, one of the most illustrious neuroanatomists of his time. The field's focus at that time was electron microscopy, and Brian was given projects on the ultrastructure and connections of primate thalamic nuclei, comparing the intralaminar nucleus of Luys with the ventrolateral relay nucleus. His thesis was transcribed into a monograph, which was published as a single issue of the *Philosophical Transactions of the Royal Society*, Dr. Powell's favored journal. Having completed his clinical training at St. George's Hospital in London, Brian stayed in London to complete his house officer jobs and subsequent training in histopathology and neuropathology. One of the best places to train in neuropathology was the National Hospital for Neurology and Neurosurgery at Queen Square. In the 70's and 80's, the department was run by a series of distinguished neuropathologists, many of whom edited Greenfield's Neuropathology, and Brian was one of two trainees mentored in the early 80's by Professor Leo Duchen (Editor for editions 4 & 5). The other trainee was Seth Love. During his training, Brian increasingly gravitated towards the pediatric neuropathology service, inspired by Dr. Magda Erdohazi, who had appointments at Queen Square and Great Ormond Street Hospital for Children (GOSH).

Brian was appointed to a joint position at Queen Square and GOSH upon Dr. Erdohazi's retirement and ran the service at GOSH single-handed for many years, until the arrival of his successor, Dr. Thomas Jacques. During that time, Brian encountered a wide range of rarities, many of which he shared with the neuropathology community in publications and at conferences. His research interests became focused on metabolic, degenerative and developmental disorders, and he provided advice to many British colleagues who welcomed his expertise when evaluating challenging cases. If Brian was not publishing his experience with pediatric neuropathology in scientific papers, he was contributing chapters on the subject in learned textbooks. He had a long association with Greenfield's Neuropathology, contributing a peerless eight chapters over five editions! Brian edited Developmental Neuropathology, with Dr. Jeff Golden, and wrote for all three editions of Neuropathology, a reference atlas of the CNS. All his chapters were beautifully illustrated with material he had seen at GOSH.

In 2009, upon retiring from the National Health Service, Dr. Harding made the journey to Philadelphia, taking up a position as Professor of Pathology and Laboratory Medicine at the Children's Hospital of Philadelphia and the University of Pennsylvania School of Medicine. Here, he worked alongside Drs. Lucy Rorke, Jeff Golden, Mariarita Santi, and Alex Judkins. At CHOP, Brian has maintained his interests in SMA and rare brainstem malformations and has even encountered new entities, such as Amish cerebro-renal disease.

Those who know Brian outside neuropathology see a cultured enthusiast of the Arts and a sociable and knowledgeable friend, always ready with an interesting story or piece of news – whether from his life or from the media. He is an excellent pianist and a devotee of the Philadelphia Orchestra and Metropolitan Opera. He is also extremely knowledgeable about ceramic art and has an impressive collection in his apartment, which is displayed mainly on the floor, prompting visitors to circle pieces with admiration and some trepidation. Brian has loved his time in Philadelphia but, in his cosmopolitan way will be returning to London in two years, where he will surely enjoy the Arts scene and be ready to entertain visiting neuropathologists with news of friends and developments in the field.



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

What Every Neuropathologist Needs to Know

Saturday, June 9, 2018

WHAT EVERY NEUROPATHOLOGIST NEEDS TO KNOW

What Practical Information Every Neuropathologist Needs to Know About the US Legal System

Time: 4:45 pm - 5:15 pm

R. Ross Reichard, MD, Mayo Clinic, Rochester, MN

I. Learning Objectives:

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

- 1. Define the types of legal proceedings a pathologist might participate.
- 2. Illustrate the medicolegal role of pathology results.
- 3. Clarify the intersection of quality, peer review and the law.
- 4. Define how a pathologist may be a witness.

II. Abstract and Relevant Resources

The legal system of the United States is complex, with nuances that are particular to its many jurisdictions. The neuropathologist may professionally interact with the legal system in both criminal and civil proceedings as either a fact or expert witness. The nature of the legal issue at hand will define the pathologist's role and determine what actions are required and/or requested. The pathology report (findings) may be critical in particular types of cases (e.g. neuropathology of alleged child abuse) or immaterial, even when clinically unexpected. The interactions between the pathologist, as a witness, and the attorneys, judge and jury will be determined by the type of legal case and venue in which their testimony is taken (e.g. jury trial, video deposition, affidavit). Peer review is intended to improve patient care and the confidentiality and protection of these activities is determined at the state level, but the intersection between quality endeavors and the law maybe blurry. Despite the complexity of the legal system, the types of situations a pathologist will be involved are relatively few and that will define their role.

