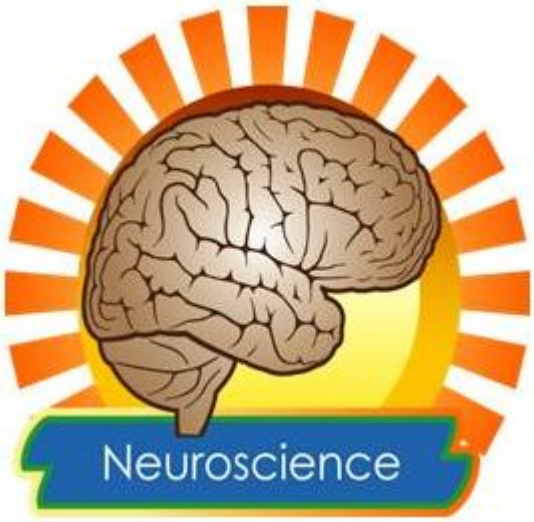
# Conference Handbook

## TM's 2nd World Neuroscience Online Conference

**June 18-20, 2013**



Dear colleagues

Thank you so much for taking time out of your busy schedule to participate in online conference - **TM's 2nd World Neuroscience Online Conference,** which will be held **on June 18-20, 2013.** It will be very helpful to speakers, attendees, and other related people. Target Meeting appreciates your attendances and generous contribution. We hope you will enjoy the difference.

Sincerely Yours

Target Meeting Team in USA

**Preparation in advance:** USB headset with microphone

# Instructions

1. Attendees can participate in all or part of tracks (sessions). It depends on your time or interest. All conference information is available on http://targetmeeting.com/Modules/Meetings/MeetingDetails.aspx?Id=51.

2. **Double click the track links** at scheduled date & time to join the conference. The conference component software will be downloaded and installed on your computer automatically (about 30 seconds) when you click the track links. If not, please manually download the component software on your computer after you click the track links. Normally the component software will be saved in the download folder, my document, desktop, or somewhere. It depends on your computer. Double click it to run this software (**You must have right to install software on your computer**). Then you will enter the "Conference Room". It is completely secure.

3. If you want to talk with speakers at the Q&A sessions, please click the icon "**hand**" on the conference control panel. The conference organizer will unmute your headset, then you can discuss with them in real time. Do not close the control panel on your computer during the sessions. Please type messages in the control panel and sent it to organizers if you have any questions during the conference. Organizers will reply you in private.

## Conference Partners

For more than four decades, LI-COR(R) Biosciences has been helping scientists advance discovery by providing innovative research tools. And with more than twenty years of experience in biotechnology, LI-COR is now a leading manufacturer of near-infrared imaging platforms, analysis software, and IRDye(R) infrared dye reagents. LI-COR pioneered the development of near-infrared fluorescence systems for DNA sequencing, and today provides systems for drug discovery, protein research, small animal imaging, and undergraduate training. These tools provide research solutions for a wide variety of applications, including quantitative Western blotting, small animal imaging, and cell-based assays. Research toward a Cure is part of our ongoing effort to develop research tools and highlight techniques that enhance scientists' technical capabilities to advance cancer research. Currently, thousands of LI-COR systems are being used in laboratories around the world for advanced research and drug development. Our website features published examples of applications and techniques where infrared fluorescence detection contributes to the understanding of cancer and the search for cures. Please visit www.licor.com/cancer for more information. In addition to the biotechnology lines of instruments and reagents, LI-COR instruments for photosynthesis, carbon dioxide analysis, and light measurement are recognized worldwide for standard-setting innovation in plant science research and environmental monitoring. Founded in 1971, the privately held company is based in Lincoln, Nebraska, with subsidiaries in Germany and the United Kingdom. LI-COR systems are used in over 100 countries and are supported by a global network of distributors.

Take part in what promises to be an unforgettable and enriching experience at the XXI World Congress of Neurology (WCN 2013) which will take place from 21-26 September in Vienna, Austria. Join thousands of colleagues and leading experts as WFN, EFNS and ÖGN, representing 140 different neurological societies worldwide, come together to provide an unparalleled scientific program and educational event which has been approved to provide up to 30 CME credits. With over 2500 abstracts submitted, 92 scientific sessions, 60 teaching courses, 10 plenary lectures, 10 sponsored symposia, over 400 renowned speakers, various debates and one stimulating Tournament of the Minds competition, the XXI World Congress of Neurology is an event not to be missed!! Find out more on the official Congress website: www.wcn-neurology.com.

The American Society for Experimental NeuroTherapeutics (ASENT) is an independent non-profit organization established in 1997 by leaders in academia, government, advocacy and industry to facilitate the process by which new therapies are made available to patients with neurological disorders. Its primary goal is to encourage and advance the development of improved therapies for diseases and disorders of the nervous system. ASENT engages in scientific

exchanges to encourage contacts between those involved in the discovery and development of neurotherapeutics and to provide opportunities for dialogue between the interested groups. The society organizes education and training and publishes a journal for healthcare practitioners, scientists and officials participating in the neurotherapeutics field, which serves as a forum for its members and interested groups addressing diverse issues. Vison Statement: To advance the science of neurotherapeutics and to improve and accelerate the development of new treatments. Mission: ASENT combines academia, government, clinical community, industry and advocacy to enable and advance the discovery, translation and development of neurotherapeutics. Goals: Drive the development of next generation therapeutics through world-class dialogue. Promote the highest scientific and ethical principles in therapeutic discovery and clinical investigation. Create a strong organization scaffold to support ASENT.

Global College of Neuroprotection & Neuroregeneration (GCNN) was established in 2003 with the aim to bring both basic and clinical neuroscientists on the same platform to exchange the ideas and eventually develop fruitful co-operation for exploring a suitable therapeutic strategies to cure of neurodegenerative diseases. The basic aim of the GCNN is to organize International Conferences, Seminars, Symposia and Workshops including Summer schools in different parts of the Globe to make people aware about the latest development in neuroprotection and neuroregeneration strategies. Also, regular programs for medical students are developed in different parts of the World including Eastern Europe, Asia and Latin America to make them aware about the recent developments in neuroprotective strategy both in the basic research and current trends in clinical practices. American Journal of Neuroprotection and Neuroregeneration is the official organ of the GCNN.

Leica Microsystems is a world leader in microscopes and scientific instruments. Founded as a family business in the nineteenth century, the company's history was marked by unparalleled innovation on its way to becoming a global enterprise. Its historically close cooperation with the scientific community is the key to Leica Microsystems' tradition of innovation, which draws on users' ideas and creates solutions tailored to their requirements. At the global level, Leica Microsystems is organized in three divisions, all of which are among the leaders in their respective fields: the Life Science Division, Industry Division and Medical Division. The company is represented in over 100 countries with 6 manufacturing facilities in 5 countries, sales and service organizations in 20 countries, and an international network of dealers. The company is headquartered in Wetzlar, Germany.

Medical News Today is the largest independent medical and health news site on the web - with over 2,500,000 unique monthly users it is ranked number one for medical news on Google and Yahoo!. Medical News Today is used by Blue Chip pharmaceutical and health organizations,

advertising agencies, PR companies and vertical ad networks to deliver targeted disease/condition and general health campaigns. For more information, please visit www.medicalnewstoday.com.

20/20 Pharma, published by IMI, is a magazine and website which presents insightful analysis of current events, developments, and trends in the pharmaceutical world.The publication has forged powerful relationships with key industry leaders to provide a platform for decision makers to have the means to procure and plan implementation strategies based on the topics covered.

World Conference Calendar is a directory publishing information on academic conferences all over the world. Knowledge is really appreciated only when it reached a user. Conferences are one of the best environments that this knowledge is delivered to a large audience. As World Conference Calendar, we are trying to be an effective medium to point out where these exchanges will take place.

Clocate.com is a leading international search engine and directory for worldwide conferences and exhibitions. The events cover the following areas: Industry and manufacturing, Health and medicine, Technology and IT, Business and finance, sciences, education, services (banking, insurance, tourism, Hospitality and more), government, environment, life style and arts. The details for each event include: description, dates, location, address, prices and more.

Since 2006, we manufacture and sell advanced chemicals for life science research, and diagnostics. Our catalog includes fluorescent dyes, phosphoramidites for oligonucleotide synthesis, Click chemicals, and other reagents. Most of the items are kept in stock, and ready for immediate shipping to worldwide locations. Quick turnaround time, stock availability, and flexible return/refund policy makes ordering quick, fun, and riskless. Wherever you are, it usually takes no more than a week to receive your order. Overnight or next day shipping is available in some locations. With our quick and qualified technical support, our customers will never be left alone facing technical difficulties. Please feel free to contact us to get additional information, and free advice. We offer special conditions for bulk and custom orders. Please inquire if you have any special needs.

# Conference Program (All times are New York Time)

**Track 1: 8:00AM – 17:00PM, June 18, 2013**

**Session 1**: **Cellular & Molecular Neuroscience-1**
8:30 AM – 10:30 AM

**Session 2**: **Cellular & Molecular Neuroscience-1I**
10:30 AM – 13:00 PM

**Session 3**: **Neurosciences & Mathematical Model-I**
13:00 PM – 14:30 PM

**Session 4**: **Cellular & Molecular Neuroscience-III**
14:30 PM – 17:00 PM

**Track 2: 8:00AM – 17:30PM, June 19, 2013**

**Session 5**: **Neurosciences & Mathematical Model-II**
8:30 AM – 10:00 AM

**Session 6**: **Neurodegenerative Diseases-I**
10:00 AM – 11:30 AM

**Session 7**: **Traumatic Brain & Spinal Cord Injury**
11:30 AM – 13:00 PM

**Session 8**: **Neuro Sensory & Nervous System Development**
13:00 PM – 15:30 PM

**Session 9**: **Neurodegenerative Diseases-II**
15:30 PM – 17:30 PM

**Track 3: 8:00AM – 17:30PM, June 20, 2013**

**Session 10**: **Pain Management**
8:30 AM – 10:30 AM

**Session 11**: **Behavioral & Cognitive Neuroscience-I**
10:30 AM – 14:00 PM

**Session 12**: **Cellular & Molecular Neuroscience-IV**
14:00 PM – 16:00 PM

**Session 13**: **Behavioral & Cognitive Neuroscience-II**
16:00 PM – 17:30 PM

## Track 1: 8:00 AM– 17:00 PM, June 18, 2013

**8:00 – 8:30 AM**
Speakers and attendees can login the online conference.

## Session 1: Cellular & Molecular Neuroscience-1
## 8:30 AM – 10:30 AM
## Session Chair: Pending

**8:30 – 9:00 AM**
**Presentation Title:** A protective role of apolipoprotein E-containing lipoproteins in glutamate-induced neurodegeneration.
**Hideki Hayashi**, Ph.D., Assistant Professor, Department of Ophthalmology, Faculty of Life Sciences, Kumamoto University, Japan.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**9:00 – 9:30 AM**
**Presentation Title:** The Phosphoinositide signal transduction Pathway and the development of human nervous system.
**Rita Lo Vasco**, Assistant Professor, Department of Sensitive Organs, Policlinic Umberto I, Sapienza University, Rome, Italy.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**9:30 – 10:00 AM**
**Presentation Title**: Effect of the simultaneous impact of opioid drug (tramadol) and electromagnetic field (EMF) on lipid peroxidation.
**Pawel Bodera**, Professor, Military Institute of Hygiene and Epidemiology, Warsaw, Poland.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**10:00 – 10:30 AM**
**Presentation Title:** The evolution of acetylcholine and GABA from waste products to neurotransmitters.
**Keith Harris,** Department of Zoology, Tel Aviv University, Israel. **Amotz Zahavi** , Emeritus Professor, Department of Zoology, Tel Aviv University, Israel.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**Panel Discussion.** This session is to provide speakers and attendees with in-depth discussion and networking. You can discuss what you want with other speakers. All speakers are unmuted, so speakers can talk freely during the session.

## Session 2: Cellular & Molecular Neuroscience-1I
## 10:30 AM – 13:00 PM
## Session Chair: Dr. Massimo Cocchi

**10:30 – 11:00 AM**
**Presentation Title:** Nervous circuit in the tail of trematode cercaria: from morphology to motility regulation.
**Oleg Tolstenkov**, Scientist, Severtsov Institute of ecology and evolution of Russian Academy of Sciences, Leninskiy prospect, 33, Moscow, 119071 Russia.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**11:00 – 11:30 AM**
**Presentation Title:** Could neurobiological correlates of Bipolar Disorder be seen as a support for differential diagnosis?
**Massimo Cocchi**, Professor, Institute 'Paolo Sotgiu' Quantitative and Evolutionary Psychiatry and Cardiology, L,U,De,S, University, Lugano, Switzerland. **Lucio Tonello**, Professor of Bio Mathematical Sciences, Institute "Paolo Sotgiu" Quantitative and Evolutionary Psychiatry and Cardiology, L.U.de.S. University, Switzerland.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**11:30 – 12:00 PM**
**Presentation Title**: New insights in neurodegeneration: Extra-mitochondrial oxidative phosphorylation in nervous system.
**Isabella Panfoli**, Aggregate Professor, School of Medical and Pharmaceutical Sciences, University of Genova, Italy.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**12:00 – 12:30 PM**
**Presentation Title:** Angiogenesis in meningiomas.
**Christina Pfister**, Researcher, Department of Neurosurgery, University of Tuebingen, Hoppe-Seyler-Str. 3, 72076, Tuebingen, Germany.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**12:30 – 13:00 PM**
**Presentation Title:** Reduced protein translation rates in a Drosophila model for GARS-associated CMT.
**Erik Storkebaum**, PhD, Max Planck Research Group Leader, Molecular Neurogenetics Laboratory, Max Planck Institute for Molecular Biomedicine, Germany.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Session 3: Neurosciences & Mathematical Model-I
## 13:00 PM – 14:30 PM
## Session Chair: Dr. Alan Palmer

**13:00 – 13:30 PM**
**Presentation Title:** Translating neuroscience research into CNS drugs.
**Alan Palmer**, FIBiol, PhD, MSc, BSc, Board member of MS Therapeutics Ltd (MST) and One Nucleus Ltd, a visiting Professor at University College London, and a Fellow of the Society of Biology UK.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**13:30 – 14:00 PM**
**Presentation Title:** Imaging 3D spatiotemporal hemodynamics of single cortical vessels in vivo using two-photon laser scanning microscopy.
**Peifang Tian**, Assistant Professor, John Carroll University, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**14:00 – 14:30 PM**
**Presentation Title:** Helping people with disabilities with Brain-Computer Interfaces.
**Pablo F. Diez**, PhD, Biomedical Engineer, Assistant Professor at the "Gabinete de Tecnología Médica" (Medical Technology Laboratory) at the "National University of San Juan" from Argentina.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Session 4: Cellular & Molecular Neuroscience-III
## 14:30 PM – 17:00 PM
## Session Chair: Dr. Lester Ingber

