Academia de Științe Medicale din România

Federation of European Academies of Medicine

THE SOCIETY FOR THE STUDY OF NEUROPROTECTION AND NEUROPLASTICITY

UMF UNIVERSITATEA DE MEDICINĂ ȘI FARMACIE IULIU HAȚIEGANU CLUJ-NAPOCA

Romanian Medical Academy Brain Days

3_5/09/2010 INTERCONTINENTAL HOTEL, BUCHAREST

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INVITED SPEAKERS

(in alphabetical order)

Antón ÁLVAREZ / Spain
Raul ARIZAGA / Argentina
Stavros J.BALOYANNIS / Greece
Ovidiu BĂJENARU / Romania
Heinrich BINDER / Austria
Angel CEDAZO-MÍNGUEZ / Sweden
Alexandru V. CIUREA / Romania
Antonio FEDERICO / Italy
Franz GERSTENBRAND /Austria
Jakub HORT / Czech Republic
Volker HÖMBERG / Germany
Tamas Z. KINCSES / Hungary
Amos KORCZYN / Israel
Steven LAUREYS / Belgium
Dafin F. MUREŞANU / Romania
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Hari Shanker SHARMA / Sweden
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THE SOCIETY FOR THE STUDY OF NEUROPROTECTION AND NEUROPLASTICITY

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GENERAL INFORMATION

Congress Venue

INTERCONTINENTAL BUCHAREST
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All materials and documentation will be available at the registration desk located at SSNN booth. The staff will be pleased to help you with all enquiries regarding registration, materials and program. Please do not hesitate to contact the staff members if there is anything they can do to make your stay more enjoyable.

GENERAL INFORMATION

Language

The official language is English.
Simultaneous translation will not be provided.

Changes in Program

The organizers cannot assume liability for any changes in the program due to external or unforeseen circumstances.

Name Badges

Participants are kindly requested to wear their name badge at all times during the congress. The badge constiutes admission to the scientific sessions, coffee breaks and lunches.

Final Program & Abstract Book

The participants documents include the final program and abstract book which will be handed out together with the congress bag at the registration desk.

Speakers Corner

Speakers are asked to hand in their CD-ROM or USB stick containing the PowerPoint presentation (IBM format or compatible) preferably one day before their session but at the latest 90 minutes prior to the presentation.
The presention will be transfered to the central congress server. Due to time and technical reasons we kindly ask the speakers not to use their own notebook.
PC working stations are provided in the speakers center where speakers can also work on their PC charts in a quiet area. Tehnical staff will be glad to assist.

