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UNIVERSITY OF
MEDICINE AND PHARMACY
CLUJ-NAPOCA
ROMANIA

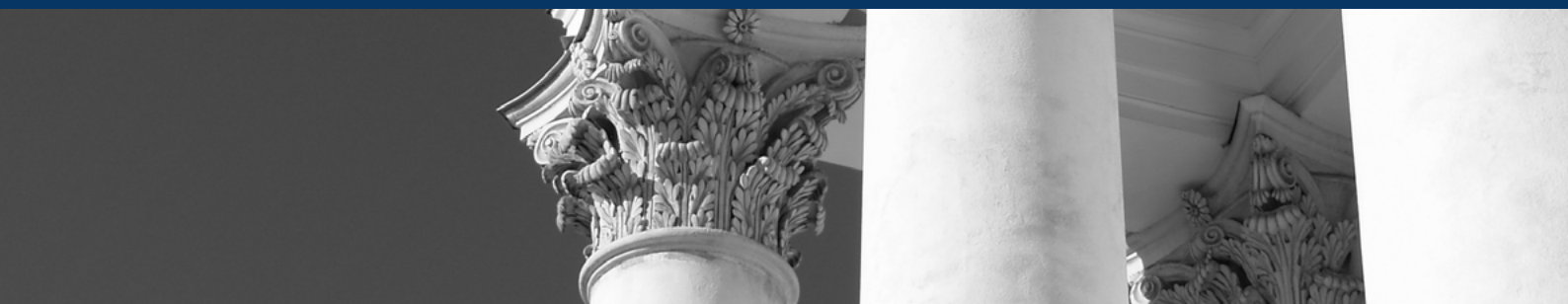
"IULIU HATIEGANU" UNIVERSITY OF MEDICINE AND PHARMACY DOCTORAL SCHOOL

NEUROSCIENCE

PROGRAM

2016-2017 | SECTION 7

8 MAY | UMF "IULIU HATIEGANU" | CLUJ-NAPOCA | ROMANIA



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President of the Society for the Study of Neuroprotection and Neuroplasticity (SSNN)

INTERNATIONAL GUEST LECTURERS

Michael Chopp

Henry Ford Hospital, Department of Neurology, Detroit, MI, USA

Oakland University, Department of Physics, Rochester, MI, USA

Marc Fisher

Professor of Neurology, Harvard Medical School

Emeritus Professor of Neurology, University of Massachusetts Medical School, USA

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2016-2017

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COURSE PROGRAM

COURSE PROGRAM

MAY 8TH, 2017

"MULTIMEDIA" AUDITORIUM, "IULIU HATIEGANU" UMF CLUJ-NAPOCA

8 VICTOR BABES STREET | CLUJ-NAPOCA | ROMANIA

09:50 – 10:00	Dafin F. Mureșanu /Romania Welcome address
10:00 – 10:45	Michael Chopp /USA Physiological and molecular mechanisms mediating tissue damage after stroke
10:45 – 11:30	Michael Chopp /USA Repair and restorative mechanisms after stroke
11:30 – 12:00	Session Break
12:00 – 12:45	Michael Chopp /USA Exosomes-biological nanoparticle therapy for stroke and neural injury
12:45 – 13:30	Michael Chopp /USA Prospective, double blinded, placebo controlled preclinical studies demonstrating the neurorestorative and neuroprotective effects of Cerebrolysin for stroke and traumatic brain injury (TBI) , and multifactorial mechanisms of action
13:30 – 14:30	Session Break

MAY 8TH, 2017

"MULTIMEDIA" AUDITORIUM, "IULIU HATIEGANU" UMF CLUJ-NAPOCA

8 VICTOR BABES STREET | CLUJ-NAPOCA | ROMANIA

14:30 – 15:15	Marc Fisher /USA Writing good scientific papers
15:15 – 16:00	Marc Fisher /USA Secondary stroke prevention
16:00 – 16:30	Session Break
16:30 – 17:15	Marc Fisher /USA Identifying and implementing translational stroke research
17:15 – 18:00	Marc Fisher /USA Cryptogenic stroke

INTERNATIONAL GUEST LECTURERS

MICHAEL CHOPP
USA

Michael Chopp, PhD, is Vice Chairman for Research of the Department of Neurology, Scientific Director of the Henry Ford Neuroscience Institute, and is the Zoltan J. Kovacs Chair in Neuroscience Research. He is also Distinguished Professor of Physics at Oakland University.

He received his MS and doctorate degrees in Mathematical and Solid State Physics from New York University. After nearly 10 years of working as a Physicist and as a Professor of Physics, Dr. Chopp made a career change and turned his interest to translational research in neuroscience Dr. Chopp's research has primarily focused on: 1) cellular and molecular biology of ischemic cell injury, 2) the pathophysiology of stroke, traumatic brain injury, peripheral neuropathy, multiple sclerosis, and glioma, 3) combination thrombolytic and neuro and vascular protective therapies for stroke, 4) mechanisms of neuroprotection, 5) cell-based and pharmacological neurorestorative therapies for stroke, traumatic brain restorative therapies for stroke, traumatic brain injury and neurodegenerative disease, 6) molecular and cellular mechanisms underlying neurogenesis and angiogenesis and the induction of brain plasticity leading to functional and behavioral recovery after neural injury,7) treatment of glioma and breast cancer, 8) exosomes/microRNA for treatment of neurological injury and disease, and 9) magnetic resonance imaging. Dr. Chopp has 639 peer reviewed publications, ~ 50 book chapters and has given 430 plenary lectures and invited presentations. He has chaired National Institutes of Health (NIH) study sections and has often served as a consultant to government agencies, the U.S. National Institutes of Health, and the pharmaceutical industry.

Awards include:

2001 Top Ten Research Advances of 2001, "Treatment of Stroke with Bone Marrow Stromal Cells", American Heart Association

2005 Distinguished Scientist Award, Henry Ford Medical Group, Board of Governors

2012 Lecture of Excellence and World Stroke Organization (WSO) Award, Remodeling and rewiring the intact CNS as a treatment for Stroke, 8th World Stroke Congress, Brasilia, Brazil, October

2014 Abraham White Distinguished Science Award. "For discovery of the role of thymosin beta 4 in the treatment of brain injuries and neurodegenerative diseases; 4th International Symposium on Thymosins in Health and Disease, Washington, DC, October

2015 Thomas Willis Lecture Award, International Stroke Conference, Nashville, TN, February

MARC FISHER
USA

Dr. Fisher was affiliated with the University of Massachusetts Medical School for 35 years and is currently an emeritus Professor of Neurology. He began work part-time at Beth Israel Deaconess Medical Center in Boston with an appointment at Harvard Medical School in August, 2014. He has a long track record in performing MRI-based experiments in rat stroke models to evaluate the presence and evolution of the ischemic penumbra. Using diffusion/perfusion MRI his experimental group has evaluated the effects of therapies on the progression of the diffusion/perfusion mismatch. Dr. Fisher has extensive experience in organizing and implementing clinical acute stroke therapy trials with a particular interest in imaging-based trials. He has performed these trials with co-investigators at multiple sites around the world. He has maintained an active clinical practice for many years with an emphasis on patients with cerebrovascular disorders as well as broad range of other neurological illnesses. He has published extensively and has published over 260 peer-reviewed articles with an h-index of 72 and has edited or co-edited 13 books. He currently serves as editor-in-chief of Stroke and will continue in that position until 2020.

ABSTRACTS

PHYSIOLOGICAL AND MOLECULAR MECHANISMS MEDIATING TISSUE DAMAGE AFTER STROKE

MICHAEL CHOPP
Henry Ford Hospital, Department of Neurology, Detroit, MI, USA
Oakland University, Department of Physics, Rochester, MI, USA

This presentation will include discussion of: 1) mechanisms underlying cerebral damage after experimental thromboembolic stroke, secondary microvascular hypoperfusion deficits post stroke-basis for no-reflow, 2) brain heart interactions ,-demonstrating secondary cardiac adverse effects of stroke , 3) comorbidity - the effects of age, gender and diabetes on stroke outcomes, and therapeutic approaches for the treatment of experimental stroke with diabetes.

REPAIR AND RESTORATIVE MECHANISMS AFTER STROKE

MICHAEL CHOPP
Henry Ford Hospital, Department of Neurology, Detroit, MI, USA
Oakland University, Department of Physics, Rochester, MI, USA

The following will be discussed: 1) Intrinsic restorative mechanisms activated after stroke, including stimulation and coupling of neurogenesis, angiogenesis, and axonal dendritic rewiring throughout the central nervous system; 2) the role of activated astrocytes in mediating restorative events; 3) molecular and microRNA pathways that contribute to remodeling of the CNS post stroke, with an emphasis on rtPA as a potential means to stimulate neurological recovery

EXOSOMES-BIOLOGICAL NANOPARTICLE THERAPY FOR STROKE AND NEURAL INJURY

MICHAEL CHOPP
Henry Ford Hospital, Department of Neurology, Detroit, MI, USA
Oakland University, Department of Physics, Rochester, MI, USA

The following will be discussed: 1) Intrinsic restorative mechanisms activated after stroke, including stimulation and coupling of neurogenesis, angiogenesis, and axonal dendritic rewiring throughout the central nervous system; 2) the role of activated astrocytes in mediating restorative events; 3) molecular and microRNA pathways that contribute to remodeling of the CNS post stroke, with an emphasis on rtPA as a potential means to stimulate neurological recovery.

PROSPECTIVE, DOUBLE BLINDED, PLACEBO CONTROLLED PRECLINICAL STUDIES DEMONSTRATING THE NEURORESTORATIVE AND NEUROPROTECTIVE EFFECTS OF CEREBROLYSIN FOR STROKE AND TRAUMATIC BRAIN INJURY (TBI) , AND MULTIFACTORIAL MECHANISMS OF ACTION

MICHAEL CHOPP
Henry Ford Hospital, Department of Neurology, Detroit, MI, USA
Oakland University, Department of Physics, Rochester, MI, USA

Here, I will summarize our data on prospective, double blinded, placebo controlled preclinical studies, performed under rigorous clinical trial conditions for the treatment of stroke and TBI. In addition, I will review and provide new insight into the multiple mechanisms of action of Cerebrolysin. Data will be shown that Cerebrolysin evokes expression of Angiopoietin 1 (Ang1), which promotes blood brain barrier integrity, is anti-inflammatory and mediates axonal outgrowth. Cerebrolysin also up regulates the expression of the developmental morphogen Sonic Hedgehog (Shh). Shh stimulates cellular expression of tissue plasminogen activator (tPA), which acts as both an endogenous thrombolytic agent and plays a pivotal role in promoting neurite outgrowth and neurological recovery. In addition , I provide novel insight into how Cerebrolysin stimulates specific sets of microRNAs (miRs). miRs are small non-coding RNAs which can simultaneously post-transcriptionally regulate the translation of many genes. Shh acts to up regulate cellular expression of the miR-17-92 cluster. This cluster of miRs, has potent anti-inflammatory effects as well as promotes axonal outgrowth. Thus, we demonstrate that Cerebrolysin has multifactorial neurovascular remodeling effects on tissue which drives neurological recovery.

WRITING GOOD SCIENTIFIC PAPERS

MARC FISHER
Professor of Neurology, Harvard Medical School
Emeritus Professor of Neurology, University of Massachusetts Medical School, USA

The key to writing good scientific papers is organization. You should pay close attention to manuscript requirements of the journal where you will submit your paper. If you are reporting the results of research study, make sure that the data are well analyzed and that the statistical methods appropriate. The manuscript should be organized into sections that include; introduction, methods, results and discussion. The abstract should be carefully written because it is the initial introduction to your manuscript that reviewers and readers will read before going further. You need to capture and hold their attention. In the introduction which typically consists of two paragraphs present the background as to why you did your study and then briefly overview what you attempted to do. In the methods section you should describe in some detail how the study was performed so that if someone wanted to reproduce it they could. A statistical methods section should be provided. In the results section, present the data from your study in a logical and comprehensive manner. Do not interpret the results because that should be saved for the discussion. Tables and figures should be used to present your data. In the discussion section that typically consists of 3-4 paragraphs briefly summarize your results and why they are important/novel. Then put them into context of prior studies in this area. You should then point out potential weaknesses or deficiencies of your data and how they might be addressed. Finally, conclude with how the results may lead to future studies. It is important to stick to the word limit of the journal you will submit to and that the English grammar be mistake free.