**14:30 – 15:00 PM**
**Presentation Title:** Immobilized laminin concentration gradients on electrospun fiber scaffolds for controlled neurite outgrowth.
**Nicole E Zander**, U.S. Army Research Laboratory, Weapons and Materials Research Directorate, Aberdeen Proving Ground, Maryland 21005, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**15:00 – 15:30 PM**
**Presentation Title:** Electroencephalographic field influence on calcium momentum waves.
**Lester Ingber**, President, Lester Ingber Research, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**15:30 – 16:00 PM**
**Presentation Title:** Neuropsychiatric Presentation of Anti-NMDA-receptor Encephalitis.
**Dong (Dan) Y. Han**, PsyD, Chief, UK Neuropsychology Service - Clinical Section, Director, UK Multidisciplinary Concussion Program, Assistant Professor, Department of Neurology, & Spinal Cord and Brain Injury Research Center, University of Kentucky College of Medicine, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**16:00 – 16:30 PM**
**Presentation Title:** N-Butanol-Induced developmental neurotoxicity in animals.
**Ambuja Bale**, Researcher, National Center for Environmental Assessment, Office of Research and Development, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**16:30 – 17:00 PM**
**Presentation Title:** Pending
**Hussein Al-Wadei**, Assistant Professor College of Veterinary Medicine, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Track 2: 8:00 AM – 17:30 PM, June 19, 2013

**8:00 – 8:30 AM**
Speakers and attendees can login the online conference.

### Session 5: Neurosciences & Mathematical Model-II
### 8:30 AM – 10:00 AM
### Session Chair: Pending

**8:30 – 9:00 AM**
**Presentation Title:** Comparing the cost-effectiveness of two brain metastasis treatment

modalities from a payer's perspective: Stereotactic radiosurgery versus surgical resection.
**Vuong Anh Duong**, MD. MBA-HM. PhD, Deputy head of medical professional and legislation Division at the Department of Medical Service Administration, under the Ministry of Health, Vietnam.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**9:00 – 9:30 AM**
**Presentation Title**: Effects of real-life experience on human face processing performance.
**Meike Ramon**, Researcher, Institute of Neuroscience and Psychology, UK. Meike Ramon.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**9:30 – 10:00 AM**
**Presentation Title:** Open questions and perspectives in DBS technology.
**Daniela Sabrina Andres**, Institute of Neuroinformatics, UZH/ETHZ, Zurich, Switzerland; Institute for Neurological Research Raul Carrea, Fleni Institute, Movement Disorders Section, Buenos Aires, Argentine; Society in Science, The Branco-Weiss Fellowship, administered by ETH, Zurich, Switzerland.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Session 6: Neurodegenerative Diseases-I
## 10:00 AM – 11:30 AM
## Session Chair: Dr. Maria Carmen Portillo

**10:00 – 10:30 AM**
**Presentation Title:** Tau Mediated Neurodegeneration: An Insight into Alzheimer's Disease Pathology.
**Magisetty Obulesu**, Assistant Professor, Department of Biotechnology, Rayalaseema University, Kurnool, Andhra Pradesh, India.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**10:30 – 11:00 AM**
**Presentation Title:** Neuropsychiatric Syndromes in a Patient with Lupus from bench to bedside.
**Reem Hamdy A Mohammed** MD, PhD, FACR, BSR member, Associate Prof. Rheumatology and Rehabilitation, Faculty of Medicine, Cairo University.Rheumatology Consultant, Alhada Military Hospital, KSA., Egypt.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**11:00 – 11:30 AM**
**Presentation Title**: ReNaCE Programme. Integrating Parkinson's Disease in patients' and carers' lives".
**Maria Carmen Portillo**, PhD MSc, Associate professor, School of nursing, University of Navarra, Spain.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Session 7: Traumatic Brain & Spinal Cord Injury
## 11:30 AM – 13:00 PM
## Session Chair: Dr. Hari Shanker Sharma

**11:30 – 12:00 PM**
**Presentation Title**: CNS Injury and Repair. Novel Therapeutic Strategies and Nanodrug Delivery.
**Hari Shanker Sharma**, Professor of Neurobiology (MRC), University Hospital, Uppsala University, Sweden.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**12:00 – 12:30 PM**
**Presentation Title:** Traumatic Brain Injury and Suicide: Current State of the Evidence.
**Lisa A. Brenner** PhD ABPP, Board Certified Rehabilitation Psychologist, Associate Professor & Director, U.S. Department of Veterans Affairs, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**12:30 – 13:00 PM**
**Presentation Title:** Inhibition of Inflammation Signaling by DNA Cox-2 "Decoy" Treatment Improves Motor Recovery and Attenuates Neuropathic Pain Behavior after Spinal Cord Injury in Rats by Neuroprotection and Microglial Inhibition.
**Claire Hulsebosch**, Professor and vice-chair, Department of Neuroscience, UTMB, USA. **J. Regino Perez-Polo**, Ph.D., I. H. Kempner Professor & Chair Dept. Biochemistry & Molecular Biology, UTMB, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Session 8: Neuro Sensory & Nervous System Development
## 13:00 PM – 15:30 PM
## Session Chair: Dr. Carole Samango-Sprouse

**13:00 – 13:30 PM**
**Presentation Title:** Plasticity in the auditory system: neuroscience evidence.
**Sridhar Krishnamurti**, Associate Professor of Audiology in the Department of Communication Disorders at Auburn University, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**13:30 – 14:00 PM**
**Presentation Title:** Neurodevelopmental Outcomes as Affected by Androgen Replacement and Family Learning Disabilities (FLD) in Common But Rarely Diagnosed Genetic Disorders.
**Carole Samango-Sprouse**, Ed.D., APIB, BNABS, and NDT Certified Neurodevelopmentalist, Director, Neurodevelopmental Diagnostic Center for Young Children, Associate Clinical Professor in the Department of Pediatrics at George Washington University, Washington, D.C., USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**14:00 – 14:30 PM**
**Presentation Title:** Embryonic stem cell-derived neurons as an in vitro model for investigating developmental neurotoxicity of environmental pollutants.
**Mohammed El Majdoubi**, PhD, Associate Professor of Neuroscience, Department of Natural Sciences and Mathematics, Dominican University of California, San Rafael, CA 94901, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**14:30 – 15:00 PM**
**Presentation Title:** Modeling circadian neural circuits: Emergent synchronization in populations of coupled phase oscillators.
**Pablo Gleiser**, Assistant Professor, Centro Atómico Bariloche, Instituto Balseiro, CONICET, Bariloche, 8400 Río Negro, Argentina.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**15:00 – 15:30 PM**
**Presentation Title:** The occipital lobe convexity sulci and gyri - Laboratory investigation.
**Raphael V. Alves**, MD, Neurosurgeon, Department of Neurosurgery, Hospital Beneficência Portuguesa, São Paulo, Brazil.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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## Session 9: Neurodegenerative Diseases-II
## 15:30 PM – 17:30 PM
## Session Chair: Dr. Gjumrakch Aliev

**15:30 – 16:00 PM**
**Presentation Title:** Autism: Theoretical Aspects.
**Helen V. Ratajczak**, PhD, Edmond Enterprises, LLC, in Danbury, Connecticut, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**16:00 – 16:30 PM**
**Presentation Title:** Deficient Repair of Genomic Damage as a Basis for Neurodegenerative Diseases Role of Transition Metals and the RNA-binding Protein TDP-43.
**Muralidhar L. Hegde**, Ph.D, Assistant Professor, Department of Neurology, Department of Biochemistry and Molecular Biology, University of Texas Medical Branch (UTMB), USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**16:30 – 17:00 PM**
**Presentation Title**: Mitochondrial Dependent Oxidative Stress Induced Cellular Hypoperfusion in Context of Neurodegeneration and Cancer.
**Gjumrakch Aliev**, MD&PhD, President "GALLY" International Biomedical Research Consulting LLC. San Antonio, TX, USA; Professor of Cardiovascular, Neuropathology and Gerontology. University of Atlanta, Atlanta, GA, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**17:00 – 17:30 PM**
**Presentation Title:** Multifunctional tacrine derivatives as potential agents to treat Alzheimer's disease.
**Praveen Rao Perampalli Nekkar**, B'Phm., M'Phm., PhD, Assistant Professor - Pharmaceutical Sciences PHR 5002, School of Pharmacy Health Sciences Campus, 200 University Ave W University of Waterloo, Waterloo, ON N2L 3G1, Canada.

**Q&A Session,** presenter answers questions from other speakers or attendees.

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discussion and networking. You can discuss what you want with other speakers. All speakers are unmuted, so speakers can talk freely during the session.

## Track 3: 8:00 AM – 17:30 PM, June 20, 2013

**8:00 – 8:30 AM**
Speakers and attendees can login the online conference.

## Session 10: Pain Management
## 8:30 AM – 10:30 AM
## Session Chair: Dr. Kamayni Agarwal-Kozlowski

**8:30 – 9:00 AM**
**Presentation Title:** Iron deposit- The possible pathogenesis in Central Post-Stroke pain?
**Gaiqing Wang**, Director, Department of Neurology, Xiangya Hospital, Central-South University, ChangSha, Hu Nan, China.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**9:00 – 9:30 AM**
**Presentation Title**: Continous percutaneous thoracic sympathetic chain blocks.
**Kamayni Agarwal-Kozlowski**, Head of the comprehensive center for palliative care and pain management, Elbe Kliniken Stade - Buxtehude GmbH, Stade, Germany.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**9:30 – 10:00 AM**
**Presentation Title:** The syndrome of new daily persistent headache.
**Matthew S. Robbins**, MD, Chief of Service, Einstein Division of Montefiore Medical Center, Director of Inpatient Services, Montefiore Headache Center, Assistant Professor of Neurology, Albert Einstein College of Medicine, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**10:00 – 10:30 AM**
**Presentation Title**: Neuropathic and myofascial pains.
**Hatem Samir**, Professor, Department of Neurology, Cairo University, Cairo, Egypt.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**Panel Discussion.** This session is to provide speakers and attendees with in-depth discussion and networking. You can discuss what you want with other speakers. All speakers are unmuted, so speakers can talk freely during the session.

## Session 11: Behavioral & Cognitive Neuroscience-I

## 10:30 AM – 14:00 PM
## Session Chair: Dr. Berit Brogaard and Dr. Kristian Marlow

**10:30 – 11:00 AM**
**Presentation Title**: The Long-Term Potentiation Model for Grapheme-Color Binding in Synesthesia.
**Berit Brogaard**, Professor of Philosophy and Psychology at the Center for Neurodynamics, University of Missouri, USA. **Kristian Marlow**, Center for Neurodynamics, University of Missouri, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**11:00 – 11:30 AM**
**Presentation Title:** Integrated neuroscience of bonding.
**Radovan Hruby**, MD, Ph.D., Faculty, Psychiatric Outpatient Clinic, Martin, Slovak Republic.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**11:30 – 12:00 PM**
**Presentation Title:** Consciousness concept from Rumi‘s view: fundamentalism or emergentism, a bridge between quantum theory and consciousness.
**Shahriar Ahmadpour**, Professor, Department of Anatomy, Medicine School, North Khorasan University of Medical Sciences (NKUMS), Bojnourd, Iran.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**12:00 – 12:30 PM**
**Presentation Title:** Targeting behavioral and psychological symptoms of dementia: comparison of antipsychotics and the novel compound, ADN-1184 in rat models.
**Adrian Newman-Tancredi**, Independent consultant & Chief Scientific Officer, NeuroAct Communication, 81100 Castres, France. Co-founder of a neuroscience-based new venture, Neurolixis Inc., France.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**12:30 – 13:00 PM**
**Presentation Title**: The role of pro-inflammatory cytokines in cancer related fatigue and depression.
**Miri Cohen**, Professor at the School of Social Work and Department of Gerontology, Faculty of Social Welfare and Health Sciences, University of Haifa, Israel.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**13:00 – 13:30 PM**
**Presentation Title:** Training the brain.

**Inge Wilms**, Ph.D., Center for Rehabilitation of Brain Injury, University of Copenhagen and Department of Psychology, University of Copenhagen, Denmark.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**13:30 – 14:00 PM**
**Presentation Title:** The 'biology of systems' or the 'systems of biology': looking at diabetes from a systemic perspective.
**Graham Ewing**, Chief Executive Officer and Director, Montague Healthcare, United Kingdom.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**Panel Discussion.** This session is to provide speakers and attendees with in-depth discussion and networking. You can discuss what you want with other speakers. All speakers are unmuted, so speakers can talk freely during the session.

## Session 12: Cellular & Molecular Neuroscience-IV
## 14:00 PM – 16:00 PM
## Session Chair: Dr. Narasimha Murthy

**14:00 – 14:30 PM**
**Presentation Title:** Maximizing outcomes in Glioblastoma.
**Michael E. Salacz**, MD, Medical Director, Saint Luke's Brain Tumor Center, Neuro-oncology Program, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**14:30 – 15:00 PM**
**Presentation Title:** Potential agents to Enhance the Expression of Neurotrophins in the Brain.
**Narasimha Murthy**, Associate Professor of Pharmaceutics at the University of Mississippi, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**15:00 – 15:30 PM**
**Presentation Title**: Rethinking the Traditional Western Blot.
**John Lyssand,** Field Application Scientist for LI-COR Biosciences in the New York City Metro area, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**15:30 – 16:00 PM**
**Presentation Title:** Vision loss during spine surgery.
**Ehab Farag**, M.D., FRCA, Associate Professor, Cleveland Clinic Lerner College of

Medicine, Outcomes Research, General Anesthesiology, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**Panel Discussion.** This session is to provide speakers and attendees with in-depth discussion and networking. You can discuss what you want with other speakers. All speakers are unmuted, so speakers can talk freely during the session.

## Session 13: Behavioral & Cognitive Neuroscience-II
## 16:00 PM – 17:30 PM
## Session Chair: Pending

**16:00 – 16:30 PM**
**Presentation Title:** Food vs Cigarettes: Neuroimaging Study of Obesity and Smoking.
**Laura E. Martin**, Associate Director of Fmri, Assistant Professor, Dept of Preventive Medicine and Public Health, Hoglund Brain Imaging Center, University of Kansas Medical Center, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**16:30 – 17:00 PM**
**Presentation Title**: Advanced uses of technology in the assessment of cognitive skills and learning.
**Joe Kush**, Associate Professor, Duquesne University, 327 Fisher Hall, 600 Forbes Avenue, Pittsburgh, PA, 15282, USA.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**17:00 – 17:30 PM**
**Presentation Title:** Genetic Essentialism and Neuroessentialism: How are people affected by learning of Personal Bioindicators?
**Ilan Dar-Nimrod**, Assistant Professor, The University of Sydney, Australia.

**Q&A Session,** presenter answers questions from other speakers or attendees.

**Panel Discussion.** This session is to provide speakers and attendees with in-depth discussion and networking. You can discuss what you want with other speakers. All speakers are unmuted, so speakers can talk freely during the session.