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DINNERS, LUNCHES & BREAKS

September 3, 2010

20:30 – 23:30 - **Welcome Reception** / Fortuna Ballroom

September 4, 2010

11:00 – 11:30 - **Coffee Break** / Foyer Ronda

13:10 – 14:10 - **Lunch** / Fortuna Ballroom

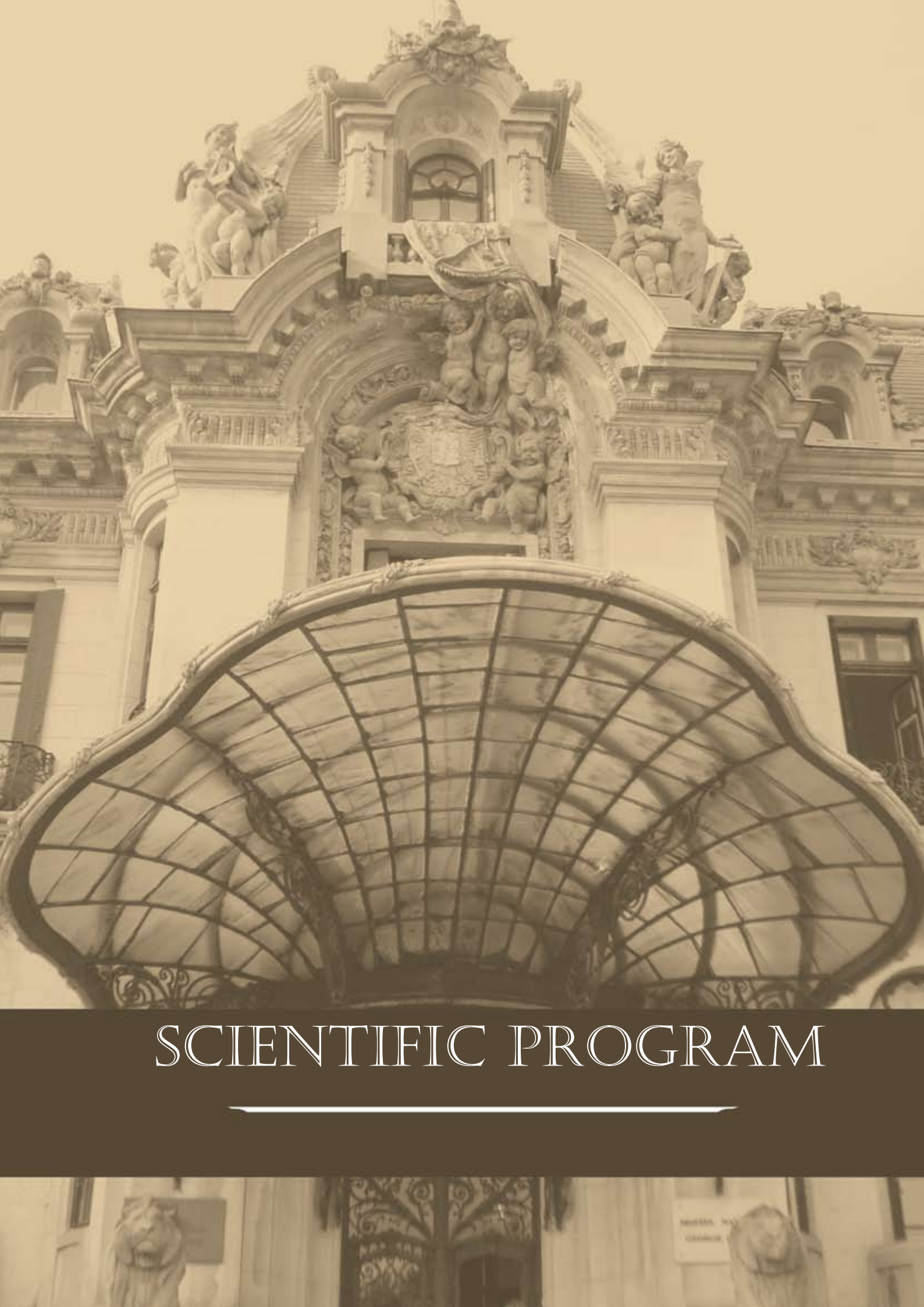
16:50 – 17:20 - **Coffee Break** / Foyer Ronda

20:30 – 23:30 - **Gala Dinner** / Fortuna Ballroom

September 5, 2010

10:20 – 10:50 - **Coffee Break** / Foyer Ronda

12:30: - **Lunch** / Foyer Ronda



SCIENTIFIC PROGRAM

SCIENTIFIC PROGRAM

SATURDAY / 4 September 2010

09:00 – 09:10	**Welcome Address**: Laurenţiu M. Popescu (Romania), Dafin F. Mureşanu (Romania), Florian Popa (Romania)	
09:10 – 09:40	Opening Lecture Dafin F. Mureşanu (Romania)	REAPPROACHING THE CONCEPTS AND REDESIGNING THE CLINICAL TRIALS FOR BRAIN PROTECTION AND RECOVERY
Session 1 – Dementia (I) / Chairmen: Raul Arizaga (Argentina), Jakub Hort (Czech Republic)		
09:40 – 10:00	Philip Scheltens (The Netherlands)	RETHINKING THE DESIGN OF CLINICAL TRIALS IN AD
10:00 – 10:20	Antón Álvarez (Spain)	ALZHEIMER'S DISEASE: PLEIOTROPIC AND MULTIMODAL TREATMENT FOR A MULTIFACTORIAL DISORDER
10:20 – 10:40	Amos Korczyn (Israel)	WHY HAVE WE FAILED TO FIND A CURE FOR AD?
	Discussion – 15 minutes	
11:00 – 11:30	**Coffee Break**	
Session 2 – Dementia (II) / Chairmen: Amos Korczyn (Israel), Philip Scheltens (The Netherlands)		
11:30 – 11:50	Angel Cedazo-Mínguez (Sweden)	APOLIPOPROTEIN E, CHOLESTEROL AND ALZHEIMER'S DISEASE
11:50 – 12:10	Jakub Hort (Czech Republic)	HUMAN ANALOGUE OF MORRIS WATER MAZE IN THE ASSESSMENT OF INDIVIDUALS AT RISK OF ALZHEIMER DISEASE
12:10 – 12:30	Antonio Federico (Italy)	RARE NEUROLOGICAL DISEASES: SIENA EXPERIENCE IN DIAGNOSIS, TREATMENT, RESEARCH AND TEACHING
12:30 – 12:50	Raul Arizaga (Argentina)	COGNITIVE IMPAIRMENT AND DEMENTIA: PATHWAYS AND BARRIERS
	Discussion – 15 minutes	
13:10 – 14:10	**Lunch**	

SATURDAY / 4 September 2010

Session 3 – TBI / Chairmen: Pieter Vos (The Netherlands), Antón Álvarez (Spain)

14:10 – 14:30	Pieter Vos (The Netherlands)	ARE S100B AND GFAP GOOD BIOMARKERS OF MODERATE AND SEVERE TRAUMATIC BRAIN INJURY?
14:30 – 14:50	Franz Gerstenbrand (Austria)	THE VERTEBRAL SPINE AND NEUROLOGICAL DISTURBANCES, DIAGNOSIS AND TREATMENT
14:50 – 15:10	Alexandru V. Ciurea (Romania)	EARLY NEUROPROTECTION AND RECOVERY IN SEVERE TRAUMATIC BRAIN INJURY
	Discussion – 15 minutes	

Session 4 – Neurorehabilitation (I) / Chairmen: Franz Gerstenbrand (Austria), Heinrich Binder (Austria)

15:30 – 15:50	Klaus von Wild (Germany)	HRQOL, HEALTH RELATED QUALITY OF LIFE, FOLLOWING TBI IN ADULTS. THE NEUROSURGEONS PERSPECTIVE.
15:50 – 16:10	Volker Hömberg (Germany)	MOTOR REHABILITATION: WHAT CAN WE LEARN FROM BASIC SCIENCE?
16:10 – 16:30	Tamás Z. Kincses (Hungary)	INVESTIGATION OF PLASTICITY TO DEVELOP NOVEL REHABILITATION APPROACHES
	Discussion – 15 minutes	

16:50 – 17:20	**Coffee Break**

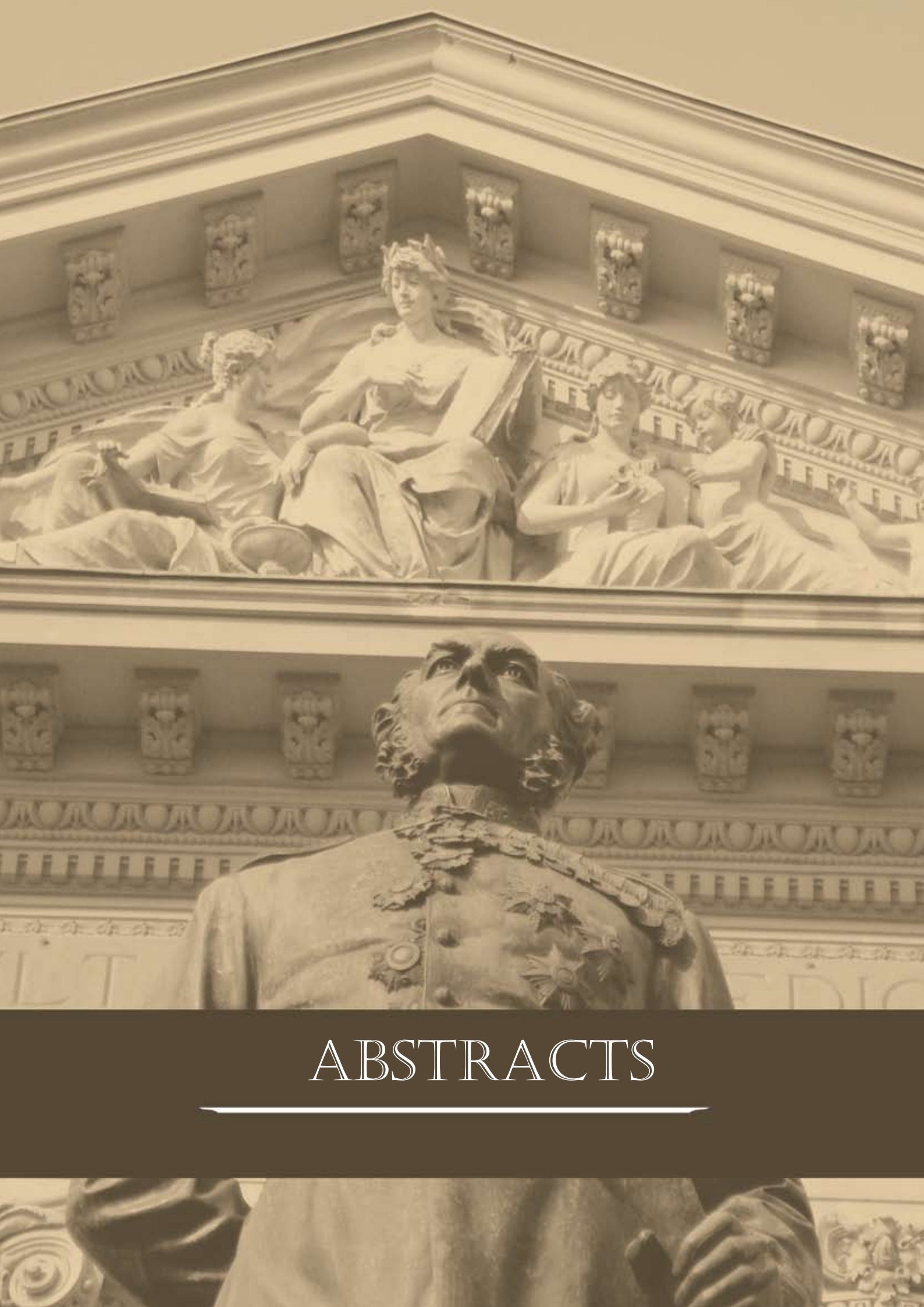
Session 5 – Neurorehabilitation (II) / Chairmen: Klaus von Wild (Germany), Volker Hömberg (Germany)

17:20 – 17:40	Heinrich Binder (Austria)	CONCEPTION OF CONSCIOUSNESS IN NEUROREHABILITATION
17:40 – 18:00	Steven Laureys (Belgium) represented by Camille Chatelle	CONSCIOUSNESS IN COMA AND RELATED STATES
	Discussion – 15 minutes	

20:30	**Gala Dinner**

SUNDAY / 5 September 2010

Session 6 – CNS Injury, protection and plasticity / Chairmen: Hari Shanker Sharma (Sweden), Ovidiu Băjenaru (Romania)		
09:00 – 09:20	Hari Shanker Sharma (Sweden)	HYPERTENSION, DIABETES OR NANOPARTICLES EXPLOSURE AS DISEASE MODIFYING FACTORS EXACERBATE PATHOPHYSIOLOGY OF HYPERTHERMIA INDUCED BRAIN DAMAGE AND ATTENUATE NEUROPROTECTIVE EFFICACY OF THERAPEUTIC AGENTS
09:20 – 09:40	Ovidiu Băjenaru (Romania)	PATHOPHYSIOLOGICAL MECHANISMS OF NEUROPATHIC PAIN
09:40 – 10:00	Moussa Youdim (Israel)	WHY IS NO WIN FOR ALZHEIMER'S AND PARKINSON'S SYNDROMES AND THE NEED FOR MULTIMODAL DRUGS
	Discussion – 15 minutes	
10:20 – 10:50	**Coffee Break**	
Session 7 – Dementia (III) / Chairmen: Antonio Federico (Italy), Moussa Youdim (Israel)		
10:50 – 11:10	Stavros J. Baloyannis (Greece)	THE PHILOSOPHY OF DEMENTIA
11:10 – 11:30	Bogdan O. Popescu (Romania)	IMPACT OF THE BLOOD BRAIN BARRIER ALTERNATIONS ON NEURODEGENERATION
11:30 – 11:50	Luiza Spiru (Romania)	PRODROMAL COGNITIVE IMPAIRMENT (PCI) - A KEY NOTION FOR DEMENTIA MANAGEMENT. CUTTING-EDGE INSIGHTS
	Discussion – 15 minutes	
12:15 - 12:30	**Closing Remarks**	Dafin F. Mureşanu (Romania), Laurenţiu M. Popescu (Romania)
12:30	**Lunch**	



ABSTRACTS

ALZHEIMER'S DISEASE: PLEIOTROPIC AND MULTIMODAL TREATMENT FOR A MULTIFACTORIAL DISORDER

ANTÓN ÁLVAREZ

DEPARTMENT OF NEUROPHARMACOLOGY, EUROESPES BIOMEDICAL RESEARCH CENTRE, A CORUÑA, SPAIN

Alzheimer's disease (AD) is a complex neurodegenerative disorder characterized by a multifactorial etiopathogenesis, involving gene mutations and genetic risk factors, abnormal processing of beta-amyloid (AB) and tau proteins, dysregulation of neuroimmune mechanisms, deficits of neurotrophic factors and alterations in several neurotransmitters (acetylcholine, noradrenaline, serotonine, glutamate). The interaction of these pathogenic factors produces a loss of neuronal contacts in early AD stages leading to apoptosis/degeneration of neurons, and finally to brain atrophy.

Since the etiopathogenesis of AD is multifactorial, its treatment should target concomitantly different levels of the AD pathogenic process. The development of drugs with pleiotropic and multimodal activity represents a very promising approach to achieve a disease-modifying treatment able to stop the progression, and eventually the onset of AD. At the present, two of the main therapeutic strategies for AD are: (1) Agents with neurotrophic activity; and (2) anti-amyloid treatments.

Neurotrophic factors mediated mechanisms influence molecular processes relevant for neuronal survival, degeneration and apoptosis, neurogenesis, synaptic plasticity, angiogenesis, and amyloid deposition into the brain. In addition, a deficit of neurotrophic factors is an early pathogenic event contributing to the degeneration of cholinergic neurons in AD. Therefore, the use of compounds with neurotrophic activity might increase and/or prolong the limited long-term efficacy of cholinesterase inhibitors (ChEIs) by protecting cholinergic neurons from degeneration.

A candidate for multimodal treatment in AD is Cerebrolysin, a brain neurotrophic preparation consisting of low molecular weight peptides able to cross the blood brain barrier. Experimental studies showed that Cerebrolysin acts as a pleiotropic drug, displays NGF- and BDNF-like activity against pathological cascade (antiexcitotoxic, antiapoptotic, inhibits reactive neuroinflammation; reduces APP phosphorylation, cerebrovascular amyloidosis and tau pathology in transgenic mice by modulating GSK3 β and CDK5 activity). It also excert a multimodal effect: neurotrophic activity (protects against neuronal degeneration and synaptic loss), neuroplastic activity (including synaptogenesis); promotes neurogenesis in vitro and in vivo. The overall effect is learning and memory improvement. Several clinical studies demonstrated the safety and the efficacy of Cerebrolysin as a monotherapy in AD. According to results of a recent clinical trial, it seems that the combined therapy with trophic (Cerebrolysin) and cholinergic (donepezil) drugs might provide long-term clinical benefits for AD patients. A synergistic effect of this combination treatment on AD clinical outcome is suggested, and is supported by research findings of its influence on the circulating levels of inflammatory and trophic factors.

COGNITIVE IMPAIRMENT AND DEMENTIA: PATHWAYS AND BARRIERS

RAÚL LUCIANO ARIZAGA

COGNITIVE NEUROLOGY UNIT, NEURAXIS INSTITUTE, NEUROLOGICAL FOUNDATION, BUENOS AIRES, ARGENTINA

Increased life expectancy along with the decline in birth rate motorize a worldwide population aging process. This process involves an increase in chronic non-communicable diseases related to age. Among them, brain diseases manifested by disorders of cognition and behavior have a marked impact.

This problem will increase in coming decades. This presentation aims to update and analyze the different pathogenic mechanisms responsible for cognitive impairment, as well as factors that act with a protective role. That way we will analyze the role of the risk factors leading to deterioration through vascular and neurodegenerative processes and their interrelationships but also, those factors that, through neuroplasticity and neurotrophism modify or moderate the impact of neurodegenerative processes.

Issues that affect the brain reserve and cognitive reserve will be discussed. Finally, the presentation will focus on the therapeutic resources available and the prospect that, in the field, shows the near future.

THE PHILOSOPHY OF DEMENTIA

STAVROS J. BALOYANNIS

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Throwing light on the fundamental nature of mind and understanding cognition, from the philosophical viewpoint, became a main subject of the philosophy of the neurosciences. It is reasonable, that in neurophilosophy dementia attracts the attention of scientifically-oriented philosophers, who search for a biological explanation of cognitive decline. Although the morphological and functional alterations of the dendritic spines may explain the dementia, however they can not give a philosophically acceptable answer in the fundamental problem of the Self in dementia. Is the Self independent of the mentality or it depends on the idea of the enduring self? Reasonably, the memory loss in dementia detaches the Self from the past and isolates it in the limited time of the present, without further perspectives.

However, we do not know the dimensions of the interior aspect of time in patients, which may be important in the duration and integration of the Self. Neuropsychological evidence demonstrates that demented persons can preserve the personality knowledge and their identity, which may be functional even in lack of memory. Personal identity may be based on concept of the body and on the character of the interior life. An important issue in dementia is the quality of the interior life and the moral principles of the amnesiac person. The interior life is understandable by the verbal, artistic and social behaviour. The criteria, therefore, are based on the phenomena and not on the existential dimensions and the substrate of the Self. The depression in the initial stages of the dementia may reflect the grief for the ongoing dysfunction, the loss of perspectives and the lack of the self-organization. The insight of the mental tragedy is an evidence of active interior life, even in the advanced dementia. The concept of the good and evil, the dignity and the moral aspect of the life are unaffected by the cognitive impairment in the majority of the cases.

The neuroscientists endeavour to approach the philosophy of dementia with scepticism, emphasizing the importance of the manifestations and the neuropathological alterations of the brain. We have the feeling that a phenomenological approach of dementia is not sufficient for a deeper understanding of the Self in dementia. A detailed analysis of the interior life of the individual would be required in order assess the continuation of the Self and the integrity of the Being during the course of dementia.

PATHOPHYSIOLOGICAL MECHANISMS OF NEUROPATHIC PAIN

OVIDIU BAJENARU

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Neuropathic pain is one of the most distressing aspects in patients with many chronic neurological conditions, and some times extremely difficult to treat, with a high negative impact on patients' quality of life, but also on their neurorehabilitation. Understanding its biology and pathophysiology offers an important pathway for an efficient therapeutic approach.

The peripheral and central sensitization leading to the development and maintenance of neuropathic pain, are key-elements in this complex biological network. This phenomena are the consequence of disturbed cellular mechanisms mainly in the dorsal horn of the spinal cord and probably in structures located in the rostral part of the brainstem and diencephalic structures, in relation with specific nuclei in the basal ganglia, responsible for the adaptive behaviour to pain perception.

Recent data emphasize an abnormal neuronal plasticity and an important glial loss in the dorsal horn of the spinal cord leading to abnormal synaptic activity and neuronal transmission of the information on the pain pathways in the central and peripheral nervous system. All these molecular and cellular events are potential targets for certain drugs or other modalities of therapeutic approach during the neurorehabilitation program of these patients.

CONCEPTION OF CONSCIOUSNESS IN NEUROREHABILITATION

HEINRICH BINDER

LANDSTEINER INSTITUTE FOR NEUROREHABILITATION AND SPACE MEDICINE VIENNA, AUSTRIA

Consciousness is an umbrella term which relays on a bundle of primarily subjective experienced conditions of the world and mental states. The content and definition of consciousness is a common challenge many disciplines are disputing for a long time.

Consciousness, above all absent of consciousness or in other words coma is one of the most important vital signs in neurology, because it is assumed to be an evidence for a life-threatening process and therefore from prime importance regarding therapeutic decisions. Usually medicine equates consciousness with alertness. But this is the improper approach. Because of ambiguous categorization we have to proceed rather on the assumption of continuous or gradual class belonging.

There is an error to believe in an existing clinically implementable definition regarding consciousness. While classification in application of strict standards seems impossible for now, implementable definitions from clinical as well as scientific point of view are urgently necessary.

APOLIPOPROTEIN E, CHOLESTEROL AND ALZHEIMER'S DISEASE.

ANGEL CEDAZO MÍNGUEZ

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One of the distinguishing features of Alzheimer's disease (AD) pathology is the deposition of beta-amyloid peptide (Aβ) in the brains of patients. There is a considerable understanding of the mechanisms by which the rare genetic mutations lead to Aβ overproduction, but the precipitating factors that lead to excessive Aβ levels in the much more common sporadic form of AD are still unknown.

A series of genetic, in vitro, in vivo and epidemiologic observations have all highlighted the importance of cholesterol metabolism in modulating AD risk and pathogenesis. Indeed, the major gene associated with the late-onset sporadic form of AD is the epsilon 4 allele of apolipoprotein E (apoE). ApoE is a cholesterol transport protein. In vitro studies have shown that intracellular cholesterol levels can modulate amyloid precursor protein (APP) processing and that the cleavage of APP by β & β-secretases occurs in cholesterol-rich microdomains within the neuronal membranes. Thus, it has been hypothesized that modulating levels of neuronal cholesterol may have therapeutic potential for the treatment of AD.

In addition, new findings on cholesterol metabolites (such as 27-OH and the brain-produced 24S-OH) suggest that these molecules are much more than subproducts of the cellular metabolism, and can be regarded as signalling molecules of importance for normal brain function as well as for neurodegenerative processes, including AD.

In this lecture, I will review what is known about how cholesterol metabolism and apoE contribute to AD pathology, as well as which target possibilities they offer for developing new treatments for AD.

CONSCIOUSNESS IN COMA AND RELATED STATES

CAMILLE CHATELLE / STEVEN LAUREYS

COMA SCIENCE GROUP, UNIVERSITY OF LIÈGE, BELGIUM

Following severe brain damage and coma some patients may loose all brain and brainstem functions and evolve to brain death while others may awaken (i.e., open their eyes) but will only show reflex behavior (i.e. they are in a vegetative state). The vegetative state is a dramatic dissociation of consciousness' two main components: arousal (which is preserved) and awareness (which is abolished). Some patients will remain vegetative for decades other may evolve to a minimally conscious state, which means they show more than simple reflex behavior but can nevertheless not communicate verbally or non-verbally. Finally, coma patients may awaken, being fully aware but paralyzed and mute, (i.e., they are in a locked-in syndrome).

Is awareness lost when overall cortical activity falls bellow a certain threshold? In the vegetative state, global metabolic activity decreases to about 50% of normal levels - similar to what is observed in sleep, anesthesia and coma. However, in patients who recover, global metabolic rates for glucose metabolism do not necessarily show substantial recovery. Hence, the relationship between global levels of brain function and the presence or absence of awareness is not absolute. It seems that some areas in the brain are more important than others for its emergence. We have identified these regions showing metabolic dysfunction when the vegetative state was contrasted to the conscious resting state in healthy controls. These studies have identified a dysfunction not in one brain region but in a wide frontoparietal network encompassing the polymodal associative cortices : bilateral dorsolateral prefrontal, parieto-temporal and mesiofrontal, posterior cingulate and precuneal cortices. Similar results have later been found for other diseases where patients also are seemingly 'wakeful' but only show reflex automatic behavior lacking 'voluntary interaction with others - such as absence and complex partial seizures and sleepwalking. Current analyzing techniques now also permit to assess awareness-related changes in functional integration - that is measuring differences in functional cerebral connectivity between vegetative patients and healthy controls. Awareness seems not exclusively related to the activity in the frontoparietal network but, as importantly, to the functional connectivity within this network and with the thalami. Indeed, long-range cortico-cortical and cortico-thalamo-cortical 'functional disconnections' have been identified in the vegetative state. Moreover, recovery is paralleled by a functional restoration of the frontoparietal network and part of its cortico-thalamo-cortical connections. In addition to measuring resting brain function and connectivity, neuroimaging studies have identified which brain areas still "activate" during external stimulation. Studies using external (pain or auditory) stimulation showed robust activation in subcortical and primary sensory cortex that was however isolated and dissociated from the frontoparietal cortical network. The activation in primary cortices in awake but unaware patients confirms the hypothesis that neural activity in primary cortices is necessary but not

sufficient for awareness. In our view, conscious awareness is an emergent property of the collective behavior of neuronal ensembles in the frontoparietal global neuronal workspace. Within this thalamo-cortical network, recent studies in healthy volunteers have recently shown that external (sensory) awareness depends on lateral prefrontal and posterior parietal cortices while internal (self) awareness correlates with precuneal and mesiofrontal midline activity. Of clinical importance, this knowledge now permits to improve the diagnosis and pain management of patients with disorders of consciousness, which remains very challenging at the bedside. Current technology now also permits to show command-specific changes in EEG or fMRI signals providing motor-independent evidence of conscious thoughts. Such brain computer interfaces now permit communication via voluntary EEG or fMRI control, enabling locked-in patients to control their surroundings and possibly giving a voice to some minimally conscious state patients.

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EARLY NEUROPROTECTION AND RECOVERY IN SEVERE TRAUMATIC BRAIN INJURY

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A.V. CIUREA

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Background: Traumatic brain (TBI) and spinal cord injuries (SCI) are two of the most devastating types of injuries, especially in young people in all official data. The Centers for Disease Control and Pre¬vention (USA) report that at least 15 million people sustain TBIs in the US annually, far more than the number of people af¬fected by breast cancer, human immunodeficiency virus/ac¬quired immunodeficiency syndrome, and multiple sclerosis combined. The estimated cost of TBI-related hospitalizations is $56.3 billion every year.

TBI delimitation was perfomed by Teasdale and Jennett (1974), in Glasgow Coma Scale (GCS), important standard in the assessment of these brain lesions: minor (13-15), moderate (12-9), severe (8-3). This standard ("golden") scale in TBI was established by motor (1-6p.), verbal (1-5p.), eyes (1-4p.) response at external stimuls. Severe brain injuries (GCS 3-8) represent an important cause of mortality and morbidity, especially in patients with active period of live (20-40 years old).

Material & Method: Severe brain injuries (GCS 3-8) represent an important cause of mortality and morbidity, especially in patients with active period of live (20-40 years old). Included criteria: the authors studied non selected consecutive 88 patients with SBI (between 6 – 66 years old), 53 male and 35 female in period 2003-2007 (5 years) at the Hospital "Bagdasar-Arseni", Bucharest. The distribution by age was children 30 cases (34,1%) and adults 58 cases (65,9%). The most frequent cause of SBI is represented by the car accidents (car to pedestrian, passenger vehicle) 58 cases (65,9%), followed by falls different higher 23 cases (26,1%) domestic accidents 4 cases (4,5%) and sport traumas 3 cases (3.4%). All intracranial haematoma was operated in the first 6 hours after admission. Excluded criteria: all patients in SBI status with multiple trauma with or without intracranial haematomas. All 88 cases were monitoring in intensive care unit (ICU). At admission GCS 3-4 was 26 cases (29,5%), GCS 5-6 was 25 cases (28,4%), GCS 7-8 was 37 cases (42%). In all cases the admission CT scan was performed in the first 6 hours; The following CT scan was performed at 24, 48, 72 hours and after 1 week to verified the brain lesion and intracranial mass lesion. In 30 cases (34,1%) intracranial mass lesions undergone to the operative procedures: extradural haematoma 14 cases (15,9%), subdural haematoma 10 cases (11,3%), intraparenchimal haematoma 6 cases (6,8%). Additional in 10 cases (11,3%) we report penetrated head injury. Also, CT scan showed hemhorragic contusion 23/88 (26,1%) SAH in 27/88 cases (30.7%), hypodense (ischemic aria lesion in 25/88 cases (28.4%), cerebral edema 40/88 cases (45.5%) and DAI 19/88 cases (21,6%); DAI was diagnosis only by MRI and the first week post-injury. In our data surgical evacuation of mass lesions was performed as needed, but only five decompression craniotomy was done. In our study no mortality was registered in the group of ICP < 20 mmHg, all the 28/88 cases (31.8%) which died had the ICP > 20 mmHg.

In the literature there are studies which correlate the GOS with GCS, metabolic, hematological, radiological and clinical profiles.

The predictors outcome factors in this series were: early neurotrophic drugs (e.g. Cerebrolysin) and active neurorehabilitation were done immediately after admission and neuroimaging diagnosis in intensive care unit. In our experience the Cerebrolysin are an significant improvement in post TBI. Cerebrolysin (mixture of low molecular polypeptide, extracted from pig brain) ncrease motor function, enhance the cognitive performances, increase memory & attention, improve of brain bioelectrical activity. Also, early and active neurorehabilitation improve the brain activities and starts the redundant neural network. At admission GCS 7-8 was preponderent 37 cases (42%) which it was in concordance with the global outcome.

In our data Glasgow Outcome Scale (GOS): good recovery was in 25 cases (28,4%), moderate disability 9 cases (10,2%), severe disability 22 cases (25%), vegetative state 6 cases (6,8%), death 26 cases (29,5%). At admission GCS 7-8 was preponderent 37 cases (42%) which it was in concordance with the global outcome. The psychological support in all SBI will be necessary to obtain social, familial and professional integration.

Conclusions: The four early important therapeutical factors may improve the outcome in SBI. Neurotrophic factors (e.g. Cerebrolysin) can improving the global outcome. The psychological support in all SBI will be necessary in both: patient and his/her family, for increase compliance at treatment to a very good recovery or autonomy and to obtain social, familial and professional integration and, finally, to a high QoL.

Keywords: traumatic brain injury (TBI), GCS, severe brain injury (SBI), DAI, ICP, neuroprotection, Cerebrolysin, neurorehabilitation, outcome, GOS, QoL.

RARE NEUROLOGICAL DISEASES: SIENA EXPERIENCE IN DIAGNOSIS, TREATMENT, RESEARCH AND TEACHING

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Rare neurological diseases are an heterogeneous group of disorders mainly affecting central, peripheral nervous system and muscle, representing almost 50% of all the rare diseases, and indicating that neurologists are one of the main specialists involving in diagnosis and research. But the classical interest of neurologists is dedicated to the main common diseases as dementia, multiple sclerosis, headache, epilepsy, stroke, avoiding to follow these diseases, that have, taken togheter, an high impact on the health system in Europe as well as in all countries. Rare diseases are also considered orphan diseases, since for few of them a treatment exists. In Europe, like in USA, in the recent years, a great interest has been dedicated to such disorders and the organization of dedicated care systems have been stimulated. In fact, the difficulty of the diagnosis and the need of superspecialization on this topic, lead to the organization of few Centres in the different Countries, that can collect patients and organize a network for diagnosis, treatment and research.

We will report our experience in Siena, as a reference Centre for these disorders, for diagnosis and treatment. We will discuss also our experience in teaching in medical school and in PhD programme, for qualification researchers in these topics. Since rare neurological diseases are a nice model for understanding the pathogenesis of more common nervous system dysfunctions, we will report some our research data on the pathogenesis of mitochondrial, lysosomal and peroxisomal diseases and also in some neurogenetic conditions, some of them leading to innovative treatments. Finally, data on the activity of an Information Centre that is able to give informations to patients and doctors will be reported.

THE VERTEBRAL SPINE AND NEUROLOGICAL DISTURBANCES, DIAGNOSIS AND TREATMENT

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The term vertebral spine goes back to the description period of the anatomy. As a consequence of the central position in the human body the spinal column should be called the "Human axis organ". The centre for the function for the axis organ is located in the brain stem using the postural and the turning reflexes, based on the stimulation of the proprioceptive system. Main receptors of the proprioceptive system are the mechano-receptors of the joints and the muscles of the extremities and the vertebral spine, supported by foot sole receptors. The human axis organ is the basis of static and kinetic functions of the human body. The axis organ is carrying the body, the extremities and the head. The inner organs and the thorax with the breathing system are fixed on the axis organ. The spinal cord is located in the spinal channel of the vertebral spine.

In the phylogenesis the upright position of the human race and the following development of manhood the bridge-bow-construction of quadrupeds had to be changed in the lattis tower system. The filigree vertebral bones and the vulnerable discs have to carry the weight of the body and the head using the "arc function model".

The situation of modern men, " the homo sedens", with the functional overload in the non-physiological body position produces a continuous damage in all parts of the vertebral spine. In addition psