PROGRAM

EIGHTY FIFTH
ANNUAL MEETING
OF THE
AMERICAN
ASSOCIATION
OF
NEUROPATHOLOGISTS

JUNE 11-14, 2009

CROWNE PLAZA RIVERWALK HOTEL

SAN ANTONIO, TEXAS

CONTENTS

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GENERAL INFORMATION

Hotel: Crowne Plaza Riverwalk San Antonio
111 E. Pecan Street
San Antonio, Texas 78205

Phone: 210-354-2800
Fax: 210-354-2700

ALL MEETING SESSIONS WILL BE HELD IN THE CROWN PLAZA RIVERWALK HOTEL

All platform presentations and general sessions (Special Lectures, Korey Lecture, DeArmond Lecture, TEVA/Parisi Lecture, Business Meetings, Diagnostic Slide Session, and Presidential Symposium) will be held in the **Texas Ballroom** of the hotel.

All poster sessions will be held in Texas Ballroom C and Grand Foyer

PRE-REGISTRATION PICK-UP

Attendees pre-registered and pre-paid for the Special Course and/or Meeting will have their name badge, course syllabus, program booklets, reception ticket(s) and registration receipt ready for pick-up at the AANP Registration Desk, located in the Grand Foyer of the hotel. On-site registration, additional tickets for the Annual Reception, and additional copies of the May 2009 JNEN will be available at the Desk.

REGISTRATION DESK

Location	**Grand Foyer**	
Time	Wednesday, June 10	6:00 pm – 9:00 pm
	Thursday, June 11	6:30 am - 12 noon 6:30 pm – 9:00 pm
	Friday, June 12	7:00 am - 12 noon 5:30 pm – 6:00 pm
	Saturday, June 13	7:00 am - 12 noon

PLEASE, wear your name badge!

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 Friday evening reception.

NOTES to PRESENTERS

Platform Presenters (PowerPoint)

All platform presentations will be held in either the **Texas Ballroom A or B** of the hotel. *All* general sessions (Special Lecture, Korey Lecture, Business Meetings, Diagnostic Slide Session, and Presidential Symposium) will be held in the **Texas Ballroom B**.

Presenters should use PowerPoint for their presentation.
All PowerPoint presentations will be transferred onto a show computer prior to the start time of each session. Each room will be equipped with a lectern, audience microphones, central computer (loaded with MS Office XP), LCD/Data projector, screens and a laser pointer.

Special Notes for PowerPoint presenters:

- Each speaker must bring his/her PowerPoint presentation on a disc (CD-ROM) or USB memory stick.
- Please title the presentation with your name (name.ppt).
- Macintosh users, be sure to save your presentation as .ppt (*your name.ppt)*. If the ".ppt" extension is not present in the file name, the file will not be recognized by the PC computer.

- Label your disc with your name, session name, time, and day of presentation. Your presentation will be transferred onto the show computer for each session by the technician. Please make sure your presentation is in its final form, since once loaded onto the show computer, no changes can be made.
- Please take your disc or memory stick to the room in which you will be presenting, Texas Ballroom A or B, at one of the times indicated below. ***It is your responsibility to get your file to the AV staff prior to your presentation.***
- The AV staff will be available to load your file onto the computer during scheduled evening and morning times. **These will be the *only* times available to you to load and test your presentation.**

Schedule for Loading PowerPoint Presentations

Load show computer in *Texas Ballroom A or B*	
Wednesday, June 10	5:30 pm - 6:30 pm
Thursday, June 11	6:45 am - 7:45 am 5:30 pm - 6:30 pm
Friday, June 12	6:45 am - 7:45 am 6:00 pm - 7:00 pm
Saturday, June 13	6:45 am – 7:45 am 6:30 pm - 7:45 pm
Sunday, June 14	6:45 am - 7:45 am

- **If you are presenting in a morning session, it is preferable to check in the previous day**. Same-day presentations may be loaded in the morning prior to session start time, but since this time necessarily is limited, you are encouraged to have your presentation loaded on the evening preceding your talk. Presenters at the evening Diagnostic Slide Session also will be able to submit their files on Saturday evening from 6:30-7:45 pm.
- To avoid time delays and potential problems with your presentation, you will ***not*** be allowed to use your own computer, although you may bring your laptop as a backup.

Notes to Poster Presenters

All poster sessions will be held in **Texas Ballroom C and Grand Foyer.** Posters will be displayed all day Friday (Poster Session I) and all day Saturday (Poster Session II). Posters should be up by 8:00 am and taken down by 7:30 pm the same day. The poster board size is 8 ft wide x 4 ft high. Please plan your poster to be at least a few inches smaller in each direction. The poster board surface and construction should accommodate either Velcro or push pins.

To encourage interaction with interested attendees, authors are asked to be present at their posters for discussion/questions during morning or afternoon refreshment breaks, at the following designated times:

	Fri June 12 ***Poster Session I*** ***Authors Present at:***	**Sat June 13** ***Poster Session II*** ***Authors Present at:***
***EVEN* Numbered Poster**	*10:00 - 10:30 am*	*3:15 – 3:45 pm*
***ODD* Numbered Poster**	*4:00 – 4:30 pm*	*10:00 - 10:30 am*

SPEAKER READY ROOM

Electronic preview equipment will be available in the ***Boardroom.***

Location	**Boardroom**	
Time	Wednesday, June 10	7:00 pm - 9:00 pm
	Thursday, June 11	7:00 am - 5:30 pm
	Friday, June 12	7:00 am - 5:30 pm
	Saturday June 13	7:00 am - 5:30 pm

MICROSCOPE VIEWING ROOM

Multi-headed microscopes will be available in the ***Directors Room 1.***

Location	**Directors Room 1**	
Time	Thursday, June 11	7:00 am - 5:30 pm
	Friday, June 12	7:00 am - 5:30 pm
	Saturday June 13	7:00 am - 5:30 pm

BUSINESS MEETING

Location	**Texas Ballroom B**	
Time	Friday, June 12	11:45 am - 12:45 pm
	Saturday June 13	11:45 am – 12:45 pm

The Award for ***Meritorious Contributions to Neuropathology*** will be presented on Friday, June 12.

SPECIAL MEETINGS BY INVITATION ONLY

Date	**Meeting**	**Time/Location**
Thurs June 11	Executive Council Meeting	6:00 pm Executive Salon 4
	Awards Committee Meeting	5:30 pm – 6:30 pm Executive Salon 3
Fri June 12	Constitution Committee Meeting	7:00 am Executive Salon 3
	JNEN Editorial Board Meeting	7:00 am Executive Salon 5
	Acta Neuropathologica Meeting	11:30 am – 3:00 pm Executive Salon 3
	Trainee Luncheon	12:45 – 2:00 Executive Salon 5
	Awards Committee Meeting	5:30 pm – 6:30 pm Executive Salon 3
Sat June 13	NP Program Directors Meeting	1:00 pm – 2:00 pm Executive Salon 3
	Awards Committee Meeting	6:00 pm 7:30 pm Executive Salon 3
	Professional Affairs	6:00 pm – 8:00 pm Executive Salon 4
	Presidential Reception	6:30 pm – 8:00 pm Executive Salon 1
Sun June 14	Founders Breakfast	7:00 am – 8:00 am Boardroom

ABSTRACTS

Abstracts of the papers presented in the program are published in the May 2009 issue of the *Journal of Neuropathology and Experimental Neurology*. Separate abstract books are not printed. Members should take their copy of the JNEN to the meeting. Non-member registrants will receive a copy at registration. A limited number of copies of the JNEN will be available for purchase at the Registration Desk for $5 each**.**

DISCLOSURE/CONFLICT OF INTEREST

Disclosures (if any) are the responsibility of ***all*** platform and poster presenters. Conflicts of interest must be duly noted during the platform presentation or displayed on the poster.

CME Credit

The American Association of Neuropathologists is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians. The annual meeting is approved for credit under Category 1 of the Physician's Recognition Award of the American Medical Association on an hour-to-hour basis of attendance.

The AANP certifies the *Special Course* as a continuing medical education activity and meets the criteria for 8 credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association. Further, the AANP certifies the *Scientific Sessions*, the *Korey Lecture*, *the DeArmond Lecture, the Teva/Parisi Lecture, the Diagnostic Slide Session* and the *Presidential Symposium* as continuing medical education activities that meet the criteria for 15, 1, 1, 1, 3, and 3 credit hours, respectively, in Category 1 of the Physician's Recognition Award of the American Medical Association.

Activity	CME Credit Hours
Special Course	8
Scientific Sessions	12
Korey Lecture	1
DeArmond Lecture	1
TEVA/Parisi Lecture	1
Diagnostic Slide Session	3
Presidential Symposium	3
Total	29

Attendees should claim credit only for those activities attended. The official record of attendance form is included with your registration materials. Please fill in the number of hours attended, sign, and keep this form as your record of your attendance.

ANNUAL RECEPTION

The annual reception will be held 6:30 to 8:30 pm. Friday in San Antonio Ballroom and Foyer of the hotel. Registrants and guests of the AANP are welcome to attend. A cash bar will be available and hors d'oeuvres will be served. Additional tickets are $20 each for non-trainees, and $10 each for residents, fellows, or students, and may be purchased at the registration desk or at the door. Several "prizes" will be awarded.

Location	San Antonio Ballroom and Foyer	
Time	Friday, June 12	6:30 pm – 8:30 pm

SPONSORS and DONORS

This meeting is sponsored in part by generous contributions from several sponsors and donors. Please visit their displays and exhibits in the Texas Ballroom C.

Location	**Texas Ballroom C**	
Time	Thursday, June 11	12:00 pm – 5:30 pm
	Friday, June 12	7:00 am - 5:30 pm
	Saturday June 13	7:00 am - 5:30 pm

MEETING SPONSORS

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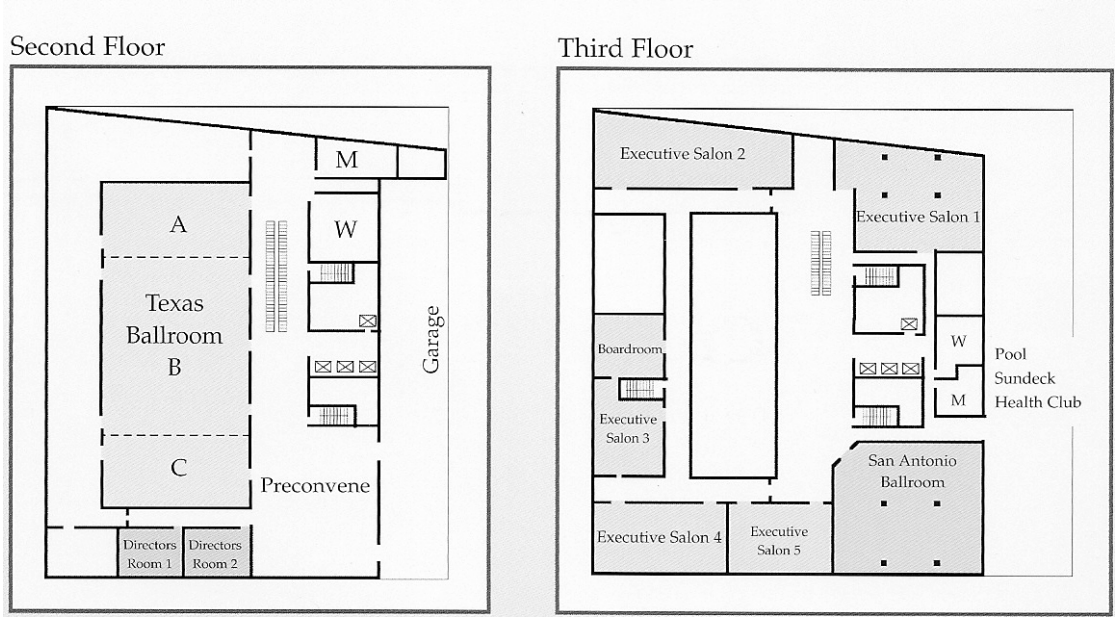
RECEPTION PRIZE CONTRIBUTORS

- Elsevier Inc.

Crowne Plaza Riverwalk San Antonio Floor Plan



PROGRAM and SCIENTIFIC SESSIONS

SPECIAL COURSE:

Location	Texas Ballroom A & B	
Date/Time	Thursday, June 11	8:00 am - 5:15 pm
	Recent Advances that Impact Research and Clinical Practices. Directors: George Perry, PhD And Amyn M. Rojiani, MD, PhD	

PLATFORM PRESENTATIONS

Location	Texas Ballroom A & B	
Date/Time	Friday, June 12	8:00 am – 4:30 pm
	Saturday, June 13	8:00 am – 4:30 pm

POSTER PRESENTATIONS

Location	Texas Ballroom C and Grand Foyer	
Date/Time	Friday, June 12 (Poster Session I)	8:00 am – 7:30 pm
	Saturday, June 13 (Poster Session II)	8:00 am – 7:30 pm

SAUL R. KOREY LECTURE

Location	Texas Ballroom B	
Date/Time	Friday, June 12	10:30 am - 11:30 am
	Mechanisms of Neurodegeneration in Prion Diseases Originating from the Neuronal Plasma Membrane Stephen J. DeArmond, MD, PhD *University California San Francisco, California, USA*	

DEARMOND LECTURE

Location	Texas Ballroom B	
Date/Time	Saturday, June 13	10:30 am - 11:30 am
	Decoding Alzheimer's Disease Gene by Gene Rudy Tanzi, PhD *Massachusetts General Hospital, Massachusetts, USA*	

TEVA/PARISI LECTURE

Location	Texas Ballroom B	
Date/Time	Saturday, June 13	4:30 pm – 5:30 pm
	Inflammation Induced Mitochondrial Injury: A Major Mechanism of Neurodegeneration Hans Lassman,PhD *University of Vienna, Vienna, Austria*	

DIAGNOSTIC SLIDE SESSION

Location	Texas Ballroom B	
Date/Time	Saturday, June 13	8:00 pm -11:00 pm

PRESIDENTIAL SYMPOSIUM

Location	Texas Ballroom B	
Date/Time	Sunday, June 14	8:00 am – 12 noon
	Oxidative Stress in Neurodegenerative Diseases	