# Presentation Summaries (Alphabetical Order)

1 Adrian Newman-Tancredi
2 Alan Palmer
3 Ambuja Bale
4 Amotz Zahavi
5 Berit Brogaard
6 Carole Samango-Sprouse
7 Christina Pfister
8 Daniela Sabrina Andres
9 Dong (Dan) Y. Han
10 Ehab Farag
11 Erik Storkebaum
12 Gaiqing Wang
13 George N. Chaldakov
14 Gjumrakch Aliev
15 Graham Ewing
16 Hari Shanker Sharma
17 Hatem Samir
18 Helen V. Ratajczak
19 Helli Merica
20 Hideki Hayashi
21 Hussein Al-Wadei
22 Ilan Dar-Nimrod
23 Inge Wilms
24 Isabella Panfoli
25 J. Regino Perez-Polo
26 Joe Kush
27 John Lyssand
28 Kamayni Agarwal-Kozlowski
29 Laura E. Martin
30 Lester Ingber
31 Lisa A. Brenner
32 Magisetty Obulesu
33 Maria Carmen Portillo
34 Massimo Cocchi
35 Matthew S. Robbins
36 Meike Ramon
37 Michael E. Salacz
38 Miri Cohen
39 Mohammed El Majdoubi
40 Muralidhar L. Hegde
41 Narasimha Murthy
42 Nicole E Zander
43 Oleg Tolstenkov
44 Pablo F. Diez
45 Pablo Gleiser
46 Pawel Bodera
47 Peifang Tian
48 Praveen Rao Perampalli Nekkar
49 Radovan Hruby
50 Raphael V. Alves
51 Reem Hamdy A Mohammed
52 Rita Lo Vasco
53 Shahriar Ahmadpour
54 Sridhar Krishnamurti
55 Vuong Anh Duong

**1, Targeting behavioral and psychological symptoms of dementia: comparison of antipsychotics and the novel compound, ADN-1184 in rat models.**

*M. Kolaczkowski1, P. Mierzejewski2, P. Bienkowski2, A. Wesolowska3, A. Newman-Tancredi4. 1 Adamed Ltd., R&D, Pienkow, Poland. 2 Institute of Psychiatry and Neurology in Warsaw, Department of Pharmacology, Warsaw, Poland. 3 Jagiellonian University Collegium Medicum, Department of Clinical Pharmacy, Krakow, Poland. 4 NeuroAct Communication, Castres, France.*

**Summary:** As well as cognitive deficits, 60%-90% of dementia patients experience behavioral and psychological symptoms (BPSD), including psychotic symptoms (e.g. delusions and hallucinations), depression, aggressivity and anxiety. Antipsychotics are frequently prescribed but their therapeutic efficacy is limited and they can elicit metabolic and cardiac side-effects which are of particular concern in elderly patients. In addition, whilst current antipsychotics only poorly address the mood deficits observed in BPSD and may accentuate cognitive dysfunction. Surprisingly, comparative preclinical studies of the effects of antipsychotics in pharmacological models relevant to BPSD are remarkably scarce. We therefore compared the activity of 8 antipsychotic drugs (chlorpromazine, haloperidol, clozapine, olanzapine, risperidone, aripiprazole, lurasidone and asenapine) as well as a lead compound from Adamed's drug discovery program (ADN-1184) upon acute administration in rat models of psychosis (inhibition of MK-801-induced hyperactivity; Conditioned Avoidance Response, CAR), antidepressant-like activity (Forced Swim test, FST) and cognitive capacities (Passive Avoidance, PA). All the drugs exhibited activity in the MK-801 and CAR tests, although aripiprazole only poorly inhibited MK-801-induced hyperactivity. In the FST, whereas chlorpromazine was inactive, most of the other drugs modestly, but significantly, reduced immobility over narrow dose ranges. In contrast, clozapine and ADN-1184 reduced immobility over a wider dose range. The reduction in immobility achieved by the antipsychotics was about half of that elicited by imipramine, whereas the effect of ADN-1184 was more pronounced, reaching 80% of that of imipramine. In the PA test, all the compounds, including clozapine and lurasidone, but not aripiprazole or ADN-1184, impaired memory performance. The MEDs for these effects were, at best, only slightly greater than those for the MK-801 or CAR tests. Finally, AND 1184 did not elicit catalepsy or inhibit spontaneous locomotor activity at therapeutic-like doses. In conclusion, amongst the 8 clinically-available antipsychotics tested, none presents an ideal profile, i.e. activity in antipsychotic-like models, efficacy in antidepressant-like models, and freedom from detrimental effects on memory/cognitive performance. In contrast, ADN-1184 has a promising profile in these in vivo tests relevant to BPSD. Taken together, the present comparative study provides a database by which future drugs being considered for treatment of BPSD can be assessed and suggests that it is feasible to identify drugs that could improve behavioral and psychological symptoms without exacerbating cognitive function or movement impairment in already-fragile elderly patients.

**2, Translating neuroscience research into CNS drugs.**

*Alan Palmer, MS Therapeutics Ltd., UK.*

**Summary:** Translating neuroscience research into CNS drugs is challenging. Compared with non-CNS drugs, CNS medicines take longer to get to market and their attrition rate is greater. This is largely because of the complexity of the human brain, the liability of

CNS drugs to cause CNS side effects and the requirement of CNS medicines to cross the blood–CNS barrier (BCNSB). In this presentation, I will consider the factors that are important in translating neuroscience research into CNS medicines. Specifically, I will cover: (i) target identification, (ii) pharmacokinetics and pharmacodynamics properties of CNS drug candidates, (iii) experimental models of CNS disorders and (iv) demonstrating clinical efficacy for CNS drug candidates.

**3, N-Butanol-Induced developmental neurotoxicity in animals.**

*Ambuja Bale, National Center for Environmental Assessment, Office of Research and Development, USA.*

**Summary:** n-Butanol is a four-carbon straight chain alcohol that exhibits potentially developmental neurotoxic effects in experimental animals. Exposure potential to n-butanol is considerably high given the high production volume (>1 billion pounds) and consideration for use as a fuel oxygenate. n-Butanol is also approved for use in flavorings by the US FDA and can be found in paints and thinners. The n-butanol published literature on the mechanisms for developmental neurotoxicity in support of EPA's Integrated Risk Information System (IRIS) Program were reviewed and the information is very limited. In one oral dosing study in female dams (Sitarek et al., 1994), it was observed that rat pups exposed to n-butanol during gestation had increased incidences of dilation of the subarachnoid space and the third lateral ventricle in the brain. Another study with the chicken embryo (McLaughlin et al., 1964) reported significant developmental neurotoxicity concerns such as nerve damage and eye-related abnormalities. However, in other studies where rat pups were exposed gestationally to n-butanol from an oral dosing (Ema et al., 2005) or an inhalation dosing (Nelson et al., 1989) to dams, the pups did not exhibit any significant neuropathological changes. The available mechanistic data for n-butanol are consistent with ethanol, a known developmental neurotoxicant and support the developmental neurotoxicity observations in some of the studies. However, careful studies evaluating the neurobehavior of developing pups in sensitive strains, as well as further characterizing the plausible mechanisms involved, need to be conducted in order to further elucidate the neurodevelopmental effects with butanols for risk evaluation. (Disclaimer: The views expressed are those of the authors and do not necessarily represent the views or policies of the US EPA.)

**4. The evolution of acetylcholine and GABA from waste products to neurotransmitters.**

*Amotz Zahavi and Keith Harris, Department of Zoology, Tel Aviv University, Tel Aviv, Israel.*

**Summary:** The evolution of any signaling system presents a logical problem: before both the signaler and the receiver have evolved the complementary parts of the system, the system cannot function. We suggest that signal molecules originate as secondary metabolites that are secreted from a cell as a consequence of a particular metabolic change. Of the secreted chemicals, the chemical which is most noxious is the most suitable to function as a signal, as neighboring cells are more likely to respond to the presence of a more harmful chemical. We will discuss our application of this model to acetylcholine and GABA, neurotransmitters that are also paracrine signals. Acetylcholine

is secreted in response to Ca2+ influx and functions as a paracrine signal that coordinates Ca2+ mediated cell motor activities. GABA is secreted when Krebs cycle activity ceases, and functions as a paracrine signal that inhibits secretion and differentiation. We will also discuss how this model could apply to glutamate, and the possible direct damage glutamate could cause in the presence of oxygen.

**5, The Long-Term Potentiation Model for Grapheme-Color Binding in Synesthesia.**
*Berit Brogaard, Kristian Marlow, Philosophy and Psychology at the Center for Neurodynamics, University of Missouri, USA.*
**Summary:** The phenomenon of synesthesia has undergone an invigoration of research interest and empirical progress over the past decade. Studies investigating the cognitive mechanisms underlying synesthesia have yielded insight into neural processes behind such cognitive operations as attention, memory, spatial phenomenology and inter-modal processes. However, the structural and functional mechanisms underlying synesthesia still remain contentious and hypothetical. The first part of the talk reviews recent research on grapheme-color synesthesia, one of the most common forms of synesthesia, and addresses the ongoing debate concerning the role of selective attention in eliciting synesthetic experience. Drawing on conclusions of the first half, the talk's second half examines the various models proposed to explain the cognitive mechanisms behind grapheme-color synesthesia, and discusses the explanatory virtues of a new model suggesting that grapheme-color synesthesia is grounded in memory. The last part offers an examination of some of the broader philosophical implications of synesthesia.

**6, Neurodevelopmental Outcomes as Affected by Androgen Replacement and Family Learning Disabilities (FLD) in Common But Rarely Diagnosed Genetic Disorders.**
*Carole Samango-Sprouse, Neurodevelopmental Diagnostic Center for Young Children, Department of Pediatrics at George Washington University, Washington, D.C., USA.*
**Summary:** The effects of early androgen treatment on neurodevelopmental performance in pre-pubertal boys with 47, XXY have not been well investigated. The influence of hormones on brain development in humans suggests that a positive effect on neurodevelopmental outcome in young boys with XXY may be plausible with hormone replacement therapy. The aim of the study was to investigate retrospectively if an early course of androgen treatment (three injections of testosterone enanthate, 25mg, each) had an impact on specific domains of neurodevelopmental function in boys with 47, XXY at 36 and 72 months of age. 101 boys with a karyotype of 47, XXY had neurodevelopmental assessments. The retrospective chart review resulted in one group (n=34) who had received androgen treatment during infancy and the second group was untreated (N= 67). Statistical analysis was completed to determine if there was a positive effect from treatment observed at 36 months and at 72 months on multiple domains of development. There were significant differences in multiple cognitive domains in the group who received androgen treatment, including multiple measures of language, intellectual and neuromotor skills. Improved function was observed in neurodevelopmental outcome in boys with 47, XXY at 36 and 72 months who had been treated with a short course of androgen treatment in infancy. Continued research is underway to expand our understanding of the relationship of androgen, brain function

and neurobehavioral and neurodevelopmental outcome in boys with 47, XXY. Keywords: 47, XXY, androgens, sex chromosome disorders, chromosomal variations, Klinefelter syndrome, KS.

**7, Angiogenesis in meningiomas.**

*Christina Pfister, Department of Neurosurgery, University of Tuebingen, Hoppe-Seyler-Str. 3, 72076, Tuebingen, Germany.*

**Summary:** Meningiomas are the most common intracranial brain tumors. The primary therapy is microsurgical resection. Benign meningiomas tend to develop recurrences despite radical extirpation (10-30%). However until now there are no appropriate chemotherapeutical treatments for recurrent and malign meningiomas. Meningiomas are highly vascularized tumors and mostly exhibit a very low proliferation rate. Therefore the reduction of proliferation plays a tangential role in meningiomas in contrast to angiogenesis. A better understanding of angiogenesis in meningiomas could make is possible to control angiogenesis chemotherapeutically in order to influence for example tumor size. The vascular endothelial growth factor receptor 2 (VEGFR2) is in most meningiomas either expressed on a very low level or not detectable, while platelet-derived growth factor receptor beta (PDGFRβ) displays elevated levels compared with normal brain. Meningioma primary cell cultures, which were stimulated with exogenous VEGFA, displayed a significant higher proliferation rate. If PDGFRβ was inhibited before administration of exogenous VEGFA, this proliferative stimulus was abolished. Furthermore VEGFA induced concentration-dependent PDGFRβ tyrosine phosphorylation comparable to PDGFB-induced PDGFRβ tyrosine phosphorylation. Based on these results several tyrosine receptor inhibitors were tested (sunitinib, tandutinib, gambogic acid). Sunitinib, an already approved chemotherapeutic agent, which inhibits preverentially KDR, but also inhibits PDGFRβ, equally impaired migration of meningioma cells as tandutinib, which displays a high activity towards PDGFRβ but a low activity towards KDR. Moreover gambogic acid, an herbal substance, which shows significant stronger effect on tumor cells then normal cells in vitro, equally impaired cell migration of meningiomas and also suppressed VEGFA-induced PDGFRβ tyrosine phosphorylation. The low expression or absence of KDR in meningiomas combined with the elevated level of PDGFRβ, suggests that VEGFA primarily regulates VEGF-mediated migration and proliferation through PDGFRβ in meningiomas. Gambogic acid, tandutinib, and sunitinib were equally potent in inhibiting meningioma growth in vitro, suggesting that selective PDGFRβ inhibitors, such as gambogic acid and tandutinib, should be evaluated as a potential therapy for recurrent and malignant meningiomas, possibly in combination with VEGF inhibitors.

**8, Open questions and perspectives in DBS technology.**

*Daniela Sabrina Andres 1,2,3, Ruedi Stoop 1, 1. Institute of Neuroinformatics, UZH/ETHZ, Zurich, Switzerland; 2. Institute for Neurological Research Raul Carrea, Fleni Institute, Movement Disorders Section, Buenos Aires, Argentine; 3. Society in Science, The Branco-Weiss Fellowship, administered by ETH, Zurich, Switzerland.*

**Summary:** Deep Brain Stimulation (DBS) is therapy for the treatment of Parkinson's disease that has been successfully used for many decades. Today, clinical approaches are trying to extend the application of DBS for psychiatric and Basal Ganglia-related

neurological disorders like substance abuse, morbid obesity, obsessive-compulsive disorder and depression. However, due to the lack of an explanatory framework of the pathophysiology of the Basal Ganglia (BG) and DBS mechanisms of action, the procedures and protocols for DBS implementation are still not standardized. Many cardinal steps of the implantation surgery as well as the postoperative programming of the stimulation-device rely on individual expertise. A first direction of efforts to be spent in order to successfully extend the indications of this therapy, is automatic target identification during the implantation surgery, where microelectrode recordings will be used for the automatic identification of neurological structures and the selection of the best implantation site, using on-line implementation of computationally efficient algorithms. Efforts also have to go into postoperative programming, which will be guided by person-specific information collected from the patient during the exploratory phase of the surgery with microelectrode recordings. Finally, the present stimulation protocols will be developed towards closed-loop stimulation with demand-controlled stimulation. These issues are the ultimate challenges in DBS-technology development.

**9, Neuropsychiatric Presentation of Anti-NMDA-receptor Encephalitis.**

*Dong (Dan) Y. Han, UK Neuropsychology Service - Clinical Section, UK Multidisciplinary Concussion Program, Department of Neurology, Spinal Cord and Brain Injury Research Center, University of Kentucky College of Medicine, USA.*

**Summary:** Anti- N-methyl D-aspartate receptor encephalitis is an acute form of encephalitis, which is caused by an autoimmune reaction against NR1 and NR2 subunits of the glutamate NMDA receptor. Very little was known about this phenomenon prior to 2007, and since then it has been conceptualized as a rare form of encephalitis considered a paraneoplastic syndrome. However, more recent updates suggest higher prevalence in clinical settings, possibly beyond the clinical picture of paraneoplastic syndrome. Presented with severe acute psychosis, Anti-NMDA-receptor encephalitis is potentially lethal but there is high probability of recovery with early diagnosis and appropriate management. Presentation, diagnosis, and treatment processes are explored.

**10, Vision loss during spine surgery.**

*Ehab Farag, Cleveland Clinic Lerner College of Medicine, Outcomes Research, General Anesthesiology, USA.*

**Summary:** Post operative vision loss (POVL) after spine surgery in prone position is devastating but a rare condition. The recent publication of case control study of 83 cases of POVL and our study in the methods in reducing intraocular pressure during spine surgery in prone position have explained some of the contributing factors for POVL development. The aim of this presentation to present the most recent evidence based facts with regards the pathophysiology of and the methods for its prevention.

**11, Reduced protein translation rates in a Drosophila model for GARS-associated CMT.**

*Erik Storkebaum, Molecular Neurogenetics Laboratory, Max Planck Institute for Molecular Biomedicine, Germany.*

**Summary:** Charcot-Marie-Tooth (CMT) neuropathy is the most common inherited neuromuscular disorder, characterized by degeneration of peripheral motor and sensory

nerves, leading to progressive muscle weakness and sensory loss. Recently, dominant mutations in at least three tRNA synthetase genes have been identified as a genetic cause of CMT. The molecular pathogenesis of CMT in general, and of CMT associated with mutations in tRNA synthetases in particular, is poorly understood, and no effective therapies are available. We have generated Drosophila models for CMT caused by mutations in tyrosyl-tRNA synthetase (YARS) and glycyl-tRNA synthetase (GARS), both of which recapitulate several hallmarks of the human disease 1. Furthermore, we could demonstrate that loss of aminoacylation activity per se is not the cause of the disease. However, this does not exclude the possibility that subcellular mislocalization of mutant YARS and GARS proteins could induce defects in (local) protein translation. Therefore, we have studied the subcellular localization of mutant YARS and GARS proteins in motor neurons in our Drosophila CMT model. Furthermore, we have used a novel metabolic labeling technique to study protein translation rates in motor neurons in vivo. Although CMT-associated mutations did not alter the subcellular localization of YARS and GARS proteins, mutant GARS expression in motor neurons resulted in reduced protein translation rates. Possible underlying mechanisms and implications of these finding will be discussed. References:Storkebaum, E., et al. Dominant mutations in the tyrosyl-tRNA synthetase gene recapitulate in Drosophila features of human Charcot-Marie-Tooth neuropathy. Proc Natl Acad Sci U S A 106, 11782-11787 (2009).

**12, Iron deposit- The possible pathogenesis in Central Post-Stroke pain?**

*Gaiqing Wang*,Hongping Zhao, Weimin Hu and Fang Xue, Department of Neurology, Xiangya Hospital, Central-South University, ChangSha, Hu Nan, China.*

**Summary:** The etiology of central post-stroke pain (CPSP) is poorly understood and such pains are often refractory to treatment. We experienced an 58-year-old woman, who, following a right posterior internal capsula hemorrhage, suffered from a left insufferable and inexpressible swollen and burning pain, this was accompanied with severe tactile allodynia and chill. This was associated with tactile hypoaesthesia, hyperalgesia and proprioceptive deficiency on her left side. A year later, Compared to ICH without CPSP which magnetic resonance image (MRI) showed that low signal intensity on T2-weighted images in the right lesion near to posterior horn of lateral cerebroventricular region. Therefore, we speculate iron(ferritin or hemosiderin) may take part in the theories of central pain generation. Key Words: Central post-stroke pain(CPSP); etiology; iron deposit; magnetic resonance image(MRI).

**13, Adipose tissue: a master in toxicology.**

*George N. Chaldakov, Laboratory of Cell Biology, Medical University, Varna, Bulgaria, Institute of Cellular Biology and Neurobiology, National Research Council, Rome, Italy and Department of Anatomy and Histology, Medical University, Varna, Bulgaria.*

**Summary:** Conventional wisdom in the pathogenesis of obesity and related cardiometabolic, malignant and neurodegenerative diseases focuses mainly on genetic predisposition and lifestyle (high caloric foods, sedentary lifestyle, smoking). The human genome project's big promise was that it could improve our understanding of the pathogenesis and therapy of diseases. However, the genes have been found to account for only about 10% of diseases, and the remaining causes appear to be from environmental exposures, hence the exposure science emerges. Note that molecular epidemiology and

toxicology may be essential partners of exposure science. Indeed, Homo sapiens recens is exposed to an overwhelming number of chemical contaminants circulating every day in the air, water, food, and general environment. The body is a well-equipped entity with capabilities to excrete watersoluble pollutants, but not as well-equipped to excrete some of the lipid-soluble xenobiotics. In the late 1990's, according to the European Environmental Agency more than 100 000 chemical compounds were registered in the European Catalogue of Commercialized Chemical Substances. Here we present data that adipose tissue may be an important participant in the environmental molecular toxicology. The discovery of adipocyte-secreted leptin in 1994 was a paradigm shift event in the study of adipose tissue. It was applauded by scientific community and thus triggered a new direction in the evaluation of endocrine function of adipose tissue, that is, adipoendocrinology. This is why the today's adipose tissue is viewed not merely as a lipid storage, but also as a dynamic secretory – endocrine and paracrine – organ, synthesizing, storing, and releasing a dazzling number of signaling proteins collectively termed adipokines. Numerous evidence demonstrates that the exposure to persistent organic pollutants (POP) may contribute to the pathogenesis of obesity and its related diseases. Noteworthy, these pollutants accumulate mainly in the adipose tissue. And xenobiotic-metabolizing cytochromes p450 (CYP) are expressed in adipose tissue, where CYP 1A1 and CYP 1B1 can bioactivate carcinogenic polycyclic aromatic hydrocarbons and xenoestrogens. Altogether, the present review highlights an adipocentric approach in molecular toxicology. It is conceptualized as adipotoxicology, that is, the study of accumulation, metabolism, and release of xenobiotics in adipose tissue in health and disease. In effect, the adipose tissue may be a new bridge between environment and health - let us call it a master in toxicology. Key words: adipobiology, adipokines, adipotoxicology, exposome, persistent organic pollutants, xenobiotics.

**14, Mitochondrial Dependent Oxidative Stress Induced Cellular Hypoperfusion in Context of Neurodegeneration and Cancer.**

*Gjumrakch Aliev, "GALLY" International Biomedical Research Consulting LLC. San Antonio, TX, USA; Cardiovascular, Neuropathology and Gerontology. University of Atlanta, Atlanta, GA, USA.*

**Summary:** Neurodegeneration [Stroke and Alzheimer disease (AD)] and cancer are fast becoming one of the leading causes of age-associated disability, dementia, and death. In addition, the Centers for Disease Control and Prevention (CDC) and the National Center for Health Statistics recently reported that AD has surpassed diabetes as a leading cause of death and is now considered the sixth-leading cause of death in the United States. Oxidative stress induced mitochondrial DNA overproliferation and/or deletion of the organ and/or tissues, especially the mitochondrial energy demands, have been implicated in the pathogenesis of several diseases, including AD, tumor growth, and metastasis. Decline in mitochondrial function during the development and maturation of the neurodegeneration, tumor growth, and metastases characterizing the tissue oxygen deficiency may lead to cellular energy defects, which will compensate vital cellular components and regulators. The overexpression of the enzymes such as NOS induce the production of unwanted large amounts of free radicals that cause the oxidative stress, cellular change, and particularly the concomitant mitochondrial lesions and decline in normal organ function. The present study has determined if an intimate, i.e. causal,

relationship between oxidative stress and mitochondrial damage and/or vascular lesions occurs before the development of human AD, in animal models that mimic human neurodegeneration and human colorectal carcinoid cancer or malignant brain cancer. In situ hybridization and ultrastructural analysis of the mitochondria (mitochondria with electron dense matrix, mitochondrial-derived lysosomes) showed that mitochondria with the abnormal structures and lipofuscin appear to be features of hippocampal damaged neurons in human AD, aged Tg (+) mice, 2 vessel occlusion model of the brain hypoperfusion, and malignant primary and metastatic cancer. The abnormal mitochondria appeared to be a permanent feature in all cellular compartments; in situ hybridization analysis with mouse and human mtDNA probes found a large amount of deleted mtDNA in human AD and in all models that mimic human AD (mice, rats etc.) hippocampus and cancer tissues compared to aged controls. The majority of these mtDNA deletions were found in mitochondrial-derived lysosomes in regions closely associated with lipofuscin and/or tumor growth regions, and suggests that proliferation, deletion, and duplication of mtDNA occurs in mitochondria, many of which have been fused with lysosomes in human AD, Tg(+) mice, and malignant tumors. Moreover, the biopsy and perfused brain samples from AD and the animals' models that mimics human AD as well as cancer patients were dominated by abnormal mitochondria as compared to a control group. In situ hybridization with a chimeric cDNA probe for the 5kb common deletion indicated that the 5kb mtDNA is increased at least 3 and 4 fold respectively in AD and malignant tumor cases as compared to controls. In quantitative analysis of the mtDNA deletion and 8OHG in the same cases, we found a strong significant positive correlation (r=0.934). Only hippocampal and cortical vulnerable neurons as well as malignant cancer tissues showed immunopositive staining for RNA oxidation markers visualized by using 8-OHG-staining, NOSs, and all oxidative stress markers. The mitochondrial DNA overproliferation and deletion detected by using cytological techniques suggests that successful dysregulation of the cell cycle is also the hallmark of neoplasm; early mitochondrial dependent cell-cycle pathophysiology in AD may recruit oncogenic signal transduction mechanisms and hence, can be viewed as an abortive neoplastic transformation. This observation indicates that the oxidative stress markers seen in the AD brain and malignant cancer selectively affects the population of vulnerable neurons, vascular EC, and perivascular cells, suggesting that oxidative stress induced mitochondrial DNA overproliferation and/or deletion plays a key role in the pathogenesis of AD and cancer. The common features on the mitochondrial abnormality were seen on the brain during tumorigenesis and AD indicating that mitochondrial DNA overproliferation and/or deletion are the key initiating factors for development, maturation, and progression of neurodegeneration as well as tumor growth and/or metastases. By using electron microscopic techniques we have found that the mitochondrial lesions appeared to be the primary hallmark of the glioblastoma. Vessel endothelium from tumor tissues shows the damage of mitochondrion cristae. The mitochondria derived lysosomes appeared to be permanent feature of the glial cells derived tumor cells. The lipid laden tumor cells and surrounding cells often show a different degree of mitochondria abnormality (such as mitochondria with broken cristae, presence of edema in their matrix, disruption of inner and external mitochondrial membrane). Moreover, giant mitochondria also appeared to be permanent features of tumor growth and metastases. Comparative characteristics of marginal and central

portion of tumor tissues obtained from patients undergoing surgery with diagnosis of the primary glioblastoma showed that distance area of tumor tissue characterized heterogeneous distribution of damage in the structure of the mitochondria. Central regions of tumor tissues in almost all of area shows astrocytes with clusters of mitochondria derived lysosomes. The same patterns of cellular and subcellular damage were seen in spinal cord tumor. One of the big challenges for treatment of neurodegenerative diseases and cancer appeared to be delivering drugs into the injury affected tissues. Our future studies are aimed to show that injection of silver nanoparticles in the brain lead to leaking on the inter endothelial contact and luminal plasma membrane, and therefore elucidate the possibility of penetrating into the cerebrovascular, neuronal, and glial cell which are especially damaged in AD and/or brain cancer. Our clinical study showed the preservation and improvement of cognitive tasks in depressed and demented patients after 24, 36 and 60 month follow up of combined pharmacological (especially the combination of the diseases and mitochondrion specific compounds) and non- pharmacological treatment. The study group consisted of 156 medically ill and physically disabled patients with mild to moderate dementia and depression. Patients were treated with antidepressants, cholinesterase inhibitors, and NMDA antagonists, along with their regular medication regimen. Non-pharmacological intervention was centered on a home-based program of physical and cognitive exercises as well as with vitamins and supplements (multivitamins, vitamin E, L-methylfolate, alpha-lipoic acid, acetyl-L-carnitine, omega-3, and coenzyme Q-10) and diet modification. Cognitive assessments were performed yearly. After 60 months of treatment, performance of all tasks remained at or above baseline. The MMSE, Cognistat–Attention, Cognistat–Judgment, and RFFT - Total Unique Designs demonstrated significant improvement. Our results also demonstrate the arrest in cognitive decline in demented/depressed patients with multiple medical co-morbidities for 60 months. Our study, for the first time, demonstrated the pattern of oxidative stress induced mitochondrial DNA overproliferation and/or deletion as well as mitochondrial enzyme activities during the development of human AD, and animals that mimic human AD, colorectal cancer in liver metastasis, and malignant brain cancers. We conclude that mitochondrial lesions, especially mitochondrial DNA abnormalities, are responsible for cell viability which can be used as new diagnostic tools and/or criteria for the earlier detection of diseases and future considerations for this approach will enable us to open new pathways, not only for the better understanding of BBB homeostasis which most likely plays a key role in the development of AD, but also for the development of new and more specific treatment strategies that will be more powerful and effective in bringing a cure for this devastating disease. Thus, our research involving the conjugation of the silver nanoparticles with mitochondrially-specific drugs would help to diminish the lesions that occur in AD and/or tumor tissues. Future investigations addressing the application of a combined, integrative treatment models in clinical practices are warranted. Acknowledgements: This study was partially supported by “GALLY” International Biomedical Research Consulting LLC, San Antonio, TX, USA.

**15, The 'biology of systems' or the 'systems of biology': looking at diabetes from a systemic perspective.**

*Graham Ewing, Montague Healthcare, United Kingdom.*

**Summary:** Background- The body is an open dynamic organism which responds to sensory and biological input. It may be studied from the bottom upwards or the top downwards however the biological bottom-up approach invariably ignores that sensory input influences the body's regula-tory mechanism and/or that environmental stressors of different nature and/or intensity influence the body's stability, alter the cellular re-sponses, activate or deactivate genes, and result in the onset of pathologies such as diabetes mellitus which affect the body's function and capacity. Accordingly, the major aim of this article is to highlight the fundamental role which the top-down cognitive approach has upon the understanding of the body's function i.e. as a measure of autonomic dysfunction and psychological stress; and to compare such approach with the bottom-up approach so favoured by biomedical researchers which avoids considering and/or largely dismisses the considerable influence of sensory input upon cellular and molecular biology. The method used in this article is based upon an appreciation that the brain regulates the autonomic nervous system and physiological systems, and that autonomic dysfunction leads to alterations at the cellular and molecular level i.e. to genotype and phenotype. Such un-derstanding, incorporated into a mathematical model and commercialized technology, is highly significant for a number of reasons. In par-ticular that (i) Diabetes is a multi-systemic disorder; (ii) type 1 Diabetes is genotypic and type 2 Diabetes is phenotypic; (iii) most Diabetes is a combination of genotype and phenotype; and (iv) type 2 Diabetes is an issue of acidity which influences the prevailing intracellular levels of essential minerals Magnesium and bioavailability of Zinc. In conclusion the term top-down systems biology more precisely refers to the role of sensory input which influences the brain's efforts to regulate the autonomic nervous system and physiological systems i.e. the regulation of acidity is a neurally regulated physiological system. The term 'top-down' encompasses the cognitive and/or psychological approach whilst the term 'bottom-up' is solely that of the biological approach. Moreover the most effective way of stimulating the autonomic nervous system and reducing pH (intracellular acidity) is by the top-down approach involving lifestyle interventions e.g. abstaining from acidic drinks, maintaining normal levels of body weight, avoiding stress, and exercise. Keywords- diabetes, autonomic nervous system, physiological systems, genotype, phenotype, proteins, blood glucose.

**16, CNS Injury and Repair. Novel Therapeutic Strategies and Nanodrug Delivery.**

*Hari S Sharma1, Dafin F Muresanu2, Hongyun Huang3, Chen Lin4, Aruna Sharma1 1Laboratory of Cerebrovascular Research, Dept. of Surgical Sciences, Anesthesiology & Intensive Care Medicine, University Hospital, Uppsala University, SE-75185 UPPSALA, Sweden. 2Deprtment of Clinical Neurosciences, University Hospital, University of Medicine & Pharmacy, Cluj-Napoca, Romania. 3Department of Neurosurgery, Beijing Rehabilitation Center, Neuroscience Institute of Taishan Medical University, Beijing, 100144,China. 4Department of Neurosurgery, Beijing Xishan Hospital, Beijing Rehabilitation Center, Xi xia zhuang, Ba da chu, Shijingshan District, Beijing 100144, China.*

**Summary:** Recent advances in CNS injury require novel therapeutic measures to attain neuroprotection and/or neuronal recovery to enhance quality of life of patients. In this connection, stem cell therapy, novel drugs, antibodies and nanodrug delivery is employed to achieve maximum neuroprotection in time. However, our knowledge regarding a

combination of various kinds of CNS injury and treatment strategies are still not well defined. As a result, several drugs or stem cell therapy is not that effective to cure the disease particularly in the long run. For example CNS injury occurring under situations of environmental pollution, exposure to various nanoparticles from environment e.g., SiO2 dust, missile explosion emanating Carbon, sulphur, Cu and Ag nanoparticles, biological warfare exposing soldiers to small sized bacteria or viruses and other hazardous substances could complicate the pathophysiology of CNS injury. In addition, internal disease factors viz. hypertension, diabetes, abnormal hormonal or metabolic disturbances could also alter the pathophysiology of brain injury and repair processes. Thus, under such conditions novel therapeutic strategies are required to achieve good neuroprotection. Our laboratory has initiated novel therapeutic strategies using nano delivery of drugs, stem cells and antibodies to treat CNS injuries in combination with nanoparticles exposure and/or with different co-morbidity factors. Our observations showed that nano delivery of multimodal drug e.g., cerebrolysin significantly enhances neurorestoration and neurorecovery in CNS injury in relation to nanoparticles exposure or in combination with different co-morbidity factors. Also in our laboratory nanodelivery of stem cells enhanced the therapeutic efficacy of neurorecovery in different models of CNS injuries. Furthermore, nanodelivery of antibodies directed against different neurochemicals or enzymes also improved neurological functions and neurorestoration in several experimental models of CNS trauma or insults. Taken together our observations open new avenues of therapeutic strategies to treat CNS dysfunction. The possible mechanism and significance of our findings are discussed. *Supported by Swedish Medical Research Council (nr 2710 HSS), Ministry of Biotechnology, Govt. of India (AS); Indo-EU Research Collaboration Program in Neuroscience (AS); European Office of Aerospace Research & Development (EOARD) London Office, London, UK (HSS/AS); Wright Patterson Air Force Research Laboratory (WPAFB), Dayton, OH, USA (HSS/AS), and Laerdal Foundation of Acute Medicine, Stavanger, Norway (HSS).

**17, Neuropathic and myofascial pains.**

*Hatem Samir, Department of Neurology, Cairo University, Cairo, Egypt.*

**Summary:** Pending.

**18, Autism: Theoretical Aspects.**

*Helen V. Ratajczak, Edmond Enterprises, LLC, in Danbury, Connecticut, USA.*

**Summary:** Autism is an epidemic throughout the world, and, is now estimated to occur in 1/50 children in the United States. In 1943, when first described by Kanner, the incidence was 1/10,000. The disorder is diagnosed by a trained investigator and defined by a pattern of difficulties across several areas including communication, social interaction, and behavior. Each subject is unique in presentation of symptoms. There is a lack of any independent biological or phychological markers. Autism is considered to have a genetic basis, with a synergy of environmental factors which overwhelm the child's innate immune system and biological balance, resulting in a wide variety of symptoms. The emphasis of this presentation is on theoretical aspects of autism: shared biologic and pathologic signs, systems of the body which are involved in autism, and biomarkers which are present in amounts significantly different from those of neurotypical individuals. An hypothesis is described in which biomarkers from each of

the systems of the body in which autism is manifest are measured and their circadian rhythm defined. The measurements will be compared to those from neurotypical individuals matched for age, sex, ethnicity, and place of residence. Multiple regression and discriminate analyses can be used to create a panel of the biomarkers, ranked according to amount of difference from the controls. Thus a subject-specific profile will be produced, which, after validation of the data, might offer a guide for effective therapy.

**19, Mathematical modeling of sleep structure.**
*Helli Merica, Laboratoire de Sommeil et de Neurophysiologie, Hôpitaux Universitaires de Genève, Belle Idée, Geneva, Switzerland.*
**Summary:** Pending.

**20, A protective role of apolipoprotein E-containing lipoproteins in glutamate-induced neurodegeneration.**
*Hideki Hayashi, Department of Ophthalmology, Faculty of Life Sciences, Kumamoto University, Japan.*
**Summary:** Lipoproteins in the central nervous system are secreted from glia, mainly from astrocytes, and are present in the form of high density lipoprotein-like particles. Apolipoprotein E is a major apolipoprotein in the central nervous system. It has been reported that cholesterol associated with apolipoprotein E-containing lipoproteins (E-LPs) stimulates synaptogenesis in retinal ganglion cells (RGCs). We have also demonstrated that glia-derived E-LPs promote axon elongation of RGCs mediated by receptor(s) of the low density lipoprotein receptor (LDLr) family. Here, we show that E-LPs protect RGCs from Ca2+-dependent and mitochondrion-mediated apoptosis induced by glutamate. Binding of E-LPs to LDLr-related protein-1 (LRP1) recruited the N-methyl-D-aspartate receptor, and interaction of these receptors blocked Ca2+ influx thereby inhibiting apoptosis. E-LPs protect RGCs from glutamate-induced neurodegeneration in vivo. Intravitreal administration of E-LPs prevented degeneration of RGCs in glutamate-aspartate transporter-deficient mice, an animal model of glaucoma. These results indicate that E-LPs have a neuroprotective role against glutamate-induced degeneration in vitro and in vivo and may provide potential therapeutic targets for glaucoma.

**21, Presentation Title: Pending.**
*Hussein Al-Wadei, College of Veterinary Medicine, USA.*
**Summary:** Pending.

**22, Genetic Essentialism and Neuroessentialism: How are people affected by learning of Personal Bioindicators?**
*Ilan Dar-Nimrod, The University of Sydney, Australia.*
**Summary:** Much research indicates that when people learn of a genetic or neurological potential cause of a disease or condition (termed genetic essentialism or neuroessentialism respectively) they view the outcome as more deterministic, immutable, of specific etiology and natural. In this light, increased accessibility of direct-to-consumer (DTC) personalized genetic reports raises an important question- how are people affected by information about their own genetic predispositions? In the first complete randomized

experiment on this question participants were led to believe that they had entered a study on the genetics of alcoholism and sleep disorders. Participants provided a saliva sample purportedly to be tested for the presence of relevant genetic susceptibility. While they waited for the results of these tests, they completed a number of measures including their emotional state. After a delay, participants received a bogus report about their genetic susceptibility and they again completed the questionnaire about emotional state as well as items assessing perceived control over drinking, relevant future drinking-related intentions, and intervention-related motivation and behavior. The results indicated that participants who were led to believe they had a gene associated with alcoholism showed an increase in negative affect, decrease in positive affect, and reduced perceived personal control over drinking. Although reported intentions for near-future alcohol consumption were not affected by the experimental manipulation, participants were more likely to enroll in a drinking intervention after learning of their alleged genetic susceptibility to alcoholism. This slew of psychological and behavioral effects of receiving personalized genetic susceptibility information is arguably similar to effects of neurological etiological claims as well. It demonstrates some of the potential perils (and benefits) of DTC genetic tests as well as potential imaging results.

**23, Training the brain.**

*Inge Wilms, Center for Rehabilitation of Brain Injury, University of Copenhagen and Department of Psychology, University of Copenhagen, Denmark.*

**Summary:** Is it possible to improve cognitive functions using computer based training? Currently, there is a growing industry of iPad and iPhone based brain training promising improved cognitive functions in healthy as well as injured populations. There are huge benefits for the society as a whole and for the individual in particular in being able to postpone the onset of cognitive decline. Quality of life, independency and the ability to provide for oneself directly depend on cognitive abilities being more or less intact. However, as with most things in life, benefits don't come easy. Although attempts have been made to create effective computer based training since the general advent of computers in the sixties, positive results in terms of cognitive improvements so far seem scarce. There are several reasons for this and in this presentation I will try to outline some of the current obstacles for success and attempt a proposal for a way forward. Using results from my own research, I will discuss the difficulties of gaining a general, cognitive improvement from specific training (the generalization effect) and the challenges translating clinical practise into computer based training. Finally, I will briefly touch upon the challenges facing researchers trying to conduct efficacy research.

**24, New insights in neurodegeneration: Extra-mitochondrial oxidative phosphorylation in nervous system.**

*Isabella Panfoli*, Silvia Ravera*, Daniela Calzia*, Martina Bartolucci*, Angelo Schenone†, Carlo Enrico Traverso†, Gianluigi Mancardi† *DIFAR, University of Genoa, Italy; †DINOGMI, University of Genoa, Italy.*

**Summary:** Retinal outer segments (OS) and brain, great oxygen consumer tissues display few mitochondria but suffer from oxygen deprivation. Evidence is gathering on an extra-mitochondrial oxidative phosphorylation (OXPHOS) in rod outer segments (OS), isolated myelin vesicles (IMV) and plasma membranes, that are devoid of

mitochondria.OS express functional ETC and ATP synthase, as well as Tricarboxylic Acid Cycle enzymes. IMV from CNS (bovine forebrain) and PNS (sciatic nerve) perform and extra-mitochondrial OXPHOS. O2 consumption was impaired in IMV from sciatic nerve of Charcot-Marie-Tooth type 1A rats, model of a dys/demyelinating disease, with respect to controls, even though OXPHOS proteins were still present. Here we show that OS consume oxygen (O2) and synthesize ATP in the presence of substrates not accessible to mitochondria. Immunofluorescence CLSM and TEM of bovine retinal sections suggest an aerobic ATP supply for visual transduction. The ability of OS to manipulate O2 sheds light on the pathogenesis of many retinal degenerative diseases ascribed to oxidative stress. We also report the functionality of all the TCA cycle enzymes in bovine IMV, and a study on human brain white matter (NAWM) and Multiple Sclerosis (MS) plaques. OXPHOS proteins in IMV from MS plaques were impaired with respect to NAWM, proportionally to the severity of the lesion. If myelin is a contributor in energy supply for the axon, its loss would cause an energetic imbalance of the axon and the progressive axonal transection. Such vision may shed a new light on demyelinating pathologies. ATP synthase of both OS and IMV is the target of ATP synthase inhibitors, suggesting a specific role of these antioxidants in neurodegenerative disease prevention and onset delay.

**25, Inhibition of Inflammation Signaling by DNA Cox-2 "Decoy" Treatment Improves Motor Recovery and Attenuates Neuropathic Pain Behavior after Spinal Cord Injury in Rats by Neuroprotection and Microglial Inhibition.**

G.-Y. XU1, *K. M. JOHNSON1, G. C. UNABIA1, C. E. HULSEBOSCH1 and J. PEREZ-POLO2 1Neuroscience and Cell Biology, Biochemistry and Molecular Biology. University of Texas Medical Branch, Galveston, Texas, USA.*

**Summary:** Spinal cord injury (SCI) results in a number of deficits and triggers primary and secondary injury signaling cascades characterized by an early and prolonged inflammatory response. Early after SCI, IL-1B increases, an inflammatory agent, that triggers increased activation of the transcription factor Nuclear Factor-kB (NF-kB). NF-kB mediates secondary injuries via regulation of synthesis of proteins that are detrimental to the recovery process and can maintain inflammation in the injured spinal cord. There are different NF-kB subunits and subunit-specific inhibition can be accomplished with synthetic double stranded "decoy" deoxyoligonucleotides containing selective NF-kB protein dimer binding consensus sequences. In this project, DNA "decoys" target the COX-2 gene promoter NF-kB binding site, attenuate the SCI-induced increases in the levels of COX-2 and iNOS protein levels and significantly decrease cell death and key molecules in inflammatory signaling. Spinal cord contusion injury and outcomes were measured as described in Experimental Design below. The results demonstrate a significant improvement in locomotor scores, mechanical allodynia (for both cutaneous and pressure), thermal hyperalgesia and improved clinical measures (weight and bladder) in the group treated with COX-2 decoys compared to the vehicle group. Decoy treatment also resulted in significant reductions in COX-2 and iNOS expression following SCI and neuronal rescue. These experiments demonstrate the efficacy of novel interventions in the inflammatory cascade triggered by SCI as a strategy for treatment of SCI-induced physiological functional impairments. The approach is innovative in that it assesses the use of a new technology (DNA promoter decoys) to selectively block injury response

mechanisms that can result in neuropathy.

**26, Advanced uses of technology in the assessment of cognitive skills and learning.**
*Joe Kush, Duquesne University, 327 Fisher Hall, 600 Forbes Avenue, Pittsburgh, PA, 15282, USA.*
**Summary:** There is growing evidence that the speed at which a problem can be solved, without the use of a cognitive strategy, is related to overall intellectual ability. Empirical data used to support this relationship between the speed of basic processing and intellectual functioning has been evidenced primarily by correlations between measures of general intelligence and measures of choice reaction time (CRT). These measures allow individuals to make a forced-choice discrimination task that typically occurs in milliseconds. In most CRT studies, the stimuli consist of a pi-figure where subjects are instructed to select the side of the figure that contained the significantly longer leg. To prevent storage in iconic memory, a backward mask consisting of longer and wider bars is placed over the vertical lines immediately after the presentation. The introduction of the mask has, however, allowed some subjects to report that they are able to use the apparent movement of the mask as a cognitive strategy to facilitate their performance. The goal of the current study was to attempt to introduce an alternative stimuli mask to the CRT paradigm that would hopefully minimize the influence of movement cues. Results indicated no difference in the amount of time to process either the widely adopted pi-based stimuli and mask and a modified letter-based, stimuli and mask. The current computer-based alternative offers researchers who suspect that this movement strategy might be present, an alternative methodology that will likely minimize the use of any cognitive strategy without an accompanying increase in the amount of time to solve the task.

**27, Rethinking the Traditional Western Blot.**
*John Lyssand, Field Application Scientist for LI-COR Biosciences, USA.*
**Summary:** Traditional Western blotting is a labor-intensive process that includes gel electrophoresis, protein transfer to a blotting membrane, incubation with primary and secondary antibodies, and chemiluminescent or fluorescent detection of target proteins. Day-to-day reproducibility is poor, because small variations in lysate preparation, gel loading, electrophoresis, transfer, and detection are unavoidable sources of technical variability. The In-Cell Western™ (ICW) Assay, a quantitative immuno-fluorescent method, is an alternative to traditional Western blots that increases both reproducibility and sample throughput. Here we describe the In-Cell Western Assay and its use in neuroscience research. Cells are cultured and treated in microplates. After treatment, cells are fixed and permeabilized, blocked, and incubated with antibodies for detection of target proteins. Fluorophore-coupled secondary antibodies and detection with a LI-COR® Odyssey® Infrared Imaging System enable superior sensitivity, accurate quantitation, and easy normalization to a loading control. The traditional Western blot protocol is streamlined, eliminating cell lysis, gel electrophoresis, and membrane transfer. The In-Cell Western Assay enables screening and analysis of many more samples in each experiment, eliminates error-prone protocol steps, and delivers higher reproducibility for biological and technical replicates. The data presented will demonstrate how ICW assays are used in Alzheimer's Disease research to screen HSP90 inhibitors for their

effectiveness in reducing tau activity levels. We will discuss how and why the In-Cell Western Assay is superior to traditional methods for screening of cell samples.

**28, Continous percutaneous thoracic sympathetic chain blocks.**
*Kamayni Agarwal-Kozlowski, the comprehensive center for palliative care and pain management, Elbe Kliniken Stade - Buxtehude GmbH, Stade, Germany.*
**Summary:** Pending.

**29, Food vs Cigarettes: Neuroimaging Study of Obesity and Smoking.**
*Laura E. Martin, Fmri, Assistant Professor, Dept of Preventive Medicine and Public Health, Hoglund Brain Imaging Center, University of Kansas Medical Center, USA.*
**Summary:** Neuroimaging studies have consistently demonstrated differences in reward processing brain regions when comparing addicted individuals to non-addicted individuals. Moreover, obese individuals have shown similar differences in reward processing brain regions. However, few studies have directly examined the impact of obesity on addiction related brain responses. The current research used functional magnetic resonance imaging (fMRI) to examine the neural systems of reward processing in response to food and smoking cues in HW to overweight/obese smokers to investigate how co-morbidities may impact reward processing. Seventeen healthy weight (Body Mass Index [BMI] 18.5 - 25 kg/m2) and 15 overweight/obese (BMI > 25kg/m2) smokers were enrolled and included in the fMRI analysis. Participants completed two scans while passively viewing food and animal pictures and two scans while passively viewing pictures of people smoking and engaged in daily activities. Smokers showed the expected pattern of activation to food images with increased responses in medial prefrontal cortex (MPFC) to food compared to nonfood images. Moroever, all smokers showed activation in the MPFC when viewing smoking compared to non-smoking cues. The number of cigarettes smoked per day correlated with prefrontal activations (r=.46, p<.01). However, no group differences were found between healthy weight and overweight/obese participants while viewing food or smoking images. Overall the results showed increased activations in reward areas to food cues and smoking cues among smokers. Comorbitiy of smoking and obesity did influence response to smoking and food cues more than smoking alone. Further studies are needed to understand the impact of these results on treatment, however, the current results indicate that treatments focused on smoking cessation may be an important first step in treatment.

**30, Electroencephalographic field influence on calcium momentum waves.**
*Lester Ingber, Lester Ingber Research, USA.*
**Summary:** Macroscopic electroencephalographic (EEG) fields can be an explicit top-down neocortical mechanism that directly drives bottom-up processes that describe memory, attention, etc. The top-down mechanism considered are macrocolumnar EEG firings in neocortex, as described by a statistical mechanics of neocortical interactions (SMNI), developed as a magnetic vector potential $bold A$. The bottom-up process considered are ${roman Ca} sup {2+}$ waves prominent in synaptic and extracellar processes that are considered to greatly influence neuronal firings. Here, the complimentary effects are considered, i.e., the influence of $bold A$ on ${roman Ca} sup {2+}$ momentum, $bold p$. The canonical momentum of a charged particle in an

electromagnetic field, $bold PI = bold p + q bold A$ (SI units), is calculated, where the charge of ${roman Ca} sup {2+}$ is $q = - 2 e$, $e$ is the magnitude of the charge of an electron. Calculations demonstrate that macroscopic EEG $bold A$ can be quite influential on the momentum $bold p$ of ${roman Ca} sup {2+}$ ions, in both classical and quantum mechanics. Molecular scales of ${roman Ca} sup {2+}$ wave dynamics are coupled with $bold A$ fields developed at macroscopic regional scales measured by coherent neuronal firing activity measured by scalp EEG.

**31, Traumatic Brain Injury and Suicide: Current State of the Evidence.**

*Lisa A. Brenner, U.S. Department of Veterans Affairs, USA. Psychiatry, Neurology, and Physical Medicine and Rehabilitation at the University of Colorado Denver School of Medicine, USA.*

**Summary:** Data suggests that those with a history of traumatic brain injury (TBI) are at increased risk for death by suicide. Current evidence in support of this relationship will be presented along with findings regarding TBI and suicide attempts and ideation. Challenges associated with research in this area will be discussed, as well and suggestions for future study designs.

**32, Tau Mediated Neurodegeneration: An Insight into Alzheimer's Disease Pathology.**

*Magisetty Obulesu, Department of Biotechnology, Rayalaseema University, Kurnool, Andhra Pradesh, India.*

**Summary:** Extracellular accumulations of Ab, hyperphosphorylation of tau and intracellular neurofibrillary tangle formation have been the hallmarks of Alzheimer's Disease (AD). Although tau and its phosphorylation play a pivotal role in the normal physiology yet its hyperphosphorylation has been a pathological manifestation in neurodegenerative disorders like AD. In this review physiology of tau, its phosphorylation, hyperphosphorylation with the intervention of various kinases, aggregation and formation of paired helical filaments has been discussed. A brief account of various animal models employed to study the pathological manifestation of tau in AD and therapeutic strategies streamlined to counter the tau induced pathology has been given. The reasons for the failure to have suitable animal model to study AD pathology and recent success in achieving this has been included. The role of caspase cascade in tau cleavage has been emphasized. The summary of current studies on tau and the need for future studies has been accentuated.

**33, ReNaCE Programme. Integrating Parkinson's Disease in patients' and carers' lives.**

*Maria Carmen Portillo, School of Nursing, University of Navarra, Spain.*

**Summary:** The ReNACE programme (Recovery, Normalisation, Acceptance, and Living with an illness) aims to promote the integration of different chronic diseases in the daily life of patients and carers, through the design, implementation and evaluation of individually tailored multidisciplinary interventions. This multidisciplinary programme involves translational and participatory research that is taking place in different community settings to foster selfcare and selfmanagement strategies. Concretely, this programme has started with some research projects focused on Parkinson' Disease due to

the high psychosocial impact that this disease has, being a health priority all over the world. At present, the ReNACE programme has four research projects under process: ReNACE Project 1 “Study of patients’ and carers’ experience of living with Parkinson’ Disease. This is a mixed method project that aims to explore how the process of living with Parkinson’s Disease is perceived by patients and carers and to determine the main factors that foster or hinder the adaptation process. ReNACE Project 2 "Realistic evaluation of the implementation of a programme to improve the adjustment to Parkinson’s Disease”. This is a Realistic Evaluation project that aims to design, implement and evaluate a multidisciplinary intervention to improve the process of living with Parkinson’s Disease with patients and carers. ReNACE Project 3. “Design and validation of an assessment tool to measure the process of living with Parkinson in patients”. This transversal observational study aims to develop a tool that measures the adaptation process of living with Parkinson’ Disease. This tools is expected to be used in healthcare practice so that professionals can identify those aspects of the process of living with Parkinson’ Disease that need to improve and consequently, plan future interventions. ReNACE Project 4. “Spanish version of Psychosocial Adjustment to Illness Scale Caregiver Self-Report (PAIS-SR): translation and validation process in carers of Parkinson´s Disease patients” This cross-sectional study aims to translate to Spanish and validate the psychometric properties of Caregiver PAIS-SR in carers of patients with Parkinson´s Disease. This will take place in Primary Care Practices and associations of PD patients of different regions in Spain. As it will be illustrated with results from the projects and discussed in this presentation, all the research projects integrated in the ReNACE programme constitute a coherent body of knowledge about the phenomenon of “living with” Parkinson. Furthermore, this research has direct and clear implications for healthcare practice for different disciplines through the implementation of interventions and the development of assessment tools.

**34, Could neurobiological correlates of Bipolar Disorder be seen as a support for differential diagnosis?**

*1,2Massimo Cocchi, 1Lucio Tonello, 1Fabio Gabrielli 1 “Paolo Sotgiu” Research Institute for Quantum and Quantitative Psychiatry and Cardiology 2Department of Veterinary Medical Sciences, University of Bologna.*

**Summary:** The identification of three platelet fatty acids (Palmitic, Linoleic and Arachidonic acids), in addition to allowing the identification of subjects affected by Mood Disorders, brought about some hypotheses which, over the time, have been proven by robust experimental data concerning also the concept of serotonin uptake on the basis of membrane viscosity, and have showed that platelets are really similar to neurons, not only as to their embryo-genetic origin but also in terms of molecular features (1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16). This evidence, together with the possibility of classifying the two main mood disorders (Major Depression-MD and Bipolar Disorder-BD)(17), led to some considerations on the molecular uniqueness of Major Depression, generally understood as a phenomenon affecting only human beings, and, more precisely, just part of them. In this context, we can trace the human states between normality, Bipolar Disorder and Major Depression, this latter considered as a bio-molecular and existential niche (18, 19). When looking into the undeniable distinction made between depressed and bipolar subjects thanks to the neural network (Self Organizing Map-SOM)

and the chemical index (B2) for indirect assessment of platelet membrane viscosity, some considerations arise: The molecular characteristics of the population affected by Major Depression are completely different from those of many other investigated living beings, be they humans or animals (excluding the dog). So, depressed subjects form a particular and isolated group having, from the metabolic and biochemical point of view, unique characteristics; On the basis of our reflections, MD would be, at this point, a real disease, with specific molecular features and expressions of consciousness; The molecular attitude of Bipolar subjects (who are far more numerous than those suffering from MD) could be considered as the alternation of behavioral expressions and, in a sense, it would imply better chances of recovery, although the possibility of dramatic and explosive onset of the psychotic symptom cannot be easily controlled. The use of biochemistry, non-linear mathematics, and human-animal comparison leads to some reflections which are not only really close to a cultural and biological interpretation of mood disorders, but also pave the way for diagnostic perspectives and predictive interpretation patterns of the disease known as "Mood Disorder". Having said that, the role of Philosophy is crucial in order to: Theoretically support the whole system (Theory of complexity); Deal with the "issue of consciousness" with epistemological accuracy; Conceptually expand the relationship between nature and culture. Bibliografia: 1, Cocchi M. Tonello L. Tsaluchidu S. Puri B.K. (2008). The use of artificial neural networks to study fatty acids in neuropsychiatric disorders. BMC Psychiatry, 8 (Suppl 1): S3. 2, Cocchi M. Tonello L. (2010). Bio molecular considerations in Major Depression and Ischemic Cardiovascular Disease. Central Nervous System Agents in Medicinal Chemistry, 10: 97-107. 3, Tonello L. Cocchi M. (2010). The cell membrane: a bridge from psychiatry to quantum consciousness? NeuroQuantology, 8: 54-60. 4, Cocchi M. Gabrielli F. Tonello L. Pregnolato M. (2010). The Interactome Hypothesis of Depression. NeuroQuantology, 4: 603-613. 5, Cocchi M. Gabrielli F. Tonello L. Pregnolato M. (2011). Consciousness and Hallucinations: Molecular Considerations and Theoretical Questions. NeuroQuantology, 9: 182-189. 6, Cocchi M. Tonello L. Gabrielli F. Pregnolato M. Pessa E. (2011). Quantum Human & Animal Consciousness: A Concept Embracing Philosophy, Quantitative Molecular Biology & Mathematics, Journal of Consciousness Exploration & Research, 2: 547-574. 7, Cocchi M. Tonello L. Gabrielli F. Pregnolato M. (2011). Biological and Anthropological-Existential Hypothesis on Depression. Quantum Biosystems. 3: 12- 18. 8, Cocchi M. Tonello L. Gabrielli F. Pregnolato M. (2011). Depression, osteoporosis, serotonin and cell membrane viscosity between biology and philosophical anthropology. Annals of General Psychiatry, 10: 9. 9, Cocchi M. and Tonello L. (2012) How mathematics can inform the diagnosis of Mood Disorders. NeuroQuantology, 10: S1-28. 10, Cocchi M. Tonello L. Gabrielli F. (2012). Considerations on Blood Platelets: A Neuron's Mirror for Mood Disorders? Open Journal of Blood Diseases, 2: 22-29. 11, Heron D.S. Shinitzky M. Hershkowitz M. Samuel D. (1980). Lipid fluidity markedly modulates the binding of serotonin to mouse brain membranes. Proc Natl Acad Sci, 77: 7463-7467. 12, Lee R.E. (1985). Membrane engineering to rejuvenate the ageing brain. Can Med Assoc J, 132: 325–327. 13, Evers C.A. Starr L. (2006). Biology: Concepts and Applications. 6th ed. United States: Thomson, 0-534-46224-3. 14, Marangos P J. Campbell I.C,. Schmechel D.E. Murphy D.L. Goodwin F.K. (1979). Blood Platelets Contain a Neuron-Specific Enolase Subunit. Journul of Neurochemistry, 34: 1254- 1258. 15, Leonard B. (2000). Clinical implications

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**35, The syndrome of new daily persistent headache.**

*Matthew S. Robbins, Einstein Division of Montefiore Medical Center, Inpatient Services, Montefiore Headache Center, Neurology, Albert Einstein College of Medicine, USA.*

**Summary:** New daily-persistent headache (NDPH) is an uncommon chronic daily headache syndrome that features daily headache from the onset without any clear etiology. This presentation reviews the history and classification of new daily persistent headache, followed by its clinical manifestations and speculation about potential etiologies and pathogenesis. The syndrome's epidemiology, differential diagnosis, treatment, and prognosis are also addressed.

**36, Effects of real-life experience on human face processing performance.**

*Meike Ramon, Institute of Neuroscience and Psychology, UK.*

**Summary:** Humans are considered to be experts at face processing. Indeed, we are extremely efficient at decoding various types of information conveyed by faces: within a couple hundred milliseconds we can e.g. infer a person's ethnicity, age, gender, or expression. Recognizing the identity of personally familiar individuals seems comparably effortless, despite at times dramatic changes in viewing conditions. This high fidelity for identification of personally familiar faces cannot be observed for unfamiliar faces and remains unparalleled by automatic face recognition systems, which humans continue to outperform. Here I present work involving both healthy individuals and a case of acquired prosopagnosia ('face-blindness' caused by brain damage) conducted with the aim of identifying processing differences related to real-life exposure to faces.

**37, Maximizing outcomes in Glioblastoma.**

*Michael E. Salacz, Saint Luke's Brain Tumor Center, Neuro-oncology Program, USA.*

**Summary:** Glioblastoma remains one of the most common primary, malignant brain cancers, as well as one of the most deadly and refractory of all cancers to conventional therapy. Although uncommon, there are patients who do quite well, tolerating therapy with minimal long-term deficits, and enjoy a long-term survival even after cessation of planned treatments. Additionally, there are a number of patients who can have effective palliation of both disease and symptoms and are able to enjoy a good quality of life with ongoing therapy. This presentation will cover management and care of these patients, from initial diagnosis, through surgery, radiation and concurrent chemotherapy and

adjuvant chemotherapy, with a focus on maximizing both quality of life as well as survival.

**38, The role of pro-inflammatory cytokines in cancer related fatigue and depression.**
*Miri Cohen, the School of Social Work and Department of Gerontology, Faculty of Social Welfare and Health Sciences, University of Haifa, Israel.*
**Summary:** Background: Cancer-related fatigue (CRF) is a long-term and prevalent symptom among cancer survivors, but its psycho-biological antecedents and its relations to psycho-immune factors are not fully understood. The theoretical background and results of a prospective study that assessed the relation between age, inflammatory markers, CRF, depression and optimism will be presented. Methods: Breast cancer patients, stages I-III, were assessed one month after completion of chemotherapy (T1) and a subsample was assessed six months later (T2). At both time points, blood samples were assessed for pro-inflammatory cytokines (IL-6 and IL-8) in serum and participants answered questionnaires for fatigue (Fatigue Symptom Inventory), depression (Center of Epidemiological Studies- Depression) and optimism (Life Orientation Test). In addition, disease-related variables were recorded from medical files. Results: Participants reported low levels of depression, moderate levels of fatigue and high levels of optimism. Depressive symptoms increased after six months, levels of optimism decreased and levels of fatigue did not change between the two time points. At T1, depressive symptoms and optimism, but not CRF, were associated with higher levels of the serum pro-inflammatory cytokines, IL-6 and IL-8. Controlling for age, IL-6 at T1 predicted levels of CRF at T2, but not of depressive symptoms. The later were predicted by optimism at T1. Conclusions: Results suggest that high serum IL-6 soon after ending chemotherapy might be an antecedent of long term CRF. High levels of IL-6 were associated with high depressive symptoms and low optimism, that may together in the long-run accelerate CRF. These results add to the existing knowledge on risk factors for CRF. The study suggests that individuals who are low on optimism, or have high depressive symptoms, or have high level of serum IL-6 are at risk for developing CRF within six months post-treatment. Their identification at the start of chemotherapy will allow to provide them with psycho-social treatment and support, that may prevent the long term CRF.

**39, Embryonic stem cell-derived neurons as an in vitro model for investigating developmental neurotoxicity of environmental pollutants.**
*Mohammed El Majdoubi, Circe McDonald and Monia Zagzoog, Department of Natural Sciences and Mathematics, Dominican University of California, San Rafael, CA 94901, USA.*
**Summary:** Because of the limitations of animal-based models and traditional cell culture models of neuronal development, the mechanisms of developmental neurotoxicity of environmental pollutants are poorly understood. In recent years, embryonic stem cell (ESC)-derived neuronal models have been developed which offer distinct advantages over traditional model systems for investigating the effects of neurotoxins directly on the developing neuron. ESCs are undifferentiated cells that can proliferate indefinitely and undergo induced differentiation into functional adult neurons in culture. In vitro neuronal differentiation recapitulates several critical processes involved in the development of the nervous system including proliferation, migration, differentiation, and synaptogenesis. In

the present study, we cultured feeder-independent mouse embryonic stem cells and induced their differentiation into neurons. Using this model we assessed developmental neurotoxicity of four heavy metal compounds found in the environment: mercury, cadmium, lead, and manganese. Changes in cell viability and replication rates were monitored at each step of the developmental process of ESCs. The efficiency of neuronal differentiation was determined by calculating the proportions of cells that are immuno-positive for MAP-2, a cytoskeleton protein unique to neurons. Undifferentiated ESCs were generally more sensitive to higher physiological doses of all four compounds, which inhibited cell proliferation and induced apoptosis. In contrast, lower physiological doses of these compounds did not impact ESCs proliferation but did interfere with their differentiation into neurons. These results, which are consistent with findings in animal-based models, demonstrate that the neuronal differentiation of ESCs is a useful alternative model system for investigating developmental neurotoxicity of environmental pollutants directly at the cellular level.

**40, Deficient Repair of Genomic Damage as a Basis for Neurodegenerative Diseases Role of Transition Metals and the RNA-binding Protein TDP-43.**

*Muralidhar L. Hegde, Department of Neurology, Department of Biochemistry and Molecular Biology, University of Texas Medical Branch (UTMB), USA.*

**Summary:** Accumulation of genome damage including oxidized bases, single- and double-strand breaks, in affected brain cells has been linked to neurodegenerative diseases including Alzheimer's (AD) and Parkinson's (PD) disease and amyotropic lateral sclerosis (ALS), whose underlying cause(s) are not completely understood. Here we show that specific inhibition of NEIL glycosylase-initiated repair of oxidized DNA bases by transition metals iron (Fe) and copper (Cu) and functional loss of RNA binding protein TDP-43 could play a role in accumulation of unrepaired genome damage in neurons leading to cell death. Inhibition of oxidative genome damage repair by transition metals and its consequences in neurodegenerative diseases. The redox active Fe and Cu, which accumulate in neurodegenerative brain induce oxidative DNA damage via generation of reactive oxygen species. Most oxidized bases in mammalian genome are repaired via the base excision repair pathway, initiated with their excision by one of major DNA glycosylases, NTH1 and OGG1 (of Nth family), and NEIL1 and NEIL2 (of Nei family). We show that Fe(II/III) and Cu(II) bind to NEIL1 and NEIL2 both in vitro and in neuroblastoma (SH-SY5Y) cells at physiologically relevant concentration, significantly inhibiting both base excision and AP lyase activities and changing their secondary structures. This inhibition is specific as indicated by the lack of similar inhibition of OGG1. Thus Fe/Cu overload in the neurodegenerative diseases could act as a double-edged sword by both increasing oxidative genome damage and preventing their repair. Functional loss of TDP-43 linked to deficient DNA Repair. The neurotoxicity of TDP-43, an RNA binding nuclear protein involved in mRNA processing, has been implicated in ALS and other neurodegenerative diseases, which is linked to its hyperphosphorylation-mediated cytoplasmic-aggregation and reduction in nuclear level. We show that TDP-43 plays a role in DNA double-strand break repair (DSBR) in neuronal cells. We observed its stable in-cell association with DSBR markers gH2AX, DNA-PKcs, Ku70/80, PARP1 in SH-SY5Y cells, which was enhanced after treatment with DSB-inducing radiation/bleomycin. TDP-43 depletion markedly increases

accumulation of DSBs in SH-SY5Y cells. These results are consistent with the small but significant increase in unrepaired DSBs in postmortem brains of affected neurodegenerative diseases. Thus deficiency in DNA strand break repair may be a key etiologic factor in neurodegenerative diseases which is supported by examples linking such inherited diseases to deficiency in DNA strand break repair genes. These results suggest that inhibition of genome damage repair in neuronal genome represents a common basis for neurodegenerative diseases. Supported by New Investigator (NIRG) grant from Alzheimer's Association)

**41, Potential agents to Enhance the Expression of Neurotrophins in the Brain.**

*Narasimha Murthy, Pharmaceutics at the University of Mississippi, USA.*

**Summary:** Neurotrophins are endogenous peptides responsible for the development and repair of neurons in the brain. Depletion of neurotrophins could potentially lead to neurodegenerative disorders. Direct delivery of neurotrophins to the brain is a challenging task. Therefore, there is an urgent need to discover safe therapeutic agents that can enhance the expression of neurotrophins in the brain. In this direction, we screened some of the natural products for their ability to enhance the expression of neurotrophins in the brain following intranasal and i.p administration in rat model. The pharmacokinetic studies were performed in Sprague dawley rats. Pharmacodynamic studies were performed in depression induced rat model (BDNF depleted rats).

**42, Immobilized laminin concentration gradients on electrospun fiber scaffolds for controlled neurite outgrowth.**

*Nicole E Zander, U.S. Army Research Laboratory, Weapons and Materials Research Directorate, Aberdeen Proving Ground, Maryland 21005, USA.*

**Summary:** Neuronal process growth is guided by extrinsic environmental cues such as extracellular matrix proteins (ECM). Recent reports have described that the growth cone extension is superior across gradients of the ECM protein laminin compared to growth across uniformly distributed laminin. In this work, we have prepared gradients of laminin on aligned electrospun nanofibers for use as substrates for neuronal growth. The substrates therefore presented both topographical and chemical guidance cues. Continuous and step gradients were prepared by the controlled robotic immersion of plasma-treated polycaprolactone fibers reacted with N-hydroxysuccinimide into the protein solution. The gradients were analyzed using x-ray photoelectron spectroscopy and confocal laser scanning microscopy. Gradients with a dynamic range of protein concentrations were successfully generated and neurite outgrowth was evaluated using neuron-like PC12 cells. A significant increase in the number of cells adhered to the fiber scaffold was observed across the gradient. After 10 days of culture, PC12 neurite lengths varied from 32.7 ± 14.2 μm to 76.3 ± 9.1 μm across the protein concentration gradient. Neurite lengths at the highest concentration end of the gradient showed a significant improvement over neurite lengths observed for cells cultured on samples with uniform protein coverage. Gradients were prepared both in the fiber direction and transverse to the fiber direction. Neurites preferentially aligned with the fiber direction in both cases indicating that fiber alignment has a more dominant role in controlling neurite orientation, compared to the chemical gradient.

**43, Nervous circuit in the tail of trematode cercaria: from morphology to motility regulation.**

*Oleg Tolstenkov, Severtsov Institute of ecology and evolution of Russian Academy of Sciences, Leninskiy prospect, 33, Moscow, 119071 Russia.*

**Summary:** The motile larval stages of trematodes – cercariae have a complex of morphological and behavioral adaptations that help them to find and infect the next host. Comparative morphological study of neuromuscular system in the tail of cercaria revealed a variety of structures composed by the single plan. The nervous system in the tail contains a limited number of neurons that make the stage of cercaria a promising model for investigating the basic mechanisms of parasite motility. In this study we investigate the spontaneous electrical activity in the tail and the role of serotonin and acetylcholine components in the regulation of motility in two model species of cercaria. The future research is also discussed. The study was supported by RFBR grants 12-04-01051-a, 12-04-01086-a and grant of President of Russian Federation MK-811.2013.4.

**44, Helping people with disabilities with Brain-Computer Interfaces.**

*Pablo F. Diez, the "Gabinete de Tecnología Médica" (Medical Technology Laboratory) at the "National University of San Juan", Argentina.*

**Summary:** In this presentation a brief introduction to Brain-Computer Interfaces (BCI) technology is presented. Later, the Steady-state Visual Evoked Potentials (SSVEP) based BCI developed in our laboratory is introduced. Then, applications of that BCI system in robotics, such as a mobile robot and a robotic wheelchair, are presented.

**45, Modeling circadian neural circuits: Emergent synchronization in populations of coupled phase oscillators.**

*Pablo Gleiser, Centro Atómico Bariloche, Instituto Balseiro, CONICET, Bariloche, 8400 Río Negro, Argentina.*

**Summary:** As an adaptation to cyclic environmental changes many species present behavioral and physiological rhythms with a period close to 24 hours, known as circadian rhythms. These daily rhythms are endogenously generated, and continue to oscillate even in the absence of any environmental cues. It is known that the generation of circadian rhythms takes place at the individual cell level (driven by transcriptional-translational negative feedback loops), however behavioral rhythms are controlled by groups of intercommunicating neurons, which synchronize their circadian phases and act as pacemakers. These neurons receive information from the environment (such as light, temperature, etc.) and transmit it for entrainment to downstream oscillators. An interesting open question is whether these groups of neurons are anatomically fixed, or if they are plastically distributed throughout the neuronal circuits and can vary their role due to environmental changes. In this talk I will show examples taken from the literature of the pacemaker neurons in the fly Drosophila and mammals, where different cell populations are said to be responsible for morning and evening peaks of activity. Then I will present a simple phase oscillator model, where the circadian rhythms of individual neurons are represented by a single phase variable. Finally I will show results of the model which allow for insight into the role that the coupling between different groups of

neurons plays in the emergent synchronization properties.

**46, Effect of the simultaneous impact of opioid drug (tramadol) and electromagnetic field (EMF) on lipid peroxidation.**

*Bodera P., Stankiewicz W., Krzyzowska M., Antkowiak B., Paluch M. Military Institute of Hygiene and Epidemiology, Warsaw, Poland.*

**Summary:** Regulation of signal transduction in the brain depends on the transportation of lipids, where plasma low density lipoproteins (LDL) and high density lipoproteins (HDL) are involved. HDL-like lipoproteins, found in cerebrospinal fluid, play a role in lipid transportation in the central nervous system. Lipoproteins are susceptible to lipid peroxidation, which is triggered by reactive oxygen species (ROS), produced and found in the brain and peripheral tissue. The presence of oxidized LDL (ox-LDL) in demyelinating plaques in the brain during the course of multiple sclerosis (MS), and the increase of antibodies against ox-LDL in plasma suggests this disease is associated with oxidative damage of lipoproteins. An increase in the production of ROS and RNS as a result of the impairment of the antioxidant system may contribute to lipoprotein peroxidation in MS. The electromagnetic field (EMF), which is widely encountered in the environment, is produced both for technological applications (e.g., power lines, mobile phones), as well as in medicine for diagnostic, e.g., magnetic resonance imaging (MRI), and therapeutic purposes as radiofrequency and microwave ablation and hyperthermia in e.g. aesthetic dermatology. EMF, as one environmental factor, may be involved in a number of metabolic processes which generate oxidants and antioxidants. Currently, several reports have been published which present deleterious effects of EMF exposure. Special attention is addressed to assessing the potentially harmful effects of EMF on the brain and other important organs for existence. Our xperiments were performed on healthy rats, and on rats with persistent inflammatory state, induced by Complete Freund's Adjuvant (CFA) injection. Animals were placed 1 meter from the EMF source, and exposed to the far-field range of an antenna at 1800 MHz with the additional modulation identical to that generated by mobile phone GSM 1800, and the value of effective electric field 20 V/m and effective magnetic field value 0.05 A/m. Rats were given one or five (once a day) 15 minutes exposures or were sham-exposed with no voltage applied to the field generator in control groups. Immediately before EMF exposure (the last one in the case of five exposures), particular groups of rats were intraperitoneally injected with tramadol in the 20 mg/kg dose or vehicle (aqua pro injectione) in the 1 ml/kg volume. Lipid peroxidation (LPO) was estimated by measuring the concentration of the colored complex formed by malondialdehyde (MDA), end product of LPO during reaction with externally added thiobarbituric acid (TBA). We observed statistically important differences in lipid peroxidation between control group (CON) and both groups CFA/EMF and tramadol(TRAM)/EMF). The differences of the lipid peroxidation level in rats' blood were particularly visible after 5 times of EMF radiation, particularly higher in the groups under exposure: control (CON/EMF) and TRAM/EMF, versus control group (CON), without EMF exposure. The received data may suggest that longer EMF exposure may affect some biological structures, which are responsible for free radical scavenging mechanisms. Our study may suggest the impact of EMF radiation on the development of brain tumors, because ROS are thought to take part in oncogenesis and cellular differentiation.

**47, Imaging 3D spatiotemporal hemodynamics of single cortical vessels in vivo using two-photon laser scanning microscopy.**

*Peifang Tian, John Carroll University, USA.*

**Summary:** The dynamics response of individual cerebral vessels to sensory-stimuli is crucial to form a mechanistic understanding of functional imaging technologies, such as functional MRI (fMRI), as well as for understanding neurovascular dysfunction, as occurs in stroke and dementia. Using optical imaging technologies such as two-photon laser scanning microscopy and the rat primary sensory cortex as our animal model, we have characterized the stimulus-evoked cerebral hemodynamic response on the level of single arterioles and capillaries throughout a significant three-dimensional volume (~1-2mm3) in vivo. Further, we will relate this characterization to the underlying neuronal electrical activity and the angioarchitecture. In this talk, I will discuss the diameter changes of three classes of vessels, i.e., surface arteries/arterioles, penetrating arterioles, and subsurface microvessels, in response to electric forepaw stimulation. In particular, I will focus on the dependence of a vessel's response on its distance from the center of the neuronal activity, its depth in the cortical tissue, and its connectivity to penetrating arterioles. This work helps to bridge the critical gap between macroscopic functional imaging technologies such as fMRI and the microscopic understanding of single vessel responses to the neuronal activation.

**48, Multifunctional tacrine derivatives as potential agents to treat Alzheimer's disease.**

*Praveen Rao Perampalli Nekkar, Pharmaceutical Sciences PHR 5002, School of Pharmacy Health Sciences Campus, 200 University Ave W University of Waterloo, Waterloo, ON N2L 3G1, Canada.*

**Summary:** Alzheimer's disease (AD) is a complex neurological disorder and its pathophysiology is not well understood. In this regard, several hypotheses such as (i) cholinergic dysfunction (ii) amyloid cascade hypothesis (iii) tau phosphorylation and (iv) oxidative stress are known to play a significant role, suggesting that developing hybrid small molecules capable of targeting multiple pathways is an attractive route toward potential disease-modifying therapies to treat AD. In order to address this, we designed small molecules based on tacrine, a known cholinesterase inhibitor to target the cholinergic and oxidative stress pathways of AD. A library of tacrine derivatives with various substituents at either C-6 or C-7 positions and antioxidant/metal chelating pharmacophores at C-9 were designed and synthesized. They were evaluated in vitro to assess their (i) cholinesterase inhibition (acetyl and butyrylcholinesterase) (ii) antioxidant activities and (iii) metal chelation properties. These tacrine derivatives exhibit dual cholinesterase inhibition in the micromolar to nanomolar range compared to reference agents: tacrine, galantamine and donepezil with excellent antioxidant and metal chelating properties. Our studies show that a tacrine ring template can be modified by incorporating suitable pharmacophores to develop hybrid molecules to target multiple-AD pathways as opposed to the traditional "one drug, one target" approach.

**49, Integrated neuroscience of bonding.**

*Radovan Hruby, Psychiatric Outpatient Clinic, Martin, Slovak Republic.*

**Summary**: A lot of living organisms exhibit various patterns of social behavior. The social aspect of existence is of special importance in humans, who are fundamentally social. All the mammalians begin to live already from the moment of fertilization as a part of the system which is represented by very close bonding between the fetus and the mother. Also the newborn consistently preserves this special kind of bonding. While during prenatal and early postnatal period is the bonding mostly physiological, postnatal period is characterized by the qualitative shift from the bio-social to psycho-social level. There is a great body of knowledge that degree of the development in utero enables the human fetus to perceive and process a lot of sensory and even emotional and social stimuli. New findings in many fields of neuroscience are showing an increasing evidence for very special role of prenatal and postnatal bonding for complex social, emotional and cognitive development. The prenatal period is the most important, critical phase for the human brain development and it is followed by the next extremely important phase of human life - attachment period. Importance of this period is reflected by the Bowlby´s attachment theory. The theory is a general concept of complex human development, which emphasizes the role of early mother-infant interactions for infant's adaptive behavioral and stress copying strategies, personality organization and mental health. Increasing findings in neuroscience enables to more precisely identify neural processes which are involved in the creation of attachment between mother and child. It could be defined as intense interactions between genes and environment, multi-level neural interactions and effects of neuromediattors, hormones and essential neurobiological processes including emotional, cognitive, social interactions and the special key role of mentalizing. The theory is unique by its complexity and consequences and could serve as a prototype of modern integrated neuroscience approaches. Key words: attachment, bonding, prenatal and postnatal neural development.

**50, The occipital lobe convexity sulci and gyri - Laboratory investigation.**

*Raphael V. Alves, Department of Neurosurgery, Hospital Beneficência Portuguesa, São Paulo, Brazil.*

**Summary:** Object. The anatomy of the occipital lobe convexity is so intricate and variable that its precise description is not found in the classic anatomy textbooks, and the occipital sulci and gyri are described with different nomenclatures according to different authors. The aim of this study was to investigate and describe the anatomy of the occipital lobe convexity and clarify its nomenclature. Methods. The configurations of sulci and gyri on the lateral surface of the occipital lobe of 20 cerebral hemispheres were examined in order to identify the most characteristic and consistent patterns. Results. The most characteristic and consistent occipital sulci identified in this study were the intraoccipital, transverse occipital, and lateral occipital sulci. The morphology of the transverse occipital sulcus and the intraoccipital sulcus connection was identified as the most important aspect to define the gyral pattern of the occipital lobe convexity. Conclusions. Knowledge of the main features of the occipital sulci and gyri permits the recognition of a basic configuration of the occipital lobe and the identification of its sulcal and gyral variations.

**51, Neuropsychiatric Syndromes in a Patient with Lupus from bench to bedside.**

*Reem Hamdy A Mohammed, Rheumatology and Rehabilitation, Faculty of Medicine,*

*Cairo University.Rheumatology Consultant, Alhada Military Hospital, KSA., Egypt.*
**Summary:** Neuropsychiatric syndromes in lupus represent a serious and potentially threatening yet treatable feature of SLE. Neuropsychiatric disease in SLE might develop in 10% and might reach up to 80% of cases with the established diagnosis of SLE. It is rather a challenge due to a multiplicity of facts including heterogeneity of the clinical spectrum, with the great similarity between the SLE related neurological syndromes and other non SLE neurological diseases, in addition to the lack of unifying diagnostic criteria that would support establishing the diagnosis in cases where the diagnosis of SLE is not fully established. The neuropsychiatric profile of CNS lupus is variable and may be classified as primary neurologic and psychiatric disease (eg, related to disease related direct involvement of the neuropsychiatric system) or secondary disease (eg, related to complications of the disease and its therapy). The exact immune-pathogenic mechanisms involved in inducing such features remains poorly understood are largely dependable on the spectrum of presentations in each individual patient. Vasculopathy and vasculitis have been identified as possible contributors especially in demyelinating forms or focal syndromes, whereas in more diffuse syndromes like the organic brain syndrome which happens to develop in a considerable proportion of these patients the pathology appears to be largely independent of the nature or severity of vascular pathology. Radio-imaging studies in such patients remain a cornerstone in identifying the associated organic pathology with computerized tomography and magnetic resonance imaging with or without angiography studies being the most sensitive techniques. The use of SPECT (single photon emission computerized tomography) is another beneficial radi-imaging modality in active cerebritis. The presentation will provide a comprehensive approach towards a better understanding of a patient with neuropsychiatric lupus, with highlights on the value of clinical, laboratory and radio-imaging studies in establishing the diagnosis as well as updates on the management.

**52, The Phosphoinositide signal transduction Pathway and the development of human nervous system.**

*Rita Lo Vasco, Department of Sensitive Organs, Policlinic Umberto I, Sapienza University, Rome, Italy.*
**Summary:** The Phosphoinositide signal transduction Pathway and the development of human nervous system. The development of nervous system is tightly regulated by a network of interconnected signal transduction pathways. The extensive crosstalk among different signal transduction systems deserves great attention. In fact, understanding the timing of the cascade of events regulating the development of the nervous system might open the way to novel therapeutic strategies. In the last 20 years, great interest was paid to the Phosphoinositide (PI) signal transduction pathway and related Phosphoinositide-specific phospholipids C (PI-PLC) family of converting enzymes, which contribute to the regulation of intracellular calcium levels. Beside their well-known role in the metabolism of calcium, PI-PLC enzymes interact with a number of molecules belonging to other signal transduction pathways, contributing to the peculiar and complex network in the developing nervous system. In the present communication, the connection of PI signalling and further transduction pathways acting during neural development will be analyzed, with special regard to the role of PI-PLC family of enzymes.

**53, Consciousness concept from Rumi‘s view: fundamentalism or emergentism, a bridge between quantum theory and consciousness.**

*Shahriar Ahmadpour, Department of Anatomy, Medicine School, North Khorasan University of Medical Sciences (NKUMS), Bojnourd, Iran. Shahriar Ahmadpour, Khadijeh Foghi.*

**Summary:** consciousness has been stated as one of the hard problem in neural sciences. At present biologists could have not explained some mysterious aspect of human consciousness such as ‘’ does consciousness emerge from the brain or dose universe possess consciousness? And dose our brain just receive universe shadow? In this study we aimed to conform Rumi’s view, a Persian Gnostic and poet, about cosmic universe to current opinions and theories about consciousness. Method and Material: About 800 years ago a Persian Gnostic, Mohammad Balkhi (Rumi), explained his spiritual contemplation as poem. We searched verses of poetical works of Shams, comparing it to the current quantum theories of mind. Results& conclusion: Based on his verses, Rumi believed that all of basic element, minute particles, of universe are conscious; he composes:’’ All of the universe basic minute particles whisper secretly days and night: we are able to hear, to see and are conscious! But just confidents will hear this whisper! According to Schrodinger’s equation and Uncertainty principle of quantum theory an electron behaves consciously .It mean that there is a hidden consciousness in the universe ,cosmic universe or pansychism or pan proto sychism, concepts which were proposed by Spinoza ( 17 th century) and expanded by David Chalmers . These concepts denote to the same philosophy, fundamentalism, as Rumi contemplated spiritually. He says: what is consciousness? From a whole wisdom! Wisdom is hidden, but the world is evident. So he coined the term of cosmic consciousness. Thus each wave or particle (electron), consciousness, carries the cosmic consciousness from past to future (principle of conservation of energy).According to his view all of the basic particles of universe dance harmoniously (electron spin), emitting waves which are perceived by our brain. Furthermore consciousness doesn’t emerge from our brain, but just perceived and interpreted by human cerebrum! .In this way reality would not be necessarily what we perceive (Holographic theory), but it is an independent entity outside our skull (Fundamentalism). Key words: Rumi, Quantum, consciousness,fundamentalism.

**54, Plasticity in the auditory system: neuroscience evidence.**

*Sridhar Krishnamurti, Audiology in the Department of Communication Disorders at Auburn University, USA.*

**Summary:** Pending.

**55, Comparing the cost-effectiveness of two brain metastasis treatment modalities from a payer's perspective: Stereotactic radiosurgery versus surgical resection.**

*Vuong Anh Duong, Medical professional and legislation Division at the Department of Medical Service Administration, under the Ministry of Health, Vietnam.*

**Summary:** Objectives: This study aims to identify the cost-effectiveness of two brain metastatic treatment modalities, stereotactic radiosurgery (SRS) versus surgical resection (SR), from the perspective of Germany's Statutory Health Insurance (SHI) System. Methods: Retrospectively reviewing 373 patients with brain metastases (BMs) who

underwent SR (n=113) and SRS (n=260). Propensity score matching was used to adjust for selection bias (n=98 each); means of survival time and survival curves were defined by the Kaplan-Meier estimator; and medical costs of follow-up treatment were calculated by the Direct (Lin) method. The bootstrap resampling technique was used to assess the impact of uncertainty. Results: Survival time means of SR and SRS were 13.0, 18.4 months, respectively (P=.000). Medians of free brain tumor time were 10.4 months for SR and 13.8 months for SRS (P=0.003). Number of repeated SRS treatments significantly influenced the survival time of SRS (R2 =.249; p=.006). SRS had a lower average cost per patient (€7212 - SD: 1047; Skewness: 7273) than SR (€10964 - SD: 1594; Skewness: 0.465), leading to an incremental cost effectiveness ratio of €-8338 per life year saved (LYS), meaning that using SRS costs €3752 less than SR per targeted patient, but increases LYS by 0.45 years. Conclusion: SRS is more cost-effective than SR in the treatment of brain metastasis (BM) from the SHI perspective. When the clinical conditions allow it, early intervention with SRS in new BM cases and frequent SRS repetition in new BM recurrent cases should be advised. However, a more precise cost-effectiveness value of SRS versus SR could only be obtained by a prospective randomized study. Keywords: brain tumors, economics, stereotactic radiosurgery, surgical resection.