References:

1. Davis, GG. Pathology and Law: A practical guide for the pathologist. New York, NY: Springer-Verlag; 2004.

III. Faculty Biography:

Dr. Ross Reichard is an Associate Professor of Pathology at the Mayo Clinic College of Medicine where he holds several leadership roles including Vice Chair of Quality for the Department of Laboratory Medicine and Pathology, Vice Chair of Practice for the Division of Anatomic Pathology and Chief Medical Examiner for the Southern MN Regional Medical Examiner's Office. His particular areas of interest are forensic neuropathology, neurodegenerative diseases, and traumatic brain injury, particular pediatric traumatic brain injury.

WHAT EVERY NEUROPATHOLOGIST NEEDS TO KNOW

What Every Neuropathologist Should Know: Update on c-IMPACT-NOW

Time: 5:15 pm - 5:45 pm

Daniel J. Brat, MD, PhD, Magerstadt Professor and Chair, Department of Pathology, Northwestern Feinberg School of Medicine

I. Learning Objectives:

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

- 1. Explain the gaps in the WHO classification that are addressed by cImpact-Now.
- 2. Describe the correct use of the Not Otherwise Specified (NOS) designation in WHO.
- 3. State which genetic markers in an IDH-wildtype diffusely infiltrating astrocytoma would qualify it for a grade IV designation.

II. Abstract and Relevant Resources

World Health Organization (WHO) central nervous system tumor classification represents the primary source of updates on diagnostic classes, grades and criteria. However, recent and ongoing advances in molecular pathogenesis warrant more rapid integration into clinical practice between WHO updates. To accomplish this, cIMPACT-NOW (the Consortium to Inform Molecular and Practical Approaches to CNS Tumor Taxonomy – Not Official WHO) was established in 2016. Since then, cIMPACT-NOW has convened three separate working committees to address classification and grading questions and challenges. My lecture will cover the progress that these working committees have made in on their specific topics.

References:

- 1. Louis DN, Aldape K, Brat DJ, et al. Announcing cIMPACT-NOW: the Consortium to Inform Molecular and Practical Approaches to CNS Tumor Taxonomy. Acta Neuropathol. 2017;133(1):1-3.
- 2. Louis DN, Wesseling P, Paulus W, Giannini C, Batchelor TT, Cairncross JG, Capper D, Figarella-Branger D, Lopes MB, Wick W, van den Bent M. clMPACT-NOW update 1: Not Otherwise Specified (NOS) and Not Elsewhere Classified (NEC). Acta Neuropathol. 2018;135:481-484.
- 3. Louis DN, Giannini C, Capper D, Paulus W, Figarella-Branger D, Lopes MB, Batchelor TT, Cairncross JG, van den Bent M, Wick W, Wesseling P. cIMPACT-NOW update 2: diagnostic clarifications for diffuse midline glioma, H3 K27M-mutant and diffuse astrocytoma/anaplastic astrocytoma, IDH-mutant. Acta Neuropathol. 2018;135:639-642.
- 4. Wijnenga MMJ, Dubbink HJ, French PJ, et al. Molecular and clinical heterogeneity of adult diffuse low-grade IDH wild-type gliomas: assessment of TERT promoter mutation and chromosome 7 and 10 copy number status allows superior prognostic stratification. Acta Neuropathol. 2017;134(6):957-959.
- 5. Aibaidula A, Chan AK, Shi Z, et al. Adult IDH wild-type lower-grade gliomas should be further stratified. Neuro Oncol. 2017;19(10):1327-1337.
- 6. Aoki K, Nakamura H, Suzuki H, et al. Prognostic relevance of genetic alterations in diffuse lower-grade gliomas. Neuro Oncol. 2018;20(1):66-77.
- 7. Weller M, Weber RG, Willscher E, et al. Molecular classification of diffuse cerebral WHO grade II/III gliomas using genome- and transcriptome-wide profiling improves stratification of prognostically distinct patient groups. Acta Neuropathol. 2015;129(5):679-693.

III. Faculty Biography:

Dr. Brat received his MD and PhD from the Mayo Medical and Graduate Schools and completed Residency in Anatomic Pathology and a Fellowship in Neuropathology at Johns Hopkins Hospital. Dr. Brat's is a practicing surgical neuropathologist with special expertise in neoplastic diseases. He also directs an NIH-funded basic and translational research lab that investigates mechanisms of glioma progression, including the contributions of hypoxia, genetics, tumor microenvironment and stem cells, using Drosophila and mice to model the human disease. He also has an interest in the in silico investigation of brain tumors and has used large-scale clinical and molecular databases, such as The Cancer Genome Atlas (TCGA), to address fundamental questions in human glioma behavior. He has over 18 years of experience in brain tumor research and has written more than 200 peer-reviewed manuscripts and reviews. Dr. Brat has served in leadership positions that oversee clinical practice and investigation in Oncology and Pathology, including the TCGA Glioblastoma and Lower Grade Gliomas Working Groups; the College of American Pathologists (CAP) Glioma Guidelines Committee; the Executive Council of the American Association of Neuropathologists; the Board of Directors for the Society of Neuro-oncology; the WHO Committee for Classification of Brain Tumors; and the AJCC Expert Panel. He is a member of the American Society for Clinical Investigation.



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Diagnostic Slide Session Saturday, June 9, 2018

Learning Objectives:

- 1. Generate a differential diagnosis of degenerative disorders affecting white matter.
- 2. Describe recently recognized neoplasms and other cellular proliferative disorders affecting the nervous system, and their underlying genetic bases.
- 3. Outline the work-up of vacuolating myopathic disorders and their genetic causes.
- 4. Distinguish among individual tau disorders causing neurodegeneration.
- 5. Recognize substrates of cystic lesions affecting the nervous system.

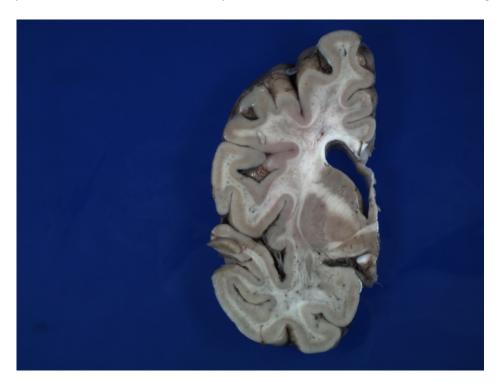
Submitted By:

Diana Thomas, MD, PhD, and Julia Kofler, MD UPMC Department of Pathology, 200 Lothrop Street, Scaife Hall S701, Pittsburgh, PA 15261

Clinical History:

The patient was a 61-year-old woman who started to experience syncopal episodes at about age 49. As part of her work-up she had a CT scan of the brain which revealed abnormalities in the white matter. She subsequently developed gait abnormalities but remained cognitively intact with preserved memory. In her late 50s, she lost the ability to walk and developed urinary incontinence. In the last few months before death, she experienced progressive lapses in her short-term memory.

Her family history was notable for a similar disease process in her father, two of her three siblings and in one cousin.



Material Submitted:

Gross image, 1 H&E slide, 1 Luxol fast blue/PAS stain

- 1. Diagnosis and differential diagnosis
- 2. Ancillary studies

Submitted By:

Julieann Lee¹, Sean Ferris¹, David Solomon¹, Dimitri Trembath², Arie Perry¹

- 1. Neuropathology, University of California, San Francisco, CA.
- 2. Neuropathology, The University of North Carolina at Chapel Hill, Chapel Hill, NC.

Clinical History:

The patient is a 25-year-old man who presented with left hand numbness and headaches. He was found to have a 4.7 cm heterogeneously enhancing intra-axial mass in the left cerebellar hemisphere, with mass effect on the fourth ventricle. A biopsy was performed.

Material submitted:

H&E slide from biopsy of cerebellar mass

- 1. Differential diagnosis
- 2. Immunohistochemical and molecular evaluation

Submitted By:

Karra A. Jones, MD, PhD and Steven A. Moore, MD, PhD The University of Iowa, Department of Pathology, Iowa City, IA 52242

Clinical History:

2-year-old male with mild global developmental delay, frequent falls, and oropharyngeal dysphagia. Neurologic exam revealed a positive Gowers' sign and abnormal gait consistent with a compensated Trendelenburg. His family history was positive for a maternal uncle with unknown neuromuscular disease requiring the use of a wheelchair since age 12. The serum CK was elevated in the range of 1300-1400 U/L.

Genetic testing prior to muscle biopsy included a non-diagnostic microarray, normal DMD del/dup testing and complete sequencing, normal GAA enzymatic assay, and negative congenital hypotonia next generation sequencing panel.

Material Submitted:

H&E stained cryosection of muscle

- 1. Differential diagnosis
- 2. Approach to diagnostic testing

Submitted By:

Anne Shepler, MD and Julia Kofler, MD University of Pittsburgh Medical Center Presbyterian Hospital Division of Neuropathology 200 Lothrop Street, Room S701 Scaife Hall Pittsburgh, PA 15213

Clinical History:

The patient was a 37-year-old lawyer with no family history of dementia, who at age 33 began to have performance issues at work with inattentiveness to detail and a lack of concern for deadlines. He became progressively abulic and socially withdrawn with a loss of interest in his hobbies and personal appearance. By age 34, he began to choke frequently on food and was noted by his wife to have developed a nasal voice and bilateral ptosis. While he could remember his two young children's names, he was unable to care for them. He also developed reduplicative paramnesia wherein he insisted that there were two "2B" apartments in his apartment building where another woman (with his wife's name) lived with two children. While he did not get agitated if corrected, he returned to this delusion repeatedly.

On neurological examination, he showed no lateralizing sensory or motor signs and muscle strength was normal. No fasciculations were present. His reflexes were brisk and symmetric with no sustained clonus or Babinski sign. He was fluent with intact comprehension, repetition, naming, reading, and writing. He often answered questions too quickly with impulsive errors as he would not wait of the conclusion of the question; he was undisturbed when he made a wrong answer. Other than ptosis and a mild weakness of the orbicularis oculi, the cranial nerve examination was unremarkable. Initial MRI and CT scans were normal. A PET scan revealed bilateral frontal diminution of glucose utilization, worse on the right than the left, with extension to the right caudate. An EMG was negative.

Autopsy findings:

The brain weight was 1160 grams (fresh). Neuropathologic examination was notable for moderate atrophy of the frontal lobe and caudate and mild atrophy of the temporal and parietal lobes.

Material Submitted:

One H&E slide of frontal cortex

- 1. Differential diagnosis
- 2. Useful immunohistochemical stains

Submitted By:

Aivi T. Nguyen and Edward B. Lee Hospital of the University of Pennsylvania 3400 Spruce Street Philadelphia, PA 19104

Clinical History:

The decedent was a 72-year-old right handed female who presented for evaluation of incoordination and frequent falls. She complained of multiple episodes of imbalance and demonstrated increasing forgetfulness, anosmia, and micrographia. She was evaluated by multiple neurologists and eventually treated with carbidopa/levodopa with no improvement. Physical exam was remarkable for masked facies, impaired extraocular movements with jerky saccades, axial rigidity, moderate bradykinesia, and a narrow-based gait with short strides, absent arm swing, and no postural control. Over the course of five years she developed severe cognitive defects, including word finding difficulties and memory decline, and was eventually transferred to hospice where she died.

Autopsy Findings:

The brain was examined 14 hours post-mortem and weighed 1147 grams. Gross findings included diffuse cerebral atrophy, severely atrophic hippocampus and amygdala, ex-vacuo hydrocephalus, and substantia nigra depigmentation.

Material Submitted:

1 H&E section of the hippocampus

- 1. Differential diagnoses
- 2. Useful immunohistochemical/additional stains
- 3. Revised differential diagnoses after additional stains and biochemical work-up

Submitted By:

E. Kelly S. Mrachek, M.D., M. Beatriz S. Lopes, M.D., Ph.D. University of Virginia Health System
Department of Pathology, Division of Neuropathology
Box 800214
Charlottesville, VA 22908-0214

Clinical History:

The patient is a 6-year-old male who presented with a two-week history of headaches with recent nausea and vomiting. His PCP diagnosed strep throat, and treatment with antibiotics failed. While getting additional labs drawn, the patient had acute onset of left sided facial drooping, and was brought to the ER. MRI of the brain with and without contrast showed a 5.2 cm avidly enhancing, extra-axial mass in the left cerebellopontine angle with extension into the internal auditory canal. A left retrosigmoid craniotomy and gross total resection were performed at the University of Virginia.

Material Submitted:

MRI image of brain demonstrating a left CPA mass, and an H&E section of the mass.

- 1. What is the final integrated histomolecular diagnosis of this left CPA mass?
- 2. What is the most common molecular genetic alteration in lesions of this type?

Submitted By:

Jason A. Gregory, CPT USA, MD, Meggen Walsh, MD and Jesse Lee Kresak, MD University of Florida
Department of Pathology, Immunology, and Laboratory Medicine
1600 SW Archer Rd
PO Box 100275
Gainesville. FL 32610

Clinical History:

A twenty-year-old male presents for esophagogastroduodenoscopy for persistent dysphagia with a specific chief complaint of "I'm unable to chew because I can't open my mouth and my teeth hurt." He has a history of hypertension, obstructive sleep apnea, and a congenital musculoskeletal disorder. During the procedure, the patient was noted to have airway instability with subsequent desaturation. Resuscitative efforts were unsuccessful and the patient expired.

Autopsy findings:

General autopsy revealed a thin man of short stature weighing 75 lbs. He had various musculoskeletal abnormalities including scoliosis, pes planus, and asymmetrical muscular atrophy. The brain weighed 1,560 grams. Gross examination revealed an enlarged brainstem with a markedly stenotic aqueduct. The medulla appeared ovoid rather than the normal "papilionaceous" shape. Internal architecture of the brainstem appeared distorted. The dentate nucleus of the cerebellum was difficult to delineate grossly. The remainder of the cerebrum and cerebellum appeared normal.

Material Submitted:

H&E-stained section of the medulla

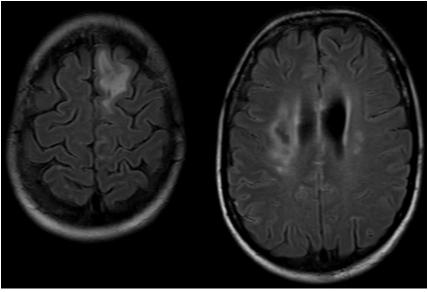
- 1. Differential diagnosis and ancillary studies
- 2. Pathogenesis

Submitted By:

Kyle Conway, Theodore Brown, David Gordon, John Kennedy, and Sriram Venneti University of Michigan Department of Pathology 1301 Catherine St. Ann Arbor, MI 48109

Clinical History:

The decedent is a 50-year-old female who presented with hemiparesis, expressive dysphasia, headaches, visual disturbances, and paresthesias. Imaging showed multifocal frontal and temporal lesions crossing the corpus callosum. A frontal lobe biopsy at this time demonstrated only reactive gliosis and perivascular inflammation. Several months later, she presented to the emergency department with acute worsening of symptoms. Repeat imaging showed findings consistent with an ischemic stroke. Serologic workups for autoimmune disease and infection were negative. She was discharged, and her neurologic condition worsened over the course of a year. She passed way after an acute deterioration in neurologic status.



Autopsy findings:

Gross autopsy findings included multiple areas of softened parenchyma in the left frontal, right fronto-parietal, right superior parietal, left mid parietal, and left occipital lobes. The lumina of the carotid arteries were grossly narrowed (approximately 0.1 cm in diameter).

Material Submitted:

- 1. MRI images
- 2. Gross photographs of cortex and base of brain
- 3. H&E stained slide of cortical lesion
- 4. Trichrome stained slide of carotid artery

- 1. Explain the differential diagnosis of this clinical history and workup
- 2. Understand the classification and subtyping for this disease

Submitted By:

Angela N. Viaene, MacLean P. Nasrallah, and Zissimos Mourelatos Hospital of the University of Pennsylvania, Department of Pathology and Laboratory Medicine, Division of Neuropathology, 6th Floor Founders, 3400 Spruce Street, Philadelphia, PA 19104

Clinical History:

A 38-year-old female with no significant past medical history presented with headache, nausea and photophobia for three days. She subsequently developed visual loss and aura in both eyes which prompted imaging studies. A large, predominantly cystic left parietal mass measuring 6.4 x 4.3 x 5.2 cm associated with irregular and nodular peripheral enhancement and areas of hemorrhage around its periphery was seen on brain MRI. There was mass effect including 7-8 mm rightward midline shift and partial effacement of the basal cisterns. The patient underwent left craniotomy for gross-total resection of the enhancing mass.



Material Submitted:

- 1. One representative H&E slide
- 2. Representative pre-operative MRI image

- 1. Differential diagnosis
- 2. Molecular findings

Submitted By:

Missia Kohler, M.D., Jamie Walker, M.D., Ph.D., Qinwen Mao, M.D., Ph.D., and Eileen Bigio, M.D. Northwestern University Feinberg School of Medicine Department of Pathology 710 North Fairbanks Ct Chicago, IL 60611

Clinical History:

The patient initially presented in 2009 at the age of 72 to neuropsychiatry with a two-year history of minor memory problems. His cognitive exam revealed memory issues that were primarily amnestic, moderate executive dysfunction and a MoCA score of 20/30. His sensory and motor exam were unremarkable. His MRI demonstrated more atrophy than expected for his age. His APOE allele status was $\varepsilon 4/\varepsilon 4$. In 2016, he was still driving, but experienced bowel and bladder incontinence and was having a "hard time fixing things" in the home. By 2017, he was displaying more word-finding difficulties and required instructions for most daily tasks. In the autumn of 2017, he had a few falls and hospitalizations for infections. Thereafter, he rapidly declined, and died at the age of 80 in October of 2017.

Material Submitted:

One H&E stained slide

- 1. Differential diagnosis
- 2. Immunohistochemical work-up

Submitted By:

M. Adelita Vizcaino M.D.¹, Verena Staedtke M.D.², Allan Belzberg, M.D.², Fausto J. Rodriguez M.D.¹

- 1. Division of Neuropathology
- 2. Department of Neurology and Neurosurgery, Johns Hopkins University School of Medicine

Clinical History:

An 11-year-old boy was previously healthy until approximately the year prior, when the family noticed that he was walking on the lateral aspect of his right foot, resulting in callus formation and closed pressure ulcer. During physical examination, it was noted that the patient had a high arch of the right foot as well as a 3-cm. leg length discrepancy. Several subtle caféau-lait spots were also identified. Subsequent radiographs demonstrated multiple stress fractures, which prompted MRI of the spine and lower extremities demonstrating enlargement of the right lumbosacral plexus and right sciatic nerve extending distally down the thigh, progressing to include the tibial nerve and common peroneal nerve to the bifurcation with continued involvement of the deep and superficial peroneal nerve branches to the foot. At the time of surgical intervention for orthopedic corrections, a biopsy of the tibial nerve was performed.

Material Submitted:

Virtual H&E image of tibial nerve biopsy

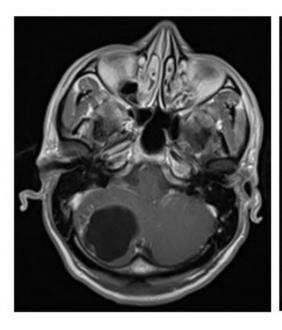
- 1. Differential Diagnosis
- 2. Pathogenesis
- 3. Role of molecular genetics

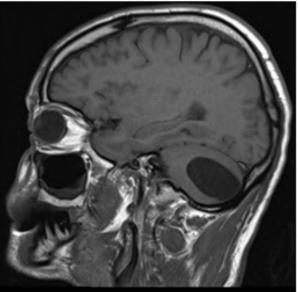
Submitted By:

Isaac Solomon, M.D., Ph.D.; Sandro Santagata, M.D., Ph.D. Department of Pathology, Brigham and Women's Hospital, 75 Francis Street, Boston, MA, 02115, USA

Clinical History:

A 53-year-old man from Cape Verde presented following an acute onset of vertigo, nausea, and vomiting. His past medical history was notable for a brain lesion resected in his 20s, Hodgkin's lymphoma treated with ABVD chemotherapy seven years earlier, and latent tuberculosis treated with nine months of anti-mycobacterial drugs. At the time of presentation, he lived in Massachusetts and had last visited Cape Verde two years earlier. He had recently traveled to Mexico. A neurological examination of the patient revealed ataxic gait, up-beating nystagmus, and right-sided dysmetria. No abnormalities were identified by routine blood testing that included a complete blood count, a basic metabolic panel and liver function tests. HIV antibody, cysticercosis IgG, and tuberculosis interferon gamma release assay were negative. Toxoplasma IgG antibody was positive. A 4-cm lobular, cystic, rim-enhancing lesion in the right hemisphere of the cerebellum was identified using MRI. The patient was taken to the OR for resection.





Material Submitted:

H&E section of cerebellar mass

- 1. Histologic characteristics
- 2. Differential diagnosis (imaging, histology)
- 3. Short review/summary



AANP

AMERICAN ASSOCIATION OF NEUROPATHOLOGISTS

Presidential Symposium Sunday, June 10, 2018

Learning Objectives:

- 1. Describe recent technological advances in methods of pathological diagnosis.
- 2. Explain how to implement and validate a digital pathology system.
- 3. Outline the role of machine learning in diagnostic pathology.

PRESIDENTIAL SYMPOSIUM

Evolution of Diagnostic Techniques in Pathology: Moving Beyond the Microscope

Time: 8:05 am - 8:30 am

Elizabeth J. Cochran, M.D., Medical College of Wisconsin, Milwaukee, WI

I. Learning Objectives:

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

- 1. List the sequence of developments that led to use of light microscopic evaluation of tissue as the primary mode of disease diagnosis in pathology.
- 2. Compare recently developed techniques of tissue evaluation that are alternatives and/or adjuncts to evaluation of light microscopic sections on glass slides by pathologists.
- 3. Define computational pathology, including an explanation of how it may change the practice of pathology.

II. Abstract and Relevant Resources

For the past 150 years, diagnostic pathologists have focused upon the evaluation of normal and abnormal tissue morphology as seen upon examination of thin tissue sections on glass slides through the light microscope. However, new technologies are emerging that provide alternatives to this approach; the development of digital pathology and computational analyses has supported this growth. These new techniques range from next generation sequencing to new methods of identifying morphology, and many require computer system analysis of the large volume complex datasets that are produced. As these new tools mature and are brought into use in clinical medicine, the role of the pathologist is evolving to include integration of molecular and computational data in addition to the traditional role of histopathologist.

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III. Faculty Biography:

Dr. Cochran obtained her M.D. degree from Rush Medical College in Chicago, Illinois in 1982. She completed residency training in anatomic pathology at Northwestern University in Chicago, Illinois, followed by a neuropathology fellowship with Drs. Pierluigi Gambetti and Uros Roessmann at Case Western Reserve University Institute of Pathology in Cleveland, Ohio. She joined the faculty of Rush University Medical Center in 1989 as an assistant professor, and she helped to establish the Alzheimer's disease brain bank at Rush. She was neuropathology core leader in the Rush NIH-funded Alzheimer's disease center and Religious Order Study grants, participating in research on Alzheimer's disease and aging. Over the last decade, she has focused on education of medical students and residents, first directing the Rush pathology residency program, medical student pathology course and clerkships, at Rush, while continuing to run the diagnostic neuropathology and autopsy services. In 2010, she joined the faculty of the Medical College of Wisconsin in Milwaukee, Wisconsin as Professor of Pathology and is currently director of the autopsy and neuropathology services and of medical student education in pathology.

PRESIDENTIAL SYMPOSIUM

Teleneuropathology for Intraoperative Consultation

Time: 9:15 am - 10:00 am

Clayton A. Wiley, MD, PhD, University of Pittsburgh Medical Center, Pittsburgh, PA

I. Learning Objectives

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

- 1. Describe how telepathology evolved to its current stage
- 2. Recognize and anticipate the challenges of implementing telepathology
- 3. Describe how to avoid problems inherent in rendering pathological diagnoses remotely
- 4. Recognize many legal issues related to telepathology are in flux with great variation between states
- 5. Recognize that telepathology can successfully distribute medical expertise to regions of need.

II. Abstract & Relevant References

Telepathology has been under development for close to half a century. With the advent of the Internet and availability of rapid information transmission, the pace of development has quickened. While technological and security hurdles have been surpassed, there are substantial challenges associated with the practice of medicine that need to be surmounted in order for telepathology to fully replace onsite intraoperative consultation. Being virtually present is not the same as being present. Many practical and unrecognized assumptions need to be addressed before telepathology can be successfully integrated into patient care. Human adoption of any new technology is always subject to substantial pushback, however, the advantages of telepathology now outweigh disadvantages and will drive adoption system wide.

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III. Faculty Biography:

Dr. Wiley did his undergraduate training at the University of Chicago and his MD/PhD training in Neurosciences at the University of California San Diego. This was followed by Anatomical Pathology residency at University of California San Francisco and Neuropathology fellowship back at UCSD. In 1985 Dr. Wiley was appointed Assistant Professor of Pathology at UCSD where he advanced to the rank of full professor. In 1993 he was recruited to the University of Pittsburgh Medical Center as Director of the Division of Neuropathology and its fellowship program. The Division has been involved in telepathology for 25 years.

Throughout his professional career he has been actively involved in educating physician scientists at both pre- and postgraduate stages. From 1997 to 2012 Dr. Wiley served as Director of the Pittsburgh Medical Scientist Training Program and Associate Dean in the University of Pittsburgh School of Medicine. Throughout this time, he was actively involved in the National Association of MD/PhD Programs and the MD/PhD Section of the GREAT group in the AAMC where he served as President and Chair respectively. He also served on the AAMC Council of Academic Societies Task Force on Dual Degree Programs. Dr. Wiley has maintained an active NIH funded research program investigating the pathogenesis of viral mediated neurodegeneration. He has published over 250 peer-reviewed publications and was elected a Fellow of the American Association for the Advancement of Science in 1997. Currently his research is focused on the role of innate and adaptive immunity in protecting the brain from viral infections.

PRESIDENTIAL SYMPOSIUM

Liquid Biopsy in Cancers of the Central Nervous System

Time: 10:45 am - 11:30 pm

Matthias Holdhoff, MD, PhD, Associate Professor of Oncology and Neurosurgery, Sidney Kimmel Comprehensive Cancer Center, Johns Hopkins University School of Medicine

I. Learning Objectives

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

- 1. Describe current concepts and potential applications of 'liquid biopsy' in oncology.
- 2. Discuss the potential role of 'liquid biopsy' as a non- or minimally invasive tool in CNS cancers.
- 3. Explain the specific hurdles in the development of blood- or CNS-based markers in CNS cancers.

II. Abstract & Relevant References

The development and clinical application of 'liquid biopsies' is a promising topic in oncology. In this lecture, we will review current concepts of circulating biomarkers in cancers with specific focus on the potential role of liquid biopsies in cancers of the central nervous system. Specific challenges for development of such markers in brain cancers will also be discussed.

References:

- 1. Bettegowda C et al. Detection of Circulating tumor DNA in early- and late stage human malignancies. Sci Transl Med. 2014 Feb 19;6(224):224ra24
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III. Faculty Biography:

Dr. Holdhoff is an associate professor of oncology and neurosurgery at The Sidney Kimmel Comprehensive Cancer center at Johns Hopkins. He is a clinical translational investigator in neuro-oncology and a clinical expert in malignant gliomas and central nervous system lymphomas. His research focuses on early phase clinical trials and biomarker research in cancers of the central nervous system. Dr. Holdhoff received his medical degree at Freie Universität Berlin and completed his dissertation at Charité University Medicine Berlin. He completed his internal medicine residency training at Johns Hopkins Bayview Medical Center and a medical oncology fellowship at The Johns Hopkins Hospital, during which he also pursued research training within the Ludwig Center for Cancer Genetics and Therapeutics.

PRESIDENTIAL SYMPOSIUM

Deep Learning Neural Networks

Time: 11:30 am - 12:15 pm

Lee Cooper, PhD, MS, Emory University School of Medicine, Atlanta, GA

I. Learning Objectives

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

- 1. Summarize the basic components of a deep learning neural network
- 2. Contrast the differences between engineered and adaptive approaches to solving machine learning problems
- 3. Explain how deep learning can be used to solve a problem like predicting clinical outcomes

II. Abstract & Relevant References

This lecture will discuss machine learning and the development and application of predictive models in digital pathology. We will discuss advanced in an area called deep learning that can create powerful models that can learn to identify important patterns in histology and perform diagnostic tasks with human-level accuracy. We will show how this technology can be used to build advanced survival models that integrate histology and genomics to accurately predict the clinical outcomes of patients diagnosed with glioma.

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- 1. P Mobadersany, S Yousefi, M Amgad, DA Gutman, JS Barnholtz-Sloan, JE Velazquez-Vega, DJ Brat, LAD Cooper, Predicting cancer outcomes from histology and genomics using convolutional networks, PNAS, 115(13), pp. 2970-9, March 2018
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- 4. S Webb, Deep learning for biology, Nature, 554, pp. 555-7, February 2018
- 5. Y LeCun, Y Bengio, G Hinton, Deep Learning, Nature, 521, pp. 436-44, May 2015

III. Faculty Biography:

Lee Cooper, PhD is an engineer and an Assistant Professor of Biomedical Informatics at Emory University. He received a PhD in Electrical and Computer Engineering in 2009 from Ohio State University, where he began research in digital pathology in 2005. His research is funded by the Informatics Technology for Cancer Research program of the National Cancer Institute and seeks to develop software algorithms and infrastructure for analyzing genomic and digital pathology data.

2017 DONATIONS

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