MEETING AT A GLANCE

THURSDAY June 11, 2009	
	Texas Ballroom A & B
8:00 am - 5:15 pm	*SPECIAL COURSE* Recent Advances That Impact Research and Clinical Practices

(Abstract Numbers in Italics)

FRIDAY June 12, 2009			
	Texas Ballroom A	Texas Ballroom B	Texas Ballroom C and Grand Foyer
8:00 - 10:00 am	Platform 1 Neurodegenerative I: Prion *#1 - 8*	Platform 2 Experimental and Developmental Neuropathology *#9 - 16*	All Posters *Neurodegenerative Diseases* *#63 -92* *Others* *#93-111* *Tumors* *#112-157*
10:00 - 10:30 am	*REFRESHMENT BREAK*		
10:30 - 11:30 am	Saul R. Korey Lecture Mechanisms of Neurodegeneration in Prion Diseases Originating from the Neuronal Plasma *Stephen J. DeArmond, MD, PhD* *University California San Francisco, CA*		
11:45 - 12:45 pm	*BUSINESS MEETING I* *Texas Ballroom B*		
12:45 - 2:00 pm	*LUNCH*		
	Texas Ballroom A	Texas Ballroom B	
2:00 - 4:00 pm	Platform 3 Neurodegenerative II: α-Synclein & Ubiquitin *#17-24*	Platform 4 Tumors I: Gliomagenesis and Cell Pysiology *#25 -32*	
4:00 – 4:30 pm	*REFRESHMENT BREAK*		
4:30 – 5:30 pm	Special Lecture Sixty Seven Years Of The JNEN: What I Learned in Sixteen Of Those Years – And Can It Benefit You? *Michael N. Hart, MD, PhD* *University Wisconsin, WI*		

6:30 - 8:30 pm *ANNUAL RECEPTION:*
Executive Salon I

MEETING AT A GLANCE

(Abstract Numbers in Italics)

<table>
<tr><th colspan="4">SATURDAY June 13, 2009</th></tr>
<tr><td></td><td>Texas Ballroom A</td><td>Texas Ballroom B</td><td>Texas Ballroom C and Grand Foyer</td></tr>
<tr><td>8:00 -
10:00 am</td><td>Platform 5
Neurodegenerative III:
Plaques and Tangles

#139-146</td><td>Platform 6
Tumors II:
Prognosis and Therapy

#147-153</td><td rowspan="9">All Posters

Neurodegenerative Diseases
#63 -92

Others
#93-111

Tumors
#112-157</td></tr>
<tr><td>10:00 -
10:30 am</td><td colspan="2">REFRESHMENT BREAK</td></tr>
<tr><td>10:30 -
11:30 am</td><td colspan="2">DEARMOND LECTURE
Decoding Alzheimer's Disease Gene by Gene
Rudy Tanzi, PhD
Massachusetts General Hospital, MA</td></tr>
<tr><td>11:45 -
12:45 pm</td><td colspan="2">BUSINESS MEETING II
Texas Ballroom B</td></tr>
<tr><td>12:45 -
2:00pm</td><td colspan="2">LUNCH</td></tr>
<tr><td></td><td>Texas Ballroom A</td><td>Texas Ballroom B</td></tr>
<tr><td>2:00 -
4:00 pm</td><td>Platform 7
Neuropathology of Cells
And Cell Populations

#154-158</td><td>Platform 8
Tumors III:
Pediatric Tumors

#159-163</td></tr>
<tr><td>4:00 -
4:30 pm</td><td colspan="2">REFRESHMENT BREAK</td></tr>
<tr><td>4:30 -
5:30 pm</td><td colspan="2">TEVA/PARISI LECTURE
Inflamation Induced Mitochondrial Injury:
A Major Mechanism of Neurodegeneration
Hans Lassman, PhD
University of Vienna, Vienna, Austria</td></tr>
<tr><td>8:00 -
11:00 pm</td><td colspan="2">DIAGNOSTIC SLIDE SESSION
Texas Ballroom B</td></tr>
</table>

SUNDAY June 14, 2009	
	Texas Ballroom A and B
8:00 am - 12:00 pm	*PRESIDENTIAL SYMPOSIUM* Oxidative Stress in Neurodegenerative Diseases

THURSDAY, June 11, 2009

SPECIAL COURSE
RECENT ADVANCES THAT IMPACT RESEARCH AND CLINICAL PRACTICE
Director: George Perry, PhD and Amyn M. Rojiani, MD, PhD
Texas Ballroom A & B

8:00 - 8:10 am	Introduction *Amyn M. Rojiani, MD,* *University South Floridia,Tampa, FL*
8:10 am	1st Step – Putting the "P" Back in Pathology *Jared N. Schwartz MD, PhD, FCAP* *President CAP* *Presbyterian Healthcare, Charlotte, NC*
9:00 am	The Role of Pathologists in the Era of Personalized Medicine *Eric E. Walk, MD* *Ventana Medical Systems*
9:50 - 10:20 am	*REFRESHMENT BREAK*
10:20 am	Methods in Morphometry – Clinical and Research Applications *Paul Jantzen, MD* *Media Cybernetics, Bethesda, MD*
11:10 am	Digitization of Pathology – No More Glass Slides at your Microscope? *Anthony Demetris, MD* *University of Pittsburgh, Pittsburgh, PA*
12:00 - 1:15 pm	*LUNCH*
1:15 pm	Training Future Neuropathologists: Where Are We Now – And Where May We Be Going? *Suzanne Z. Powell, MD,* *The Methodist Hospital, Houston, TX*
2:05 pm	Clinical and Research Directions for Muscle Biopsies: A Few Things New, A Few Things Old *Steven A. Moore, MD, PhD* *University of Iowa, Iowa City, IA*
2:55 -3:25 pm	*REFRESHMENT BREAK*
3:25 pm	Ophthalmic Pathology: A Look Through the Window to the World *Thomas J. Cummings, MD* *Duke University Medical Center, Durham, NC*
4:15 pm	When The Phone Rings *Doug Cameron, MD* *Armed Forces Institute of Pathology*
5:05 pm	Closing Remarks *George Perry, PhD* *University of Texas at San Antonio, San Antonio, TX*

Friday June 12, 2009
12:45 pm – 2:00 pm **TRAINEE LUNCHEON**

Research Careers – Opportunities and Challenges *Dennis Dickson, MD* *Mayo Clinic, Jacksonville, Florida*
The "Triple Threat" Career – Research/Clinical Service/Teaching *Daniel J. Brat, MD, PhD* *Emory University, Atlanta, GA*
AP/CP/Neuropath/Private Practice *Gary Pearl, MD, PhD* *Orlando Regional Medical Center, Orlando, FL*

SUNDAY, JUNE 14, 2009

PRESIDENTIAL SYMPOSIUM
OXIDATIVE STRESS IN NEURODEGENERATIVE DISEASES

Texas Ballroom A & B

8:00 – 8:10 am	Introduction *George Perry, PhD* *University of Texas, San Antonio, TX*
8:10 - 8:40 am	Bioenergetics in the Pathogenesis of Neurodegenerative Diseases *M. Flint Beal, MD* *Weill Medical College, New York, NY*
8:40 – 9:10 am	Free Radical Damage as a Therapeutic Target in Alzheimer's Disease *Thomas J. Montine, MD, PhD* *University Washington, Seattle, WA*
9:10-9:40	Abeta Amyloid Oligomers as Therapeutic and Diagnostic Targets for Alzheimer's Disease *Colin Masters, MD* *University Melbourne, Melbourne, Australia*
9:40 – 10:00 am	*AWARDS CEREMONY*
10:00 – 10:30 am	*REFRESHMENT BREAK*
10:30 - 11:15 am	***Matthew T. Moore Distinguished Lecture*** From Charcot to Lou Gehrig: Mechanisms and Treatment of ALS *Donald W. Cleveland, MD* *UCSD, San Diego, CA*
11:15-11:45 am	Causes and Consequences of Oxidative Damage in Alzheimer's Disease *Mark A. Smith, PhD* *Case Western Reserve University, Cleveland, OH*
11:45 -12:00 pm	*INSTALLATION OF NEW OFFICERS*
12:00 pm	*ADJOURNMENT*

FRIDAY, JUNE 12, 2009
Texas Ballroom A
8:00 am – 2:00 pm

Platform 1 Neurodegenerative I: Prions
Chairpersons: Wen-Quan Zou, MD, PhD, Case Western Reserve University
Francoise Gray, MD, PhD, INSERM, Paris

8:00- 8:15	1	NEURONAL EXPRESSION OF PHOSPHORYLATED RNA-DEPENDENT PROTEIN KINASE (PKR) IN CREUTZFELDT-JAKOB DISEASE (CJD) Claire Paquet, Stéphane Haik, Marc Polivka, Hugon Jacques, Francoise Gray
8:15- 8:30	2	EXPRESSION OF APOPTOSIS-RELATED PROTEINS DISTINGUISHES SPORADIC CREUTZFELDT-JAKOB DISEASE SUBTYPES Gabor Kovacs and Herbert Budka
8:30- 8:45	3	PATHOGENIC MUTATIONS IN THE PRION PROTEIN INCREASE SUSCEPTIBILITY TO OXIDATIVE STRESS IN NEURONAL CELLS Hyoung-gon Lee, Sandra Richardson, Xiongwei Zhu, Rudy Castellani, Mark Smith, George Perry, Robert Petersen
8:45- 9:00	4	THE CO-EXISTENCE OF PRPSC TYPE 1 AND 2 IN SPORADIC CREUTZFELDT-JAKOB DISEASE AFFECTS THE PHENOTYPE AND PRPSC CONFORMATION Ignazio Cali, Ivonne Cohen, Janis Blevins, Rudolph Castellani, Amer Al-Shekhlee, Jue Yuan, Piero Parchi, Jiri Safar, Wen-Quan Zou, Pierluigi Gambetti
9:00- 9:15	5	PROTEASE-SENSITIVE PRIONS IN THE FAMILIAL PRION DISEASE LINKED TO 144-BP INSERTION OF THE PRION PROTEIN GENE Wen-Quan Zou
9:15- 9:30	6	TAU PROTEIN IN A NOVEL PRION DISEASE WITH GSS FEATURES Gianfranco Puoti, Wenquan Zou, Qingzhong Kong, Fabrizio Tagliavini, Piero Parchi, Pierluigi Gambetti
9:30- 9:45	7	NOVEL HUMAN PRION DISEASE AFFECTING 3 PRION CODON 129 GENOTYPES: THE SPORADIC FORM OF GERSTMANN-STRÄUSSLER-SCHEINKER DISEASE? Pierluigi Gambetti, Gianfranco Puoti, Qingzhong Kong, Wenquan Zou, Fabrizio Tagliavini, Piero Parchi
9:45- 10:00	8	GERSTMANN-STRÄUSSLER-SHEINKER SYNDROME WITH VARIABLE PHENOTYPE AND THE PRP-P102L MUTATION IN ARGENTINA Miguel Riudavets, Horacio Martinetto, Michele Equestre, Maurizio Pocchiari, Naomi Arakaki, Marcelo Schultz, Eugenia Arias, Christián Begué, Gustavo Sevlever, Ana Lia Taratuto

10:00 - 10:30 am REFRESHMENT BREAK

10:30 – 11:30 am Saul R. Korey Lecture
Mechanisms of Neurodegeneration in Prion Diseases Originating from the Neuronal Plasma
Stephen J. DeArmond, MD, PhD, University California San Francisco, CA

11:45 am – 12:45 pm Business Meeting I

12:45 – 2:00 pm Lunch

FRIDAY, June 12, 2009
Texas Ballroom B
8:00 am 2:00 pm

Platform 2 Experimental and Developmental Neuropathology
Chairpersons: Kathryn McFadden, MD, University of Pittsburgh
Steven A. Moore, MD, PhD, University of Iowa

8:00- 8:15	9	EVIDENCE FOR A POSITIVE FGF8-BMP PATHWAY IN THE DORSAL TELENCEPHALIC MIDLINE Linda Doan, Anna Javier, Ira Blitz, Ken Cho, Edwin Monuki
8:15- 8:30	10	AUTOPSY CASE OF CAMPOMELIC DYSPLASIA AND SUBPIAL NEURONAL HETEROTOPIAS: POTENTIAL ROLE OF SOX9 IN CORTICAL NEURONAL MIGRATION Hilary Nickols, Flavia Nunes, Hernan Correa, Ty Abel
8:30- 8:45	11	DYSTROGLYCAN AND CEREBELLAR GRANULE NEURON DEVELOPMENT Huy Nguyen, Adam Ostendorf, Jakob Satz, Kevin Campbell, Steven Moore
8:45- 9:00	12	INHIBITION OF THE ACTIVIN RECEPTOR TYPE IIB PRODUCES TRANSIENT INCREASES IN STRENGTH IN MYOTUBULARIN DEFICIENT MICE Michael Lawlor, Ben Read, Matt Stein, Jennifer Lachey, Jas Seehra, Alan Beggs
9:00- 9:15	13	NOVEL LMNA MUTATION IN A NORTH AMERICAN FAMILY WITH LGMD1B AND DILATED CARDIOMYOPATHY Efrem Cox, Ben Darbro, Katherine Mathews, Peter Nagy, Barry Cabuay, Steven Moore
	14	**Withdrawn**
9:15- 9:30	15	HYPOXIA/ISCHEMIA-INDUCED REACTIVE GLIOSIS AND ATROPHY IN THE ROSTRAL FRONTAL CORTEX OF POSTNATAL RATS Cho Lwin, Kathleen Burke, Chad Agy, Michael McCaughan, Carol Miller, Jack Turman, Jr.
9:30- 9:45	16	IN VIVO MARKERS OF ENCEPHALITIS IN A MACAQUE MODEL OF HIV ENCEPHALITIS Sriram Venneti, Dafna Bonneh-Barkay, Guoji Wang, Stephanie Bissel, Brian Lopresti, Chester Mathis, Clayton Wiley

10:00 - 10:30 am REFRESHMENT BREAK

10:30 – 11:30 am Saul R. Korey Lecture
Mechanisms of Neurodegeneration in Prion Diseases Originating from the Neuronal Plasma
Stephen J. DeArmond, MD, PhD, University California San Francisco, CA

11:45 am – 12:45 pm Business Meeting I

12:45 – 2:00 pm Lunch

FRIDAY, JUNE 12, 2009
Texas Ballroom A
2:00 – 8:00 pm

Platform 3 Neurodegenerative II: α-Synuclein and Ubiquitin
Chairpersons: Irina Alafuzoff MD, PhD, Kuopio University
Rudy Castellani, MD, University of Maryland

2:00 – 2:15	17	REPRODUCIBILITY IN THE ASSESSMENT OF ALZHEIMER'S DISEASE AND LEWY BODY DISEASE-RELATED PATHOLOGIES: A STUDY BY BRAIN NET EURO Irina Alafuzoff, Safa Al-Sarraj, Thomas Arzberger, Nenad Bogdanovic, Herbert Budka, Charles Duyckaerts, Isidro Ferrer, Steven Gentleman, Giorgio Giaccone, Piero Parchi
2:15 -2:30	18	UNIFIED STAGING SYSTEM FOR LEWY BODY DISORDERS:CORRELATION WITH NIGRAL DEGENERATION AND COGNITIVE/MOTOR DYSFUNCTION Thomas Beach, Charles Adler, LihFen Lue, Lucia Sue, Reed Brooks, Jennifer Eschbacher, Charles White, Haru Akiyama, Holly Shill, Marwan Sabbagh, Douglas Walker
2:30 – 2:45	19	CD3 IN LEWY PATHOLOGY: DOES THE ABNORMAL RECALL OF NEURODEVELOPMENTAL PROCESSES UNDERLIE PARKINSON'S DISEASE Summer Nugent, Peggy Harris, Xiongwei Zhu, Shu Chen, Hyoung-Gon Lee, George Perry, Peter Oettgen, Mark Smith, Rudy Castellani
2:45- 3:00	20	THE RELATIONSHIP BETWEEN MITOCHONDRIA, OXIDATIVE STRESS, AND A-SYNUCLEIN TOXICITY IN PARKINSON DISEASE Pavan Auluck, Julie Su, Susan Lindquist
3:00 – 3:15	21	TDP-43 MICROVASCULOPATHY IN FRONTOTEMPORAL LOBAR DEGENERATION AND OTHER TDP-43 PROTEINOPATHIES Wenlang Lin, Monica Castenedes-Casey, Alex Kitto, Dennis Dickson
3:15- 3:30	22	ASSOCIATION OF A COMMON VARIANT IN GRN WITH TDP-43 IMMUNOREACTIVITY AND HIPPOCAMPAL SCLEROSIS IN NEURODEGENERATIVE DISORDERS Dennis Dickson, Matthew Baker, Rosa Rademakers
3:30 – 3:45	23	CLINICOPATHOLOGIC CHARACTERIZATION OF UBIQUITIN-POSITIVE, TDP-43-NEGATIVE FRONTOTEMPORAL LOBAR DEGENERATION Salvatore Spina, Rose Richardson, Jill Murrell, Pietro Pietrini, Eric Wassermann, Jordan Grafman, Bernardino Ghetti

4:00 - 4:30 pm ***REFRESHMENT BREAK***

4:30 – 5:30 pm ***Special Lecture***
Sixty Seven Years Of The JNEN: What I Learned in Sixteen Of Those Years – And Can It Benefit You?
Michael N. Hart, MD, PhD, University Wisconsin, WI

6:30 – 8:30 pm ***Annual Reception***
Executive Salon I

FRIDAY, June 12, 2009
Texas Ballroom B
2:00 – 8:00 pm

Platform 4 Tumors I: Gliomagenesis and Cell Physiology
Chairpersons: Daniel Brat, MD, PhD, Emory University
Werner Paulus, MD, University of Muenster

2:00 – 2:15	24	THE PTEN-REGULATING MICRORNA MIR-26A IS AMPLIFIED IN HIGH-GRADE GLIOMA AND FACILITATES GLIOMAGENESIS *IN VIVO* Jason Huse, Cameron Brennan, Dolores Hambardzumyan, John Pena, Sara Rouhanifard, Cherin Sohn-Lee, Carlos le Sage, Reuven Agami, Thomas Tuschl, Eric Holland
2:15 -2:30	25	SOX2 SILENCING IN GLIOMA CELL CULTURES SIGNIFICANTLY DECREASES CELL NUMBER Jennifer Eschbacher, Stephen Coons, Anna Joy, Burt Feuerstein
2:30 -2:45	26	THE ROLE OF DROSOPHILA BRAIN TUMOR (BRAT) HOMOLOGS IN REGULATING MYC EXPRESSION AND GLIOMA DIFFERENTIATION. Gang Chen, Fahmia Rahman, Yuan Rong, Carol Tucker-Burden, Constantinos Hadjipanayis, Erwin Van Meir, Daniel Brat
2:45 – 3:00	27	PDGFR SIGNALLING IN GLIOMAS ANALYZED USING A HIGH-THROUGHPUT IN VIVO SCREEN IN DROSOPHILA MELANOGASTER Werner Paulus, Astrid Jeibmann, Hanna Witte, Christian Klämbt
3:00 – 3:15	28	POSSIBLE TUMOR NEURAL STEM CELL ORIGIN FOR NEW ENDOTHELIAL CELLS IN GLIOBLASTOMAS Lucas Bradley, Douglas Miller, Norman Litofsky
3:15 – 3:30	29	IDH1 MUTATIONS IN GLIOMAS Hiroko Ohgaki, Takuya Watanabe, Sumihito Nobusawa, Paul Kleihues
3:30 – 3:45	30	MICRORNA EXPRESSION IN EARLY PASSAGE PRIMARY HIGH-GRADE GLIOMA CELL LINES AND POSSIBLE ROLE IN GLIOMA INVASION Paula Kinsella, Rachel Howley, Michael Farrell, Martin Clynes, Verena Amberger-Murphy
3:45 – 4:00	31	THE CELL CYCLE REGULATOR EMI1 IS HIGHLY OVEREXPRESSED IN EPENDYMOMAS, ANAPLASTIC ASTROCYTOMAS AND GLIOBLASTOMAS Norman Lehman, Nivedita Tiwari, Tom Mikkelsen

4:00 - 4:30 pm ***REFRESHMENT BREAK***

4:30 – 5:30 pm ***Special Lecture***
Sixty Seven Years Of The JNEN: What I Learned in Sixteen Of Those Years – And Can It Benefit You?
Michael N. Hart, MD, PhD, University Wisconsin, WI

6:30 – 8:30 pm ***Annual Reception***
Executive Salon I

Poster Session I

Neurodegenerative Diseases:

63	NEUROPATHOLOGICAL FINDINGS IN NON-AMNESTIC MILD COGNITIVE IMPAIRMENT Jennifer Molano, Joseph Parisi, Dennis Dickson, Kris Johnson, Bradley Boeve, David Knopman, Ronald Petersen
64	DISTRIBUTION OF NEURITIC AND DIFFUSE Aß PLAQUES IN BRAINS OF SUBJECTS WITH AD AND MCI: IMPLICATIONS FOR BRAIN IMAGING OF Aß Miguel Riudavets, Susan Resnick, Richard Obrien, Alan Zonderman, Yang An, Gustavo Sevlever, Gay Rudow, Olga Pletnikova, Juan Troncoso
65	LATE ONSET NEURODEGENERATION WITH BRAIN IRON ACCUMULATION WITH DIFFUSE NEUROFIBRILLARY TANGLES AND INCIDENTAL LEWY BODIES Carolyn Orr, Keith Josephs, Dennis Dickson
66	MAMMILLARY BODY ATROPHY IN THE DIFFERENTIAL DIAGNOSIS OF HIPPOCAMPAL ATROPHY Melissa Murray and Dennis Dickson
67	THE FREQUENCY OF APOE2 IS INCREASED IN ASYMPTOMATIC AD: THE NUN STUDY COHORT Diego Iacono
68	NEWLY DIAGNOSED ALZHEIMER'S DISEASE PATIENTS SHOW INCREASED DISSOCIATED Aß ANTIBODY LEVELS Mark Smith, Katarzyna Gustaw- Rothenberg, Sandra Siedlak, Hyoung-gon Lee, Rudy Castellani, Xiongwei Zhu, George Perry, Robert Petersen, Robert Friedland, Alan Lerner
69	HIGH-DEFINITION CHARACTERIZATION OF MICROVASCULAR ß-AMYLOID DEPOSITION IN ALZHEIMER'S DISEASE BRAINS Virawudh Soontornniyomkij, Cecilia Choi, Justine Pomakian, Harry Vinters
70	PATTERNS OF CORTICAL BETA SECRETASE-1 IN HUMAN CORTEX Robert Struble, Shari Beckman-Randall, Xiao-Xin Yan, Brian Moore
71	CYTOPLASMIC SEQUESTRATION OF SMAD 2/3 AFTER TAU HYPERPHOSPHORYLATION INDUCED BY OKADAIC ACID OR OLIGOMERIC Aß Shabnam Baig, Zoe van Helmond, Seth Love
72	ABETA PEPTIDE VACCINATION INDUCES AN INCREASE IN NEURONAL ACTIVATED PKR STAINING IN THE HIPPOCAMPUS OF PATIENTS WITH AD Claire Paquet, Delphine Boche, Francoise Gray, Jacques Hugon, James Nicoll
73	TEMPORAL CHANGES OF CSF GLIAL ACTIVATION BIOMARKERS FOLLOWING ACTIVE IMMUNIZATION AGAINST BETA-AMYLOID IN NON-HUMAN PRIMATES Julia Kofler, Chris Janssen, Russell Salter, Anita Trichel, Guoji Wang, Mark Stauffer, Clayton Wiley
74	INFLUENCE OF APOLIPOPROTEIN E GENOTYPE ON RAGE EXPRESSION IN ALZHEIMER'S DISEASE John Donahue, Amanda Duffy, Jane Lim, Miles Miller, Edward Stopa
75	HYDROXYNONENAL-GENERATED CROSSLINKING FLUOROPHORES ACCUMULATION IN ALZHEIMER DISEASE REVEALS A DICHOTOMY OF PROTEIN TURNOVER Justin Shenk, Rudy Castellani, Paula Moreira, Gjumrakch Aliev, Sandra Siedlak, Peggy Harris, Lawrence Sayre, Pamela Szweda, Luke Szweda, Mark Smith, George Perry

FRIDAY, June 12, 2009
Texas Ballroom C and Lobby
Poster Session I continued

Neurodegenerative Diseases Continued:

76	EP2-DEPENDENT DOCK2 EXPRESSION REGULATES MICROGLIAL RESPONSE P.J. Cimino and Thomas Montine
77	ELEVATED EXPRESSION OF CELL CYCLE PROTEINS IN VERY EARLY STAGES OF ALZHEIMER'S DISEASE Miguel Riudavets, Susan Resnick, Richard Obrien, Alan Zonderman, Yang An, Marcelo Schultz, Gay Rudow, Olga Pletnikova, Gustavo Sevlever, Juan Troncoso
78	OVEREXPRESSION OF MYC IN POST-MITOTIC NEURONS INDUCES CELL CYCLE RE-ENTRY AND NEURODEGENERATION Hyoung-gon Lee, Gemma Casadesus, Sandra Richardson, George Perry, Robert Petersen, Mark Smith
79	THE LOCUS CERULEUS CONTAINS MITOCHONDRIAL DNA DELETIONS IN ALZHEIMER'S DISEASE. Bradley Miller and James Bennett
80	EXPRESSION OF TAU ISOFORMS IN A TRIPLE TRANSGENIC AD MOUSE. SPECIFICITY OF SUBCELLULAR LOCALIZATION DURING DEVELOPMENT Jonathan Fratkin, Zelda He, Junming Wang
81	ARGYROPHILIC GRAIN DISEASE: PREVALENCE IN AN AUTOPSY SERIES AND LOCATION OF GRAINS IN EARLY TO ADVANCED DISEASE John Hedreen
82	LATERALITY IN ARGYROPHILIC GRAIN DISEASE (AGD) Tadashi Adachi, Yuko Saito, Hiroyuki Hatsuta, Sayaka Funabe, Shigeo Murayama
83	Withdrawn
84	CHOLINE ACETYL TRANSFERASE AND TYROSINE HYDROXYLASE IMMUNOREACTIVITY IN LEWY BODIES Brittany Dugger, Tae-Boem Ahn, Tanis Ferman, Dennis Dickson
85	FRONTOTEMPORAL LOBAR DEGENERATION - INTERMEDIATE FILAMENT (FTLD-IF): UNUSUAL CLINICAL-PATHOLOGICAL FINDINGS Tzvetan Kozarski, Howard Crystal, Jenny Libien
86	TUBULOFILAMENTOUS STRUCTURES IN TSE REPRESENT MORPHOLOGICAL MARKERS OF SPIROPLASMOSIS Frank Bastian, William Todd, Charles Boudreaux, Fred Enright
87	DENTATORUBRAL-PALLIDONIGRAL-LYUSIAL DEGENERATION WITH BASOPHILIC NEURONAL INCLUSIONS Keith Josephs, Wenlang Lin, Dennis Dickson
88	CLINICAL AND NEUROPATHOLOGIC ANALYSES OF ADULT NEURONAL CEROID LIPOFUSCINOSIS (KUFS DISEASE, CLN4A) Masaki Takao, Masaharu Hayashi, Kuniaki Tsuchiya, Eiji Ikeda, Ban Mihara, Atsuo Koto
89	ADULT POLYGLUCOSAN BODY DISEASE: AN UNDERDIAGNOSED CAUSE OF BOTH CENTRAL AND PERIPHERAL NERVOUS SYSTEM DISEASE Ana Lia Taratuto, Maria Sacolitti, Fernando Caceres, Graciela Sanchez Balcarcel, Ricardo Reisin, Martin Nogues, Alexander Lossos, Hasan Akman, Salvatore DiMauro
90	THE DORSAL ROOT GANGLION IN FRIEDREICH'S ATAXIA Arnulf Koeppen, Jennifer Morral, Ashley Davis, Mitchell Knutson, Zewu Chen, Danhong Li, Alexei Vershinin, Matthew Cusack, Jiang Qian, Simone Petrocine
91	THE NEUROPATHOLOGY OF BROWN-VIALETTO-VAN LAERE SYNDROME: A DETAILED CASE REPORT Hilary Nickols, Julianne Qualtieri, Marcia Wills, Hannah Kinney, Mark Becher

Neurodegenerative Diseases Continued:

92	OXIDATIVE AND MITOCHONDRIAL CHANGES IN FRAGILE X AND RELATED DISORDERS Xinglong Wang, Rudy Castellani, George Perry, Sandra Siedlak, Jeremy Stone, Mark Smith, Xiongwei Zhu

Others:

93	BRAIN PATHOLOGY IN A LARGE COHORT OF SOLVENT ABUSERS Marc Del Bigio and Zahra al-Hajri
94	INFLAMMATION AND DEMYELINATION IN PATIENTS WITH MULTIPLE SCLEROSIS AFTER ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANTATION Jian-Qiang Lu, Jeffrey Joseph, Anne Stevens, Jan Storek, Richard Nash, Luanne Metz, Arthur Clark, Edward Johnson, V. Wee Yong
95	BRAIN GENE ARRAY REVEALS CNS ADAPTATION AND TWO DIFFERENT KINDS OF HIV-ASSOCIATED NEUROCOGNITIVE DISORDER Benjamin Gelman, Tiansheng Chen, Joshua Lisinicchia, Jonathan Starkey, Susan Morgello, Eliezer Masliah, Deborah Commins, Elyse Singer, Igor Grant
96	TUMEFACTIVE MULTIPLE SCLEROSIS CONTINUES TO BE A DIAGNOSTIC AND THERAPEUTIC CHALLENGE Amyn Rojiani, Stanley J. Krolczyk, Reed Murtagh
97	MICROARRAY ANALYSIS OF CEREBELLAR RNA IN THE ATAXIC CHD6 KO MOUSE MODEL OF THE HUMAN SPINOCEREBELLAR ATAXIA SCAR6 Justin Kreuter, Melissa Lathrop, Lisa Chakrabarti, Jeremiah Eng, Sandra Warner, Jennifer Fields, Reinhard Stöger, C. Harker Rhodes, Steve Fiering
98	PRIMARY APLASIA/AGENESIS OF GRANULAR NEURONS: A CASE REPORT Veena Rajaram and Jeffrey Golden
99	"PONTINIZATION" OF THE MEDULLA: TWO CLINICAL CASE STUDIES Andrew Teich, E. Hedley-Whyte, James Goldman
100	PREMATURE GYRAL FORMATION AND POLYMICROGYRIA Steven Dubner, E. Bigio, P. Fitchev, S. Crawford, L. Rorke, L. Ernst, K. Matkowskyj, R. Hevner
101	EMBOLIC FOREIGN MATERIAL IN THE CENTRAL NERVOUS SYSTEM OF CHILDREN DYING WITH HEART DISEASE AND A HISTORY OF INSTRUMENTATION Mirna Lechpammer, Audrey Marshall, Amy Juraszek, Robert Padera, Sara Vargas, Rebecca Folkerth
102	INTRAVASCULAR POLYMER MATERIAL FOLLOWING INTERVENTIONAL PROCEDURES Rupal Mehta,Rashi Mehta, Michael Fishbein, Orestes Solis, Reza Jahan, Noriko Salamon, William Yong, Harry Vinters
103	PROGRANULIN EXPRESSION IN HIV-1-INFECTED CELLS IN BRAIN Leroy Sharer, Meilan Do, Mainul Hoque, Tsafi Pe'ery
104	PRIMARY ANGIITIS OF THE CNS (PACNS) WITH PREDOMINANT CRANIAL NEUROPATHY AND MYELOPATHY Orestes Solis, Rupal Mehta, Yvette Bordelon, Noriko Salamon, Harry Vinters
105	HYPOKALEMIC PERIODIC PARALYSIS: AN ATYPICAL PRESENTATION OF A RARE DISEASE – A CASE REPORT Josh Gioaccotto, Chunyu Cai, Kevin Felice, Margaret Grunnet, Charles Whitaker, Qian Wu

6:30 – 8:30 pm ***Annual Reception***
Executive Salon I

Saturday June 13, 2009
Texas Ballroom A
8:00 am – 2:00 pm

Platform 5 Neurodegeneration III: Plaques and Tangles
Chairpersons: Seth Love, PhD, University of Bristol
Ann McKee, MD, Beauont Hospital, Dublin

8:00- 8:15	32	CHRONIC TRAUMATIC ENCEPHALOPATHY IN PROFESSIONAL FOOTBALL PLAYERS Ann McKee, Robert Stern, Christopher Nowinski, Brandon Gavett, Robert Cantu
8:15- 8:30	33	NEUROFIBRILLARY TANGLES PLUS NO PLAQUES DOES NOT EQUAL ALZHEIMER'S DISEASE: A CLINICAL-PATHOLOGICAL STUDY Peter Nelson and William Markesbery
8:30- 8:45	34	TOPOGRAPHIC PROGRESSION OF τ HYPERPHOSPHORYLATION IN ALZHEIMER'S DISEASE Sozos Papasozomenos
8:45- 9:00	35	HEPATIC CERAMIDE MEDIATES BRAIN INSULIN RESISTANCE AND ALZHEIMER-TYPE NEURODEGENERATION IN OBESITY-TYPE 2 DIABETES MELLITUS Suzanne de la Monte, Lascelles Lyn-Cook, Margot Lawton, Elizabeth Silbermann, Ming Tong, Lisa Longato, Ping Jiao, Haiyan Xu
9:00- 9:15	36	ENDOTHELIN-CONVERTING ENZYME-2 IS ELEVATED IN ALZHEIMER'S DISEASE AND UPREGULATED BY Aß Jen Palmer, Shabnam Baig, Patrick Kehoe, Seth Love
9:15- 9:30	37	OVERPRODUCTION OF AMYLOID-BETA CAUSES ABNORMAL MITOCHONDRIAL DYNAMICS Xiongwei Zhu, Xinglong Wang, Bo Su, Hisashi Fujioka, Yang Wang, George Perry, Mark Smith
9:30- 9:45	38	A RECESSIVE MUTATION IN THE APP GENE WITH DOMINANT-NEGATIVE EFFECT ON AMYLOIDOGENESIS: A NEW PERSPECTIVE FOR AD THERAPEUTICS Guiseppe Di Fede, Marcella Catania, Michela Morbin, Marco Gobbi, Laura Colombo, Antonio Bastone, Laura Cantù, Efrat Levy, Mario Salmona, Fabrizio Tagliavini
9:45- 10:00	39	INCREASED NONSTEROIDAL ANTI-INFLAMMATORY DRUG EXPOSURE IS ASSOCIATED WITH INCREASED ALZHEIMER-TYPE NEUROPATHOLOGY Joshua Sonnen, Eric Larson, Rod Walker, Sebastien Haneuse, John Breitner, Thomas Montine

10:00 am – 10:30 am ***REFRESHMENT BREAK***

10:30 am – 11:30 am ***DeArmond Lecture***
Decoding Alzheimer's Disease Gene by Gene
Rudy Tanzi, PhD

11:45 am – 12:45 pm ***Business Meeting***

12:45 pm – 2:00 pm ***Lunch on your own***

Saturday June 13, 2009
Texas Ballroom B
8:00 am – 2:00 pm

Platform 6 Tumors II: Prognosis and Therapy
Chairpersons: David Louis, MD, Harvard
Francesca Brett MD, Beaumont Hospital, Dublin

8:00- 8:15	40	MSH6 SOMATIC MUTATIONS IN MEDIATING TEMOZOLOMIDE RESISTANCE IN RECURRENT GLIOBLASTOMAS Stephen Yip, Jiangyong Miao, Daniel Cahill, A. Iafrate, Kenneth Aldape, Catherine Nutt, David Louis
8:15- 8:30	41	HYPOXIA INCREASES CD133-PERCENTAGE AND CLONOGENICITY IN BRAIN TUMOR NEUROSPHERES Charles Eberhart, Alex Lin, Eli Bar
8:30- 8:45	42	PDGF DICTATES THE INVASIVE AND VASCULAR PROLIFERATIVE PHENOTYPE OF GLIOMA-LIKE TUMORS IN A NOVEL SPONTANEOUS MOUSE MODEL Karen SantaCruz, Stephen Wiesner, Stacy Decker, Colleen Forster, Brent Clark, John Ohlfest
8:45- 9:00	43	POLYSOMY FOR CHROMOSOMES 1 AND 19 PREDICTS EARLIER RECURRENCE IN ANAPLASTIC OLIGODENDROGLIOMAS WITH CONCURRENT 1P/19Q LOSS Matija Snuderl, April Eichler, Keith Ligon, Quynh Vu, Michael Silver, Rebecca Betensky, Azra Ligon, Patrick Wen, David Louis, A. Iafrate
9:00- 9:15	44	LOSS OF HETEROZYGOSITY AT 1P-19Q INDUCES A GLOBAL CHANGE IN OLIGODENDROGLIAL TUMOR GENE EXPRESSION Gustavo Sevlever, Ruben Ferrer-Luna, Manuel Mata, Lina Nuñez, Naomi Arakaki, Andres Cervio, Miguel Riudavets, Ana Lia Taratuto, Bernardo Celda, Horacio Martinetto
9:15- 9:30	45	INVESTIGATION OF SIGNALLING PATHWAYS IN HIGH GRADE GLIOMA Rachel Howley, Paula Kinsella, Francesca Brett, Verena Amberger-Murphy, Michael Farrell
9:30- 9:45	46	CO-EXPRESSION OF ATP CITRATE LYASE WITH ENOLASE 1 AMONG THE UP-REGULATED GLYCOLYTIC ENZYMES ASSOCIATED WITH POOR SURVIVAL Marie Beckner
9:45- 10:00	47	EXPRESSION OF NFATC1 IN PRIMARY CNS LYMPHOMAS IS DECREASED IN HIV-POSITIVE PATIENTS Konstantinos Linos, Suzanne Homan, Christine Sheehan, Alida Hayner-Buchan, Jeffrey Ross, Jiang Qian, Tipu Nazeer

10:00 am – 10:30 am ***REFRESHMENT BREAK***

10:30 am – 11:30 am ***DeArmond Lecture***
Decoding Alzheimer's Disease Gene by Gene
Rudy Tanzi, PhD

11:45 am – 12:45 pm ***Business Meeting***

12:45 pm – 2:00 pm ***Lunch on your own***

Saturday June 13, 2009
Texas Ballroom A
2:00 pm – 8:00 pm

Platform 7 Neuropathology of Cells and Cell Populations
Chairpersons: Harry Vinters, MD, UCLA
Hannah Kinney, MD, Harvard

2:00 – 2:15	48	SELECTIVE ASTROCYTE INJURY DISTINGUISHES EARLY LESIONS IN DEVIC'S DISEASE FROM MULTIPLE SCLEROSIS Boleslaw Lach
2:15 – 2:30	49	LARGE VACUOLES IN DORSAL ROOT GANGLION NEURONS FOLLOWING EXPOSURE OF RATS TO NEUROTOXIC ORGANOPHOSPHATES Bernard Jortner, Thomas Rogers-Cotrone, Melanie Burgess, Sandra Hancock, Marion Ehrich
2:30 – 2:45	50	A GESTATIONAL TRYPTOPHAN-FREE DIET DECREASES SEROTONERGIC NEURONS IN THE DORSAL RAPHE Guadalupe Flores-Cruz
2:45 – 3:00	51	NOVEL SEROTONERGIC ABNORMALITIES IN THE MEDULLA IN THE SUDDEN INFANT DEATH SYNDROME Hannah Kinney, Jhodie Duncan, David Paterson, Jill Hoffman, Natalia Borenstein, Henry Krous, Elizabeth Haas, David Mokler, Felicia Trachtenberg, Richard Belliveau
3:00 – 3:15	52	ISOLATION AND CHARACTERISATION OF AN UNDIFFERENTIATED BALLOON CELL FROM FOCAL CORTICAL DYSPLASIA Thomas Jacques, Shireena Yasin, Kate Latak, Anita Ganapathi, Francesca Becherini, Khadijah Miller, Oliver Campos, Helen Cross, William Harkness, Brian Harding
3:15 – 3:30	53	ACTIVATION OF IBA1+ MICROGLIA IN PATIENTS WITH RASMUSSEN'S ENCEPHALITIS Martin Wirenfeldt, Ryan Clare, Spencer Tung, Alexander Bottini, Gary Mathern, Harry Vinters
3:30 – 3:45	54	DIFFERENTIAL EXPRESSION OF TOLL-LIKE RECEPTORS IN ASTROCYTES IS ASSOCIATED WITH VIRAL NEUROTROPISM Ehud Lavi and Lin Cong

4:00 - 4:30 pm REFRESHMENT BREAK

4:30 – 5:30 pm ***Special Lecture***
Sixty Seven Years Of The JNEN: What I Learned in Sixteen Of Those Years – And Can It Benefit You?
Michael N. Hart, MD, PhD, University Wisconsin, WI

8:00 pm ***Diagnostic Slide Session***
Texas Ballroom B

Saturday June 13, 2009
Texas Ballroom B
2:00 pm – 8:00 pm

Platform 8 Tumors III: Pediatric Tumors
Chairpersons: Charles Eberhardt, MD, PhD, Johns Hopkins University
Ho Keung Ng, MD, Chinese University, Hong Kong

2:00 – 2:15	55	DIFFICULTIES IN CLASSIFYING PEDIATRIC CNS TUMORS Michael Johnson and Peter Burger
2:15 – 2:30	56	HISTOPATHOLOGICAL FACTORS IN RECURRENT PILOCYTIC ASTROCYTOMAS Matthew Karafin, Peter Burger, Patricia Goldthwaite, Charles Eberhart
2:30 – 2:45	57	MORPHOLOGIC AND MOLECULAR BIOMARKERS IN PILOCYTIC ASTROCYTOMAS Craig Horbinski, Ronald Hamilton, Ian Pollack
2:45 – 3:00	58	ACTIVATION OF THE MAPK PATHWAY: A SIGNATURE GENETIC DEFECT IN POSTERIOR FOSSA PILOCYTIC ASTROCYTOMAS David Ellison, Tim Forshew, Ruth Tatevossian, Jing Ma, Geoff Neale, William Ogunkolade, Jim Dalton, Simon Bailey, Tracy Chaplin, Amar Gajjar, Denise Sheer
3:00 – 3:15	59	MIRNA-124 IS FREQUENTLY DOWN-REGULATED IN MEDULLOBLASTOMA AND IS A NEGATIVE REGULATOR OF SLC16A1 Ho Keung Ng, Jesse Pang, Kay Kwok
3:15 – 3:30	60	ANAPLASTIC PROGRESSION IN NODULAR MEDULLOBLASTOMA: RELATIONSHIP WITH MYC AMPLIFICATION Marie Rivera-Zengotita, Monika Nunez, Vidya Mehta, Girard Courteau, Murali Chintagumpala, Robert Dauser, Adekunle Adesina
3:30 – 3:45	61	ATRT: IMPORTANCE OF BAF47 AND THERAPEUTIC UPDATE Dolly Aguilera, Stewart Goldman, Veena Rajaram
3:45 – 4:00	62	GENETIC ANALYSIS OF PEDIATRIC DIFFUSE INTRINSIC PONTINE GLIOMAS BY HIGH-RESOLUTION SINGLE NUCLEOTIDE POLYMORPHISM ARRAYS Cynthia Hawkins, Eric Lee, Pawel Buczkowicz, Eric Bouffet, Ute Bartels

4:00 - 4:30 pm ***REFRESHMENT BREAK***

4:30 – 5:30 pm ***Special Lecture***
Sixty Seven Years Of The JNEN: What I Learned in Sixteen Of Those Years – And Can It Benefit You?
Michael N. Hart, MD, PhD, University Wisconsin, WI

8:00 pm ***Diagnostic Slide Session***
Texas Ballroom B

SATURDAY June 13, 2009
Texas Ballroom C and Lobby

Poster Session II

Others:

106	A RETROSPECTIVE REVIEW OF MUSCLE BIOPSIES AT ORLANDO REGIONAL MEDICAL CENTER, 1995 - 2008 Aaron Wagner, Dana Altenburger, Orlando Gonzales, Gary Pearl
107	TOXIC MYOPATHY DUE TO A COMBINATION OF HYDROXYCHLOROQUINE AND COLCHICINE – A CASE REPORT Justin Kreuter, Timothy Lonesky, Robert Wortmann, C. Rhodes
108	NEMALINE MYOPATHY 5: A CASE REPORT. James Lapinski and Richard Prayson
109	REVIEW OF HISTOPATHOLOGY AND ULTRASTRUCTURAL CHARACTERISTICS IN TWO CASES OF TUBULAR AGGREGATE MYOPATHY Negar Khanlou, Michael Marvi, Anthony Lin, Brandon Hirota, Perry Shieh, Joseph Chung, Prasanth Manthena, Nazanin Matloubi, Rani Gowrinathan, Harry Vinters
110	PEDIATRIC MACROPHAGIC MYOFASCIITIS IN A PATIENT WITH ATRX SYNDROME, WITHOUT EVIDENCE OF ALUMINUM INCLUSIONS: A CASE REPORT Howard Chang, I. Dyme, Robert Conway, Michael Netzloff
111	REVERSIBLE MYOPATHY INDUCED BY LONG-TERM PEGASYS MONO TREATMENT FOR A CHRONIC HEPATITIS C PATIENT UNDER HALT-C TRIAL Chunyu Cai, Josh Gioaccotto, Kevin Felice, Margaret Grunnet, Charles Whitaker, Qian Wu

Tumors:

112	THE UCLA BRAIN TUMOR TRANSLATIONAL RESOURCE (BTTR): BIOREPOSITORY APPROACHES FOR FACILITATING TRANSLATIONAL RESEARCH William Yong, Sergey Mareninov, Jason De Jesus, Negar Khanlou, Rupal Mehta, Haijing Zhang, Alina Leung, Derrek Hibar, Zhi Wu, Harry Vinters, Timothy Cloughesy
113	RAPID IMUNOHISTOCHEMISTICAL STAINING OF PARAFFIN AND FROZEN BRAIN TUMOR SAMPLES USING THE CELERUS WAVE IMMUNOSTAINER William Yong, Jason De Jesus, Sergey Mareninov, Veeraou Konkankit, Rupal Mehta, Negar Khanlou, Haijing Zhang, Bob Shafa, Linda Liau, Marvin Bergsneider, Harry Vinters
114	DIGITAL IMAGE ANALYSIS OF SIGNAL PROFILING IN HIGH GRADE GLIOMAS Rachel Howley, Paula Kinsella, Francesca Brett, Verena Amberger-Murphy, Michael Farrell
115	MISMATCH REPAIR DEFECTS IN HUMAN GLIOBLASTOMAS POST-ALKYLATOR TREATMENT Yeowon Kim, Stephen Yip, Eudocia Quant, Gayatry Mohapatra, David Louis
116	GLIOBLASTOMA WITH SIGNET-RING MORPHOLOGY: A CASE REPORT AND REVIEW OF THE LITERATURE Sarah Martin, Jose Bonnin, David Hall, Eyas Hattab
117	PRIMARY GLIOSARCOMA WITH EPENDYMAL DIFFERENTIATION Oleksandr Kryvenko, Thomas Christopherson, Jon Wilson, Norman Lehman
118	CONGENITAL/PEDIATRIC GLIOBLASTOMA MULTIFORME OR PRIMITIVE NEUROECTODERMAL TUMOR? Veena Rajaram, Dolly Aguilera, Stewart Goldman
119	WT1 DOES NOT RELIABLY DISTINGUISH REACTIVE FROM NEOPLASTIC ASTROCYTES T. Bourne

Tumors Continued:

120	CASE REPORT-SUBARACHNOID EN PLAQUE GLIAL PROLIFERATION OVERLYING REMOTE CYSTIC LESION: NEOPLASTIC OR REACTIVE? Susan Staugaitis, Richard Prayson, Stephen Hantus
121	SYNCHRONOUS CEREBELLAR MEDULLOBLASTOMA AND JUVENILE PILOCYTIC ASTROCYTOMA: REPORT OF A RARE OCCURRENCE Abir Mukherjee, Andrew Jea, Meena Bhattacharjee
122	NF1-ASSOCIATED LOW GRADE GLIOMAS: AN ULTRASTRUCTURAL STUDY Mark Jentoft, Caterina Giannini, Bernd Scheithauer, Patrice Abell-Aleff, Fausto Rodriguez
123	COMPARATIVE CHARACTERIZATION OF DIFFUSE GLIOMAS BY FISH FOR 1p AND 19q Suash Sharma and Richard Hessler
124	OLIGODENDROGLIAL TUMOR CLASSIFICATION: PHENOTYPE VS GENETIC SIGNATURE Gustavo Sevlever, Eugenia Arias, Andres Cervio, Ruben Ferrer-Luna, Lina Nuñez, Bernardo Celda, Manuel Mata, Naomi Arakaki, Miguel Riudavets, Ana Lia Taratuto, Horacio Martinetto
125	IMMUNOHISTOCHEMICAL DIFFERENTIATION OF SUBEPENDYMOMA, EPENDYMOMA, AND GANGLIOGLIOMA Joseph Fullmer, Born Don, Jing Zhang
126	CEREBRAL MYXOPAPILLARY EPENDYMOMA Mary Jo Martin, Christine Fuller, Nitya Ghatak
127	CORTICAL EPENDYMOMA: A DISTINCT ENTITY FROM ANGIOCENTRIC GLIOMA? Caterina Giannini, Federico Roncaroli, Jamie VanGompel, Richard Marsh, Gregory Cascino, Peter Burger
128	ANGIOCENTRIC GLIOMA IN THE TECTAL REGION Douglas Miller, Eric Johannesen, Norman Litofsky, Faris Fakhoury
129	EPITHELIOID MALIGNANT GLIONEURONAL TUMOR: A DIAGNOSTIC CONUNDRUM IN AN ADULT PATIENT WITH HISTORY OF CARCINOMA Christine Fuller and William Broaddus
130	PAPILLARY TUMOR OF THE PINEAL REGION WITH MARKED NUCLEAR PLEOMORPHISM, NON-PAPILLARY PATTERN, AND FOCAL ONCOCYTIC FEATURES Monika Wrzolek, Tzvetan Kozarski, Anthony Alastra, Elaina Mastrangelo, Marc Rosenblum
131	THE ROLE OF STAINING INTENSITY IN THE EVALUATION OF PROLIFERATION INDEX(PI) BY KI67 IMMUNOHISTOCHEMISTRY IN MENINGIOMAS(MEN) Murat Gokden
132	ONCOCYTIC MENINGIOMA: FEATURES SUGGESTIVE OF CHORDOID VARIANT Roy Rhodes
133	RHABDOID MENINGIOMA ARISING IN A PATIENT WITH PREVIOUSLY IRRADIATED COMPOSITE PXA/OLIGODENDROGLIOMA Sarah Martin, Jose Bonnin, Scott Shapiro, Khaled Hamada, Annette Douglas-Akinwande, Eyas Hattab
134	INTRACRANIAL PHOSPHATURIC MESENCHYMAL TUMORS Jack Raisanen, Kimmo Hatanpaa, Joseph Beshay, Emily Herndon, Christa Hladik, Bruce Mickey, Dennis Burns, Charles White

SATURDAY, June 13, 2009
Texas Ballroom C and Lobby
Poster Session II continued

Tumors Continued:

135	PHOSPHATURIC MESENCHYMAL TUMOR OF THE THORACIC SPINE Sarah Martin, Narasimhan Agaram, Jey-Hsin Chen, Eric Horn, Annette Douglas-Akinwande, Eyas Hattab
136	A RARE CASE OF PEDIATRIC ECTOMESENCHYMOMA ARISING FROM THE FALX Dana Altenburger and Gary Pearl
137	INTRACEREBRAL EXTRAVASCULAR MASSON'S TUMOR MIMICKING GLIOMA Mika Fujiwara, Griffith Harsh, Jason Karamchandani, Iris Gibbs, Nancy Fischbein, Hannes Vogel
138	ATYPICAL TERATOID/RHABDOID TUMOR OF THE PINEAL REGION IN AN ADULT: A CASE REPORT Hidehiro Takei, Adekunle Adesina, Suzanne Powell, Lauren Langford
139	A POSTERIOR FOSSA EMBRYONAL TUMOR IN A 4-YEAR-OLD MALE WITH FEATURES OF ATRT BUT WITHOUT INI1 MUTATION Aaron Wagner, Anthony Yachnis, Suzanne Powell, Adekunle Adesina, Gary Pearl
140	MICRORNA PROFILING OF PRIMARY CNS HEMANGIOBLASTOMAS AND METASTATIC CLEAR CELL RENAL CELL CARCINOMAS TO THE BRAIN Sriram Venneti, Aihua Liu, John Tobias, Donald Baldwin, Priti Lal
141	CLEAR CELL RENAL CELL CARCINOMA METASTATIC TO CAPILLARY HEMANGIOBLASTOMA IN THE SETTING OF VON HIPPEL-LINDAU Sarah Martin, Sohaib Al-Khatib, Annette Douglas-Akinwande, Michael Turner, Eyas Hattab
142	p63 IS A SENSITIVE MARKER OF SKELETAL MUSCLE DIFFERENTIATION: A CASE REPORT OF MEDULLOMYOBLASTOMA Sarah Martin, Matthew Kuhar, Jodi Smith, Annette Douglas-Akinwande, Eyas Hattab
143	CYCLIN D1 IMMUNOEXPRESSION IN ADENOHYPOPHYSIAL TUMORS B. Scheithauer, D. Soni, K. Kovacs, R.V. Lloyd, U. DiGirolami, J. Fletcher, V. Nose
144	TRANSDIFFERENTIATION OF PITUITARY THYROTROPHS TO "LACTOTHYROTROPHS" IN PRIMARY HYPOTHYROIDISM: CASE REPORT Mark Jentoft, Bernd Scheithauer, Kalman Kovacs
145	CNS IMMUNODEFICIENCY-ASSOCIATED LYMPHOPROLIFERATIVE DISORDERS: CLINICOPATHOLOGIC AND CYTOGENETIC FEATURES Kliment Donev, Ahmet Dogan, Mark Law, Brian O'Neill, Caterina Giannini
146	PRIMARY T-CELL LYMPHOMA OF THE CNS EXPRESSING PD-1, A MARKER OF GERMINAL CENTER T-CELLS Jason Karamchandani, Gregory Moes, Kern Guppy, Hannes Vogel
147	LYMPHOMATOSIS CEREBRI, A TREACHEROUS MIMIC AT THE CLINICAL & PATHOLOGICAL LEVEL; REPORT OF TWO BIOPSIED AND AUTOPSIED CASES Pieter Wesseling, Saskia Zomer, Annemieke Smeets, Sander Kok, Albert Twijnstra, Christian Mawrin, Wim Spliet, Johan Kros, Konnie Hebeda
148	PRIMARY CENTRAL NERVOUS SYSTEM LYMPHOMATOID GRANULOMATOSIS: TWO CASES AND REVIEW OF LITERATURE Abir Mukherjee, Youli Zu, Jeff Chang, Hidehiro Takei, Suzanne Powell
149	CHOROID PLEXUS PLASMA CELL GRANULOMA: REPORT OF 2 CASES Li Li, Ravi Gandhi, Yu-Hung Kuo, Tipu Nazeer, Jiang Qian

Tumors Continued:

150	HEPATOID YOLK-SAC TUMOR OF THE CERVICAL SPINE: CASE REPORT AND REVIEW OF THE LITERATURE Tracie Pham, Jennifer Eschbacher, Stephen Coons
151	GERMINOMA OF CORPUS CALLOSUM OVERSHADOWED BY CASEATING GRANULOMA Laura Kidd, Gregory Fuller, Min Wang
152	LIPOMATOUS NEUROFIBROMA OF THE SPINAL CORD Kenneth Fallon, Warren Boling, Kymberly Gyure
153	VALUE OF CYTOKERATINS 7 AND 20 IN EVALUATING DEVELOPMENTAL CYSTS WITH UNUSUAL PRESENTING FEATURES Kathy Newell and Bette Kleinschmidt-DeMasters
154	UTILITY OF THE PATHCHIP™ TISSUE OF ORIGIN TEST FOR CHARACTERIZATION OF A METASTATIC CARCINOMA OF UNKNOWN PRIMARY Christine Fuller, Gary Tye, Catherine Dumur
155	TUMOR-INHIBITORY ROLE OF P75NTR AND TIMP1 INTERACTIONS IN METASTATIC CARCINOMA AND GLIOMA Amyn Rojiani, Steven Brem, Mumtaz Rojiani
156	CASE OF ADENOVIRUS ENCEPHALITIS PRESENTING AS A MASS LESION Matthew Schniederjan and Daniel Brat
157	HISTOPATHOLOGY OF AN “UNIDENTIFIED BRIGHT OBJECT” IN THE BRAIN OF AN ADULT PATIENT WITH NEUROFIBROMATOSIS TYPE 1 Douglas Miller, Michael Muzinich, M. Tacazon, Norman Litofsky

4:30 – 5:30 pm ***Special Lecture***
Sixty Seven Years Of The JNEN: What I Learned in Sixteen Of Those Years – And Can It Benefit You?
Michael N. Hart, MD, PhD, University Wisconsin, WI

8:00 pm ***Diagnostic Slide Session***
Texas Ballroom B

American Association of Neuropathologists

Endowed Lectureships

Meritorious Awards

Presidential Symposium

The Saul R. Korey Lectureship–a brief history

The *Korey Lectureship* 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 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.

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 Dr. Stephen J. DeArmond 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 deF 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 Orgininating from the Neuronal Plasma Membrane

2009 SAUL R. KOREY LECTURE

Mechanisns of Neurodegeneration in Prion Diseases Originating from the Neuronal Plasma Membrane

Stephen J. DeArmond, MD, PhD

Stephen J. DeArmond, MD, PhD

Dr. Stephen J. DeArmond is a professor of Pathology and Neuropathology at the University of California San Francisco, where he directs the DeArmond Neuropathology Research Laboratory in the Department of Pathology and Institute for Neurodegenerative Diseases. Dr. DeArmond has collaborated over 24 years with Dr. Stanley Prusiner on prion diseases. His main contribution has been on the molecular pathogenesis of those diseases. His lab was the first to describe that protease-resistant PrP^{Sc} is a major component of amyloid plaques in animal and human prion diseases and that the non-plaque PrP^{Sc} localizes precisely with sites vacuolization and reactive astrocytic gliosis. Western blot and histoblot analysis showed the kinetics of the PrP^{Sc} in a quantitative manner. Later the kinetics of PrP^{Sc} was followed in sub-cellular domains. Numerous neuropathology and post-doctoral fellows working in his laboratory showed that the accumulation of PrP^{Sc} in cell membranes causes many of the abnormalities attributed to scrapie and Creutzfeldt-Jakob disease. The latest data emphasizes the treatment with a gamma-secretase inhibitor and quinacrine stops the progression of the disease. Dr. DeArmond was a member of the FDA Transmissible Spongiform Encephalopathy Advisory Committee. He has written chapters on prion diseases in the 6th and 7th editions of Greenfield's Neuropathology as well as in numerous other textbooks. He also has an active clinical role at UCSF Neuropathology in the diagnosis of Nerve and Muscle diseases, neurosurgical frozen sections, and the autopsy service.

Abstract:

About 80% of PrP^{Sc} accumulates in the plasma membranes and about 15% in endosomes and lysosomes of neurons during the common forms of human and animal prion diseases such as Creutzfeldt-Jakob disease in humans and scrapie in animals. About 85% of PrP^{Sc} in plasma membranes is in cholesterol rich-membrane rafts. The objective here is to present the evidence that membrane PrP^{Sc} causes early dysfunction and degeneration of multiple critical functions attributed to membranes. We believe that nerve cell death is a late event attributable to accumulation of PrP^{Sc} in lysosomal compartments. Patient death does occur because of the early insults and does not require massive nerve cell loss. Here I will present the highlights of research in my Neuropathology Research Laboratory for the last 24 years. This will include the following:

Objectives:

1. Measurement of PrP^{Sc} by a Conformation Dependent Immunoassay that shows its exponential accumulation in subcellular fractions and shows how these measurements relate to Western blot and histoblot measurements.
2. Determining the temporal sequence of PrP^{Sc} in subcellular fractions shows how PrP^{Sc} spreads in individual neurons.
3. The mechanism of spread of disease in brain is found to be by axonal transport of PrP^{Sc} from one brain region to another.

4. PrP^{Sc} changes the physical properties of plasma membranes explains the spontaneous membrane rupture identified by Peter Lampert and Nick Gonatas in CJD and kuru.
5. Transynaptic spread of PrP^{Sc} formation will be discussed.
6. PrP^{Sc} in synapses has a profound effect on synaptic transmission.
7. Dendritic degeneration is the first morphological seen in prion disease. Activation of Notch-1, which is known to have a repressive effect on dendrites, links PrP^{Sc} accumulation in membranes to early occurring dendritic degeneration.
8. Treatment of scrapie infected mice with a γ-secretase inhibitor plus quinacrine prevented dendritic degeneration and prevented formation of PrP^{Sc}, which supports the hypothesis that Notch-1 signaling was the cause of dendritic degeneration.

Disclosures: Dr. DeArmond has nothing to disclose.

The DeArmond Lecture

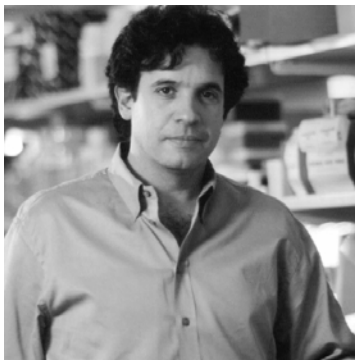
The DeArmond lecture was established in recognition of Stephen J. DeArmond's excellent leadership and organization of the scientific program for the 2006 International Congress of Neuropathology. This successful meeting garnered significant support intended for the future advancement of the mission of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

We are pleased to have Dr. Rudy Tanzi 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

2009 DE ARMOND LECTURE

Decoding Alzheimer's Disease Gene by Gene
Rudy Tanzi, PhD



Rudy Tanzi, PhD

Dr. Rudolph Tanzi is a Professor of Neurology and holder of the Joseph P. and Rose F. Kennedy Endowed Chair in Neurology at Harvard University. At Massachusetts General Hospital (MGH), Dr. Tanzi serves as the Director of the Genetics and Aging Research Unit, which consists of eight laboratories investigating the genetic causes of Alzheimer's disease. Dr. Tanzi has been investigating the molecular and genetic basis of neurological disease since 1980 when he participated in the pioneering study that led to location of the Huntington's disease gene, the first disease gene to be found by genetic linkage analysis. Since 1982, Dr. Tanzi has investigated the genetic causes of Alzheimer's disease (AD). He co-discovered all three genes that cause early-onset familial AD, including the first familial AD gene, known as the amyloid β-protein (A4) precursor (APP), and the presenilin genes. In 1993, Dr. Tanzi discovered the gene responsible for the neurological disorder known as Wilson's disease, and over the past 25 years, he has collaborated on studies identifying several other disease genes including those causing amyotrophic lateral sclerosis and autism. Dr. Tanzi currently spearheads the Alzheimer's Genome Project, which recently identified four new AD gene candidates. This achievement was named one of the "Top Ten Medical Breakthroughs of 2008" by Time Magazine. In 1994, Dr. Tanzi discovered that the metals, zinc and copper, are necessary for the formation of neurotoxic assemblies of the Aβ peptide, the main component of β-amyloid deposits in brains of AD patients. Based on this discovery, Dr. Tanzi developed the "Metal hypothesis of Alzheimer's disease", which has led to successful clinical trials for treating and preventing AD by targeting Aβ−metal interactions. These trials were carried out by Prana Biotechnology, LTD, for which Dr. Tanzi served as a co-founder. Dr. Tanzi is one of the ten most cited researchers in AD, having co-authored over 340 research articles. He is also a co-author of a popular trade book on Alzheimer's disease entitled "Decoding Darkness: The Search for the Genetic Causes of Alzheimer's Disease". Dr. Tanzi has received several awards for his work including the two highest awards for Alzheimer's disease research: The Metropolitan Life Foundation Award and The Potamkin Prize. He has also received the Reagan National Alzheimer's Disease Research Award, an NIH MERIT Award, and the "Oneness of Humanity" Global Award, and is an AAAS Fellow. In 2007, he was included on the list of the "Harvard 100: Most Influential Alumni" of over 220,000 living alumni. His invited honorary lectures include a Nobel Forum Lecture, Smithsonian Institution Distinguished Lecture, and the Society for Neuroscience Public Lecture. Dr. Tanzi is the Chairman of the Cure Alzheimer's Fund Research Consortium and serves over 40 editorial and scientific advisory boards.

Abstract:

Alzheimer's disease (AD) is inherited in up to 80% of cases. Over the past two decades, we have co-discovered three genes, *APP, PSEN1,* and *PSEN2*, which can carry >200 mutations causing early-onset familial AD. The ε4 variant of *APOE* is the only established risk factor for late-onset AD. Meanwhile, >50% of the genetic variance of AD remains unexplained by the four known AD genes. More than 550 candidate genes and >1500 DNA variants have been tested for

association with leading to major confusion regarding which are the leading AD gene candidates. To address this problem, we have created a website, AlzGene.org, which contains data from studies of >500 AD candidate genes (>1500 variants). For variants tested in at least four independent samples, AlzGene provides meta-analyses to determine the most promising AD candidate genes. These studies have led to ~30 candidate AD genes with statistical significance beyond *APOE*. To identify the remaining late-onset AD genes in an unbiased manner, we have completed a genome-wide association screen of >400 late-onset AD families testing 500,000 single nucleotide polymorphisms (SNP). We have reported four novel loci that achieved genome-wide significance (besides *APOE*) including a novel gene on chromosome 14q, GWA-14q, the ataxin 1 gene, the innate immune system gene, *CD33*, and the synaptic gene, *DLGAP1*. With regard to *CD33*, new data implicating Aβ as an anti-microbrial peptide in the innate immune system will also be presented.

Objectives:

1. Understand how candidate gene analysis is used to identify genetic contributions to Alzheimer disease.
2. Understand genetics of Alzheimer Disease.
3. Understand role of late onset genes in the pathogenesis of Alzheimer disease.

Disclosures: Dr. Tanzi is a consultant for Eisai, Siemens, Pathway Genomics, Neuroptix, Prana and Smart and a stockholder in Torreypine Terapeutics, Prana, Pathway Genomicws, Neuroptix and Smart

The Teva Neuroscience/Parisi Lecture

The *Teva Neuroscience/Parisi Lecture* was established with a generous endowment from Teva Pharmaceuticals in 2007. Teva Neuroscience, a subsidiary of Teva Pharmaceuticals, is devoted to the study and development of products and services that address the health management needs of people in the field of neurology. One of the focal points of their efforts is multiple sclerosis.

The lecture was named the Teva Neuroscience/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 Dr. Hans Lassmann 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

2009 PARISI/TEVA PHARMACEUTICAL LECTURE
Inflammation Induced Mitochondrial Injury: A Major Mechanism of Neurodegeneration
Hans Lassmann, PhD

Hans Lassmann, PhD

Inflammation in the central nervous system in the course of infections or autoimmune diseases leads to damage of neurons and glia. Such tissue injury can be induced in an antigen-specific way by cytotoxic T-lymphocytes or autoantibodies or in a bystander reaction by toxic products of activated effector cells, such as macrophages, microglia or granulocytes. Macrophage mediated tissue injury may involve cytotoxic cytokines, proteo- and lipolytic enzymes, reactive oxygen or nitrogen intermediates or even excitotoxins. Recently a new mechanism of bystander tissue injury became apparent, which involves damage to and dysfunction of mitochondria. Thus, in conditions of very severe and fulminate inflammation hypoxia-like tissue injury is seen, which in the white matter is reflected by distal oligodendrogliopathy, caspase independent oligodendrocyte apoptosis and expression of molecules, involved in tissue preconditioning. Tissue injury is associated with loss and functional inactivation of complex IV of the mitochondrial respiratory chain. Besides oligodendrocytes, small calibre axons are preferentially destroyed. In axons, loss of mitochondrial function leads to an imbalance of ion-homeostasis, finally resulting in Ca^{++} overload and the activation of Ca^{++} dependent proteases. Microarray studies suggest that pro-inflammatory cytokines may directly or indirectly induce mitochondrial dysfunction. Nitric oxide radicals may impair the function and stability of COX-1, the heme-containing subunit of complex IV of the respiratory chain and is, thus, one possible candidate inducing cellular energy failure. This pathway of tissue injury is seen in various different inflammatory brain diseases and seems to be particularly prominent in a subset of multiple sclerosis lesions.

Objectives: At the end of the lecture, attendees should be able to:

1. Severe brain inflammation may lead to a pattern of tissue injury, which is closely similar to hypoxic brain damage seen in white matter stroke lesions. This is seen in subsets of patients with very severe active multiple sclerosis or virus induced encephalomyelitis.
2. Initial lesion in white matter hypoxia show characteristic neuropathological alterations: distal (dying back) oligodendrogliopathy with selective loss of distal myelin proteins (myelin associated glycoprotein and cyclic nucleotide phosphodiesterase), caspase 3 independent apoptosis of oligodendrocytes and the expression of molecules, involved in hypoxic preconditioning. These alterations are associated with preferential destruction of oligodendrocytes or thin axons and partial preservation of perivascular tissue components.
3. In inflammatory conditions such hypoxia-like lesions are associated with profound microglia activation, selective loss of COX-1, the heme-containing protein of Complex IV of the respiratory chain, and reduction of Complex IV activity.
4. In axons, loss of mitochondrial function leads to an imbalance of ion-homeostasis, finally resulting in non-physiological intra-axonal calcium accumulation.
5. In inactive lesions an increase in total mitochondria and activity of the respiratory chain is seen, possibly reflecting a compensatory mechanism.

6. Microarray studies suggest that pro-inflammatory cytokines may induce mitochondrial dysfunction. Nitric oxide radicals may impair the function and stability of COX-1 and are, thus, one possible candidate.
7. In less severe inflammatory conditions, which do not lead to the complete picture of hypoxia-like tissue injury, similar alterations of mitochondria have been observed.
8. Conclusions: Mitochondrial injury induced by inflammatory mediators, appears to be a major driving force for tissue injury in acute and chronic inflammatory diseases of the nervous system.

Disclosures: Dr. Lassmann is a consultant for Merck Serono and Bayer Schering

Awards for Meritorious Contributions to Neuropathology

The *Award for Meritorious Contributions to Neuropathology* recognizes a member who has made significant contributions to the advancement of knowledge in neuropathology and provided service to the American Association of Neuropathologists. Each recipient of the award is nominated by the president, in conjunction with the nominating committee and with the approval of the executive council.

The qualities of outstanding scientific achievement and service are embodied in this year's recipients, Drs. Peter C. Burger, Pierluigi Gambetti and Nicholas K. Gonatas. They join the rich roster of distinguished former award recipients.

Year	Recipient
1959	Armando Ferraro
	Arthur Weil
1960	Joseph H. Globus
	George B. Hassin
1968	Abner Wolf
	Paul I. Yakovlev
	Harry M. Zimmerman
1970	Webb E. Haymaker
1971	James W. Kernohan
1972	George A. Jervis
1979	Raymond D. Adams
	David Cowen
	Matthew T. Moore
1981	Richard Lindenberg
1983	Orville T. Bailey
1984	Margaret Murray
1985	Kenneth M. Earle
	Nathan Malamud
	Leon Roizin
1986	Martin G. Netsky
1987	*No Award Presented*
1988	Edward P. Richardson, Jr.
	F. Stephen Vogel
1989	Lucien J. Rubinstein
	Robert D. Terry
1991	Lysia K. S. Forno
1992	John Moossy
	Gabriele M. ZuRhein
1993	Peter W. Lampert
	Elias E. Manuelidis
1994	Murray B. Bornstein
	Samuel P. Hicks
	Lowell W. Lapham
1995	Amico Bignami
	Asao Hirano
1996	Pasquale A. Cancilla
	Franz Seitelberger
1997	Henryk M. Wisniewski
1998	Richard L. Davis
	Wolfgang Zeman
1999	Lucy B. Rorke
2000	William R. Markesbery
2001	John J. Kepes
	Henry de Forest Webster
2002	Dikran S. Horoupian
	Fusahiro Ikuta
	Kurt A. Jellinger
2003	Bernardino F. Ghetti
2004	Michael N. Hart
2005	E. Tessa Hedley-Whyte
	Suzanne S. Mirra
2006	Joseph E. Parisi
	Jeannette J. Townsend
2007	James M. Powers
	Cedric S. Raine
2008	Kinuko Suzuki
	Margaret G. Norman
2009	Peter C. Burger
	Pierluigi Gambetti
	Nicholas K. Gonatas

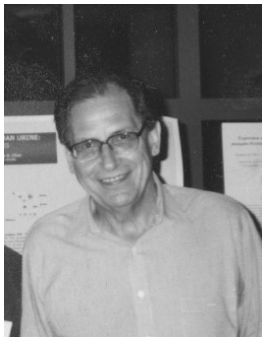
Awards for Meritorious Contributions to Neuropathology

2009 AWARD RECIPIENTS

Peter C. Burger, MD; Pierluigi Gambetti, MD and Nicholas K. Gonatas, MD

Dr. Peter C. Burger, MD

Dr. Peter C. Burger is Professor of Pathology, Oncology, and Neurosurgery at the Johns Hopkins University School of Medicine. He received his M.D. degree from Northwestern University Medical School in 1966, did a rotating internship at Los Angeles County Hospital, moved to Duke University Medical Center for his training in anatomic and neuropathology, and joined the Duke faculty in 1973. After 20 very productive years there, he moved to Johns Hopkins in 1993. Dr. Burger is internationally known for his contributions to the field of adult and pediatric brain tumors. His diagnostic skills are world-renowned, as evidenced by his busy consult service. He has over 400 papers and numerous chapters and monographs to his credit, but he is probably best known for his influential textbooks in surgical neuropathology. His first book, *Surgical Pathology of the Nervous System and Its Coverings,* written with Dr. F. Stephen Vogel, was published in 1976, just three years after he finished his residency; it is now in its fourth edition. He also teamed with Dr. Bernd W. Scheithauer to write the *Atlas of Tumor Pathology - Tumors of the Central Nervous System* for both the *Third Series* and the *Fourth Series*. Over the years, Dr. Burger has been a member of multiple brain tumor study groups and advisory boards, including the Board of Directors of the Registry of Pathology. He has received numerous awards, and at last count had given 245 invited lectures around the country and around the world. He has been a long-time member of the AANP, an active participant in its scientific programs, and an inspiration to numerous trainees to take up their own careers in academic neuropathology.



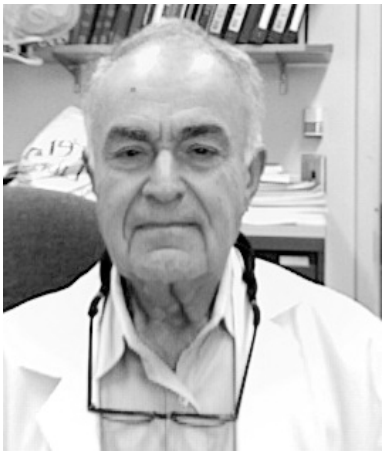
Dr. Pierluigi Gambetti, MD

Dr. Pierluigi Gambetti was born in Imola (Bologna), Italy and attended the University of Bologna School of Medicine, graduating Summa Cum Laude. He served in the army special corps of the "Alpini" and then trained in the Department of Neurology of the University of Bologna. In 1963, Dr. Gambetti served as a Fellow in Neuropathology at the Institut Bunge in Antwerp, Belgium and then as a Fellow of the Italian Ministry for Foreign Affairs in Rome, Italy. Dr. Gambetti received a fellowship from the Multiple Sclerosis Society to work in pathology at the University of Pennsylvania in 1966 and was later promoted to Associate Professor. He assumed the position of Professor and Director of the Division of Neuropathology at Case Western Reserve University School of Medicine and University Hospitals in Cleveland, Ohio in 1977.

Dr. Gambetti quickly established his group in the field of neurological diseases, focusing mainly on Alzheimer's Disease research. At case he trained many of our members Mark Cohen, George Perry, Rudy Castellani and Steven Younkin. He has been honored with the Weil Award for Best Paper in Experimental Neuropathology in 1983, the Potamkin Prize from the American Academy of Neurology in 1997, the Method to Extend Research in Time (Merit) Award from the National Institute on Aging in 1989, and the Leadership and Excellence in Alzheimer's Disease (LEAD) Award in 1991. He is also the past President of the American Association of Neuropathologist.

After discovering Fatal Familial Insomnia (FFI) in collaboration with Elio Lugaresi, Dr. Gambetti began his studies of prion diseases. His research led to the discovery of a novel mechanism of phenotype heterogeneity, protein typing, and novel variants of sporadic Creutzfeldt-Jakob Disease. With the sponsorship of the Centers for Disease Control and Prevention, Dr. Gambetti created the National Prion Disease Pathology Surveillance Center in 1997. This was the first center of its type in the U.S. and was established to track, collect, and test suspected cases of human prion diseases. Currently, he is the Professor of Pathology and Neurology at Case Western Reserve University and the Director of the National Prion Surveillance Center in Cleveland, Ohio.

Dr. Gambetti serves on numerous editorial boards of scientific journals and grant-making organizations. He has authored or contributed to more than 326 publications, journals, and books.



Dr. Nicholas K. Gonatas

Dr. Gonatas graduated from the Aristotle University in Thessaloniki, Greece, with an M.D. degree. He continued his training in Pathology and Neuropathology with H.M.Zimmerman and Robert Terry at the Montefiore Hospital (MH) in the Bronx (1957-61). Zimmerman was a critical pathologist who preached that " Patholgy is not Theology". He experimentally induced and studied brain tumors when most Neuropathologists were obsessed with "pattern recognition" and clinicopathological correlations. In 1960-61, Dr. Terry left MH to join Saul Korey at the newly founded Albert Einstein College of Medicine; on January 1962, Dr. Gonatas joined them to continue his training in clinical and experimental Neuropathology. The Terry laboratory was close to Saul's , which in addition to Neurologists included two neurochemists, Bill Norton and Stanley Samuels, and a most unusual and unique cell biologist, the late Elliott Robbins. The interactions among the members of this multidisciplinary, group were spontaneous and the best way to describe the style of "governance" exerted by Korey and Terry was " benign neglect", a term coined by Terry. The existence of such a group was unique to Academic Departments of Neurology of that era, most of which were focused in clinical Neurology and Neuropathology; the combination of electron microscopy , cell biology and neurochemistry were indeed novelties for most Departments of Neurology in the USA and Europe.

In 1964, Dr. Gonatas was recruited by the late G.Milton Shy, Chairman of the department of Neurology at the University of Pennsylvania to take over Neuropathology and collaborate with him in the pathology of skeletal muscle. Dr. Gonatas has been in Philadelphia for the last ..41 years where he became the Director of the Division of Neuropathology and was very fortunate to have over 25 trainees who upon "graduation" developed into outstanding Neuropathologists and made major contributions to both basic Neurobiology and Neuropathology, individuals such as Pierluigi Gambetti, John Trojanowski, Bill Hickey, Albee Messing, to name just a few.

Dr. Gonatas' work took us from muscle pathology, to Neurodegenerative diseases, and to Cell biology, especially the role of the Golgi apparatus in secretion and receptor-mediated endocytosis. Two of his 220 papers became Citation Classics, one from the Einstein years on the mitotic cell with the late E. Robbins, and the second, from the University of Pennsylvania, on the application of lectins in studies of anatomic connections.

AANP PRESIDENTIAL SYMPOSIUM
Sunday, 14 June 2009

Oxidative Stress in Neurodegenerative Diseases

8:00 a.m. **Opening and Welcoming Remarks**
George Perry, PhD
University of Texas, San Antonio, TX

8:10 a.m. **Bioenergetics in the Pathogenesis of Neurodegenerative Diseases**
M. Flint Beal, MD
Weill Medical College, New York, NY

8:40 a.m. **Free Radical Damage as a Therapeutic Target in Alzheimer's Disease**
Thomas J. Montine, MD, PhD
University of Washington, Seattle, WA

9:10 a.m. **Abeta Amyloid Oligomers as Therapeutic and Diagnostic Targets for Alzheimer's Disease**
Colin Masters, MD
University Melbourne, Melbourne, Australia

9:40 am **Awards Ceremony**

10:00 am **Refreshment Break**

10:30 am **Matthew T. Moore Distinguished Lecture**
From Charcot to Lou Gehrig: Mechanisms and Treatment of ALS
Donald W. Cleveland, MD
UCSD, San Diego, CA

11:15 am **Causes and Consequences of Oxidative Damage in Alzheimer's Disease**
Mark A. Smith, PhD
Case Western Reserve University, Cleveland, OH

2009 PRESIDENTIAL SYMPOSIUM

Opening and Welcoming Remarks
George Perry, PhD
University of Texas, San Antonio, TX

Dr. George Perry, PhD

Dr. George Perry is currently Dean of the College of Sciences at The University of Texas at San Antonio. Perry received his doctoral degree from the University of California at San Diego. He served as a postdoctoral fellow in cell biology at Baylor College of Medicine and earned a bachelor's degree in zoology from the University of California at Santa Barbara. Perry's most known work, published with Mark A. Smith and Lawrence Sayre, highlight oxidative stress in the study of AD. Perry has directed Case Western Reserve University's Department of Pathology. He has authored more than 800 scientific publications and 600 abstracts and is among the most cited authors in Alzheimer's disease research. He serves on the editorial board of more than 70 publications and is founding editor-in-chief of the highest impact journal in AD, *The Journal of Alzheimer's Disease*. In 1998, Perry was elected as a Fellow by the American Association for the Advancement of Science. He recently won the Denham Harmon Lifetime Achievement Award (discoverer of the Free Radical Theory of Aging) from the American Association for Aging, and currently holds the Zenith Award from the Alzheimer Association. He collaborates extensively with leading scientists and educators in Portugal, Spain and Latin America. He was awarded a *Doctorado Honoris Causa* from Chile and membership in the Iberoamerican Molecular Biology Organization for his educational and economic development efforts in Iberia and Latin America

The role of oxidative stress in neurological disease is one of the few new concepts moving to the forefront in the past decade with demonstration of oxidative damage and mutations in oxidative enzymes. This symposium reviews the state of knowledge in the topic by pioneering leaders in the field. Our own work began with trying to unravel protein insolubility in Alzheimer disease, a property first described at the 1981 AANP meeting. The intricate nature of oxidative stress and response opens new opportunities to the study and treatment of disease.

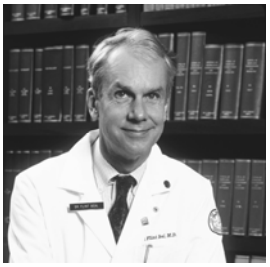
Objectives:
1. Introduce topic of oxidative stress.
2. Present overview of oxidative chemistry and its measurement.

Disclosures: Dr. Perry is a consultant for Takeda Pharmaceutical, Neurotez Pharmaceuticals and Memory XL and own equity in Neurotez Pharmaceuticals, Voyager Pharmaceuticals and Panacea Pharmaceuticals.

2009 PRESIDENTIAL SYMPOSIUM

Bioenergetics in the Pathogenesis of Neurodegenerative Diseases

M. Flint Beal, MD
Weill Medical College, New York, NY



M. Flint Beal, MD

Dr. M. Flint Beal is an internationally recognized authority on neurodegenerative disorders. Dr. Beal received his medical degree from the University of Virginia in 1976 and did his internship and first year residency in Medicine at New York-Cornell before completing his residency in Neurology at The Massachusetts General Hospital. He joined the neurology faculty at Harvard in 1983. Dr. Beal was Professor of Neurology at the Harvard Medical School and Chief of the Neurochemistry laboratory at Massachusetts General Hospital before moving to Cornell. Dr. Beal's research has focused on the mechanism of neuronal degeneration in Alzheimer's Disease, Huntington's Disease, Parkinson's Disease and amyotrophic lateral sclerosis (ALS).

Dr. Beal is the author or co-author of more than 400 scientific articles and more than 125 books, book chapters and reviews. He serves on the editorial boards of seven journals, including the Journal of Neurochemistry, the Journal of Neurological Sciences, Journal of Molecular Neuroscience, Experimental Neurology and Neurobiology of Disease. He is a co-editor of the "Dana Guide to Brain Health".

Dr. Beal is a member of the Alpha Omega Alpha Medical Honorary Society and received the Derek Denny-Brown Neurological Scholar Award of the American Neurologic Association. He has served on the Council of the American Neurologic Association and on the Science Advisory Committees of the Hereditary Disease Foundation, Huntington's Disease Society of America, Parkinson's Disease Study Group, Parkinson' Disease Foundation, Bachman-Strauss Foundation, The ALS Association, and the American Health Assistance Foundation. Dr. Beal is a member of the Institute of Medicine of the National Academy of Sciences.

Objectives:

1. Understand the role of mitochondrial dysfunction in neurodegenerative disorders.
2. Understand the pleotropic mechanisms involved in energetic failure in neurodegenerative disorders.
3. Understand therapeutic opportunities targeted at restoring bioenergetics.

Disclosures: Dr. Beal has nothing to disclose.

2009 PRESIDENTIAL SYMPOSIUM

Free Radical Damage as a Therapeutic Target in Alzheimer's Disease

Thomas J. Montine, MD, PhD
University of Washington, Seattle

Tomas J Montine MD, PhD

Thomas J. Montine is a Professor of Pathology and Neurological Surgery at the University of Washington in Seattle, and Adjunct Professor of Neurology at Oregon Health & Sciences University. He holds the Nancy and Buster Alvord Endowed Chair in Neuropathology at the University of Washington where he is Director of the Division of Neuropathology. Dr. Montine received his BS in Chemistry from Columbia University, his PhD in Pharmacology from the University of Rochester with Dr. Rick Borch, and his MD from McGill University where he also did post-doctoral research with Dr. Ted Sourkes. He trained in Anatomic and Neuropathology at Duke University before becoming Assistant and then Associate Professor of Pathology and Pharmacology at Vanderbilt University. Dr. Montine's research is focused on molecular mechanisms of injury and therapeutic targets in age-related neurodegenerative diseases.

Objectives:

1. Understand the biochemistry of free radical mediated damage.
2. Understand regional free radical injury as a shared mechanism of injury among several diseases that cause dementia.
3. Understand free radical injury as a therapeutic target for some neurodegenerative diseases.

Abstract:
Regional free radical injury to select components of brain is a mechanism shared by several neurodegenerative diseases. Here we will review the biochemical mechanisms of free radical injury to macromolecules in brain, and the cellular targets of free radical injury in common causes of dementia. In addition, we will review data suggesting that free radical injury is a potential therapeutic target for patients with early disease, and present emerging data from recent clinical trials.

Disclosures: Dr. Montine is a consultant to Bristol-Myers Squibb, Medisyn, and Xenova.

2009 PRESIDENTIAL SYMPOSIUM

Aβ oligomers as tractable targets for Alzheimer's disease diagnostics and therapeutics

Colin L. Masters, MD
Mental Health Research Institute; The University of Melbourne

Colin L. Masters, MD

Dr. Colin Masters has specialized in research into Alzheimer's disease and other neurodegenerative disease, including Creutzfeldt-Jakob and other prion diseases, and his work over the last 35 years is widely acknowledged as having had a major influence on Alzheimer's disease research world-wide. His scientific achievements have provided a path to the current development of therapeutic strategies for Alzheimer's and other neurodegenerative diseases. From the discoveries of the Aβ amyloid protein in the brain plaques of Alzheimer's disease in the 1980's, he and his colleagues have gone on to elucidate the pathways leading to the toxicity and accumulation of Aβ in the aging human brain and have been instrumental in developing these as therapeutic targets for drug development. He is widely acknowledged as having had a major influence over the last two decades on the direction of a now world-wide research effort into the cause and diagnosis of Alzheimer's disease, and continues to do so in the emerging drug development and biotechnology arena.

Masters' work has also opened up new insights into other major neurodegenerative diseases (such as Creutzfeldt-Jakob and Parkinson's diseases) in which aggregated proteins accumulate, work that has thus provided clues to therapeutic interventions for multiple disease states. Masters has held many senior scientific positions and is currently the Executive Director of the Mental Health Research Institute, and Laureate Professor at the University of Melbourne. He is the Chair of the NHMRC's National Heath Committee, a consultant in neuropathology at the Royal Melbourne Hospital and a scientific advisor to Neurosciences Australia. His achievements have been recognised by the receipt of many international awards - including the Potamkin Prize from the American Academy of Neurology (1990), the King Faisal International Prize in Medicine (1997), the Alois Alzheimer Award from the University of Munich (1998), the Lennox K. Black Prize and Grand Hamdan International Award for Medical Sciences (2006), the Victoria Prize (2007) and the Leach Medal (2009).

Objectives:

1. To present an overview of where the neuropathology of Alzheimer's disease has progressed since 1985, when the nature of the Aβ amyloid plaques was elucidated;
2. To explain the current rationale for drug development with Aβ as the target;
3. To present he results of clinical trials of developing Aβ as a therapeutic target;
4. To present the latest evidence that Aβ may be used as a diagnostic target in both molecular neuroimaging studies (PET scanning) and for immunoassays of $A\beta_{40,\,42}$ in CSF and blood;
5. To emphasize the continuing need for sophisticated neuropathology in demarcating the Alzheimer neurodegeneration process from confounding factors such as microvascular disease and other causes of neurodegeneration.

Abstract:
Compelling evidence now shows that the Aβ-amyloid peptide is the central biochemical marker of Alzheimer's disease, and is the most likely cause of the neurodegeneration manifest in synaptic dysfunction and eventual neuronal loss. Pathways up-stream of Aβ production provide therapeutic targets amenable to protease inhibition/modulation. Strategies which affect APP trafficking may also prove of

value. Downstream, pathways promoting the degradation of Aβ or modulating clearance from the brain also offer windows for therapeutic opportunity.

Central interest lies in the mechanism through which Aβ undergoes toxic gain-of-function, inducing neuronal damage. This provides the most direct route for therapeutic intervention, with least risk of therapeutic side-effects, since Aβ toxicity is unlikely to mimic any normal function. Two principal hypotheses have emerged to explain Aβ toxicity: redox chemistry associated with the Cu/Zn metal binding sites on Aβ, and lipid interactions associated with the α/β conformation of the hydrophobic C-terminus. Drugs targeting these mechanisms are now in clinical development with encouraging preliminary results. The normal function of APP remains elusive despite two decades of research. Dimerization of APP through the transmembrane and other domains leads to proteolytic release and signalling through its cytoplasmic domain. Perturbation of processing may lead to excessive production of Aβ peptide. Either as a soluble oligomer or insoluble amyloid fibril, the accumulation of the Aβ fragment provides a pivotal biomarker, currently being developed as a neuroimaging target and a blood/CSF biomarker for efficacy of therapeutic intervention, and for gene-linkage discovery.

Disclosures: Prof. Masters has interests in Prana Biotechnology and has served with several major pharmaceutical companies as a consultant.

2009 PRESIDENTIAL SYMPOSIUM

Matthew T. Moore Distinguished Lecture
From Charcot to Lou Gehrig: Mechanisms and Treatment of ALS
Donald W. Cleveland, MD
UCSD, San Diego, CA

Donald W. Cleveland, MD

Dr. Don Cleveland is Professor and Chair of the Department of Cellular and Molecular Medicine at the University of California at San Diego, as well as a member of the Ludwig Institute for Cancer Research. Valedictorian of his class at New Mexico State University, he has been elected to the National Academy of Sciences and the American Academy of Arts and Sciences. A recipient of three NIH Merit Awards, he has also won the Wings Over Wall Street MDA Outstanding Scientist award and The Sheila Essey Prize from the ALS Association and American Academy of Neurology. As a graduate student at Princeton, he discovered the microtubule associated protein tau which accumulates aberrantly in the intracellular tangles of Alzheimer's disease. As a postdoctoral fellow at UCSF, he was the first to clone and identify the gene families of each of the three major cytoskeletal proteins (tubulin, actin and keratins). While on the faculty of Johns Hopkins, he demonstrated that extreme asymmetry of neurons is achieved with a deformable array of interlinked neurofilaments, microtubules and actin. He showed that disorganization of neurofilaments causes selective failure of motor neurons in mice and humans. He then demonstrated that similar disease could also arise by a toxicity of mutant superoxide dismutase unrelated to its normal activity, thereby uncovering the mechanism underlying the major genetic form of Amyotrophic Lateral Sclerosis (ALS). His efforts identified key steps that trigger disease and that accelerate its progression. Most importantly, he also showed that motor neuron death in inherited ALS is non-cell autonomous, requiring mutant damage to both motor neurons and the neighboring supporting cells. These findings have redirected efforts at stem cell and gene silencing therapies in ALS with wide implications for the other major neurodegenerative diseases, since the inherited forms of each are also caused by widely expressed mutant genes. His team has developed a gene silencing therapy for ALS that will enter clinical trial in 2009. An extension of this approach to Huntington's disease is expected to enter trial in 2011.

Objectives:

1. Describe the known genetic causes of ALS.
2. Describe how mutation in superoxide dismutase causes age-dependent loss of motors through damage within motor neurons and their non-neuronal neighbors.
3. Describe how gene silencing or stem cell therapies may be useful in treatment of inherited ALS.

Abstract:
Since its description by Charcot in 1869, the mechanism of selective death of motor neurons in Amyotrophic Lateral Sclerosis (ALS) has remained elusive. An inherited form is caused by mutation in superoxide dismutase (SOD1) that causes disease from an acquired toxicity unrelated to dismutase activity. A proportion of both dismutase active and inactive mutants is stably bound to spinal cord mitochondria. Misfolded SOD1 has also been implicated within microglia or astrocytes in aberrant binding to the small G protein Rac1. Paradoxically, binding of misfolded superoxide dismutase locks Rac1 in its GTP bound form that then chronically activates Nox2 to produce >100 times the normal amount of extracellular superoxide. Similarly, misfolded SOD1 binds derlin, thereby inhibiting the ERAD system for ejection of misfolded proteins from the endoplasmic reticulum. Using mice carrying a

deletable mutant gene or viral encoded siRNA to diminish mutant expression within motor neurons, disease onset is slowed but progression is not. Conversely, reducing mutant SOD1 synthesis in astrocytes or microglia has little effect on disease onset, but strikingly slows disease progression. Thus, toxicity is non-cell autonomous, with mutant SOD1 acting within motor neurons driving disease onset, while damage within neighboring astrocytes and microglia accelerates disease progression. These findings validate therapies, including stem cell replacement approaches or gene silencing approaches, that target astrocytes or microglia.

Causes and Consequences of Oxidative Stress in Alzheimer's Disease

Mark A. Smith Ph.D., FRCPath
Case Western Reserve University

Mark A. Smith, Ph.D.

Dr. Mark A. Smith received his B.Sc. with Honors in Molecular Biology and Biochemistry from Hatfield College, Durham University, England (1986). He went on to earn a Ph.D. in Biochemistry from Nottingham University, England, in 1990. Dr. Smith spent the next two years as a Research Fellow at Sandoz Forschungsinstitut in Vienna, Austria. Presently, he is Professor of Pathology at Case Western Reserve University and serves as Director of Basic Research at the University Memory and Cognition Center. Dr. Smith is a Fellow of the American Aging Association and a Fellow of the Royal College of Pathologists. The focus of Dr. Smith's research involves investigating the pathological mechanism(s) underlying selective neuronal death in neurodegenerative diseases such as Alzheimer's disease. Dr. Smith's research involves a variety of techniques ranging from histological to molecular biology to cellular models and encompasses diagnostic, mechanistic, and therapeutic strategies. Current projects are directed towards 1) fundamental metabolic alterations; 2) homeostatic dysregulation of transition metals; 3) signal transduction alterations; and 4) inappropriate re-entry into the cell cycle. Dr. Smith has authored over 600 peer-reviewed manuscripts and chapters and is recognized as one of the top cited researchers in the fields of Neuroscience & Behavior (http://www.in-cites.com/nobel/2007-neu-top100.html), Alzheimer Disease (http://www.esi-topics.com/alzheimer/authors/b1a.html) and Free Radical Biology (http://www.freeradicalscience.com/labs.php). Dr. Smith is the recipient of several awards including the Ruth Salta Junior Investigator Achievement Award from the American Health Assistance Foundation, Young Scientist Lectureship Award from the International Society for Neurochemistry, the Nathan Shock New Investigator Award from The Gerontological Society of America, the Zenith Award from the Alzheimer's Association, the Jordi Folch-Pi Award from the American Society of Neurochemistry, and the Hermann-Esterbauer Award from the HNE Society. Dr. Smith has also been recognized for his contributions to teaching with, among others, the Outstanding Mentor Award, School of Graduate Studies and the Jackson Award for Undergraduate Mentoring, Case Western Reserve University.

Objectives:

1. Describe the major oxidative modifications associated with Alzheimer disease
2. Describe the upstream causes of oxidative stress
3. Describe the downstream consequences of oxidative stress
4. Give examples of therapeutic strategies to reduce oxidative stress

Abstract:
Over the past two decades, the study of oxidative stress in relation to the pathogenesis of Alzheimer's and other neurodegenerative diseases has been transformed from an esoteric backwater to a leading paradigm. This rapid ascension is perhaps not surprising for two major reasons. First, it is now established that oxidative stress is the earliest cytological feature of Alzheimer's disease and, consequently, an attractive therapeutic target. Second, oxidative stress seemingly accounts for the pleiotropic nature of the disease both in terms of etiology and pathogenesis. While there is now agreement on the importance of oxidative stress, the major controversies and challenges ahead are in establishing the source of the reactive oxygen, as well as determining the multiplicity of downstream consequences of redox imbalance. These aspects will certainly help us further understand the complexity of Alzheimer's disease as well as provide viable therapeutic targets.

Disclosures: Dr. Smith receives, or has received, lecture fees, consulting fees and/or equity from Neuropharm, Neurotez, Medivation, Voyager Pharmaceutical Corporation, Anavex and Canopus.

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