SECONDARY STROKE PREVENTION

MARC FISHER
Professor of Neurology, Harvard Medical School
Emeritus Professor of Neurology, University of Massachusetts Medical School, USA

After an initial ischemic stroke an important aspect of patient care is to reduce the risk of subsequent strokes. Good control of vascular risk factors such as hypertension, diabetes and hypercholesterolemia are key components of the effort to reduce recurrent stroke risk. For patients with large or small vessel disease as the mechanism for their stroke, antiplatelet therapy should also be employed. Either aspirin or clopidogrel can be prescribed and it is unclear if one drug reduces subsequent stroke risk more than the other. The combination of aspirin and extended release dipyridamole is another option that in the large PROFESS trial reduced the risk of subsequent ischemic stroke similarly to clopidogrel but with more side effects such as headache and dizziness. The combination of aspirin and clopidogrel should be considered for 3 months in patients with intracranial large vessel stroke. For patients with stroke secondary to atrial fibrillation anticoagulation is recommended. Warfarin was the only option for many years, but four newer oral anticoagulants are now available. I recommend that dabigatran or apixaban be considered for some atrial fibrillation related stroke patients because both have a lower risk of intracranial hemorrhage than warfarin and dabigatran also significantly reduced the risk of subsequent ischemic stroke as compared to warfarin. Apixaban was at least as good as warfarin in reducing ischemic stroke risk as compared to warfarin and had a substantially lower risk of all types of major bleeding side effects. The data are less compelling for rivaroxaban and edoxaban so I do not recommend them.

IDENTIFYING AND IMPLEMENTING TRANSLATIONAL STROKE RESEARCH

MARC FISHER
Professor of Neurology, Harvard Medical School
Emeritus Professor of Neurology, University of Massachusetts Medical School, USA

Translational stroke research represents the interface between basic science advances in the cerebrovascular field and determining if these advances are helpful for the diagnosis and treatment of stroke patients. The traditional approach to translational stroke research has been to identify basic research advances that may potentially be clinically useful such as the discovery of a novel pathway of ischemic brain injury that can ameliorated by a drug targeted towards this mechanism of injury. At the translational stage this new drug will be tested in appropriate animal models and if it is effective future clinical trials will be organized based upon the stroke modeling data. Another approach to translational research is reverse translation that occurs when a clinical advance triggers basic science research studies such as understanding how a novel therapy may improve stroke outcome or determining how an imaging modality can distinguish between infarction and the ischemic penumbra. A third approach to translational research is lateral translation that is characterized by basic research to improve upon a currently effective therapy. An example of this approach would be the development of better thrombolytic agents than tPA that have enhanced clot lysis effects and a better safety profile.

CRYPTOGENIC STROKE

MARC FISHER
Professor of Neurology, Harvard Medical School
Emeritus Professor of Neurology, University of Massachusetts Medical School, USA

Cryptogenic stroke is defined as a stroke of uncertain source despite an adequate search for the potential cause. The percentage of ischemic strokes that are cryptogenic vary among case series but contemporary studies suggest that approximately 25-30% of ischemic strokes do not have a determined cause. A recently defined group of cryptogenic stroke patients is those who are likely to have a cardioembolic stroke and they have been called embolic stroke of undetermined source (ESUS). The evaluation of ischemic stroke patients should include an extensive array of tests such as brain and vascular imaging, blood tests, an echocardiogram and monitoring of the cardiac rhythm. Since many cryptogenic strokes are thought to fall into the ESUS category, a more extensive cardiac evaluation should be considered. This would include transesophageal echocardiography and prolonged ECG monitoring in selected patients. The risk for recurrence of cryptogenic stroke is similar to patients with a determined source for their stroke. Secondary prevention should include antiplatelet therapy and risk factor modification. For ESUS patients, it is tempting to consider anticoagulation but current recommendations do not support this approach. Several ongoing clinical trials are comparing direct oral anticoagulants to antiplatelet therapy and the results should be available in a few years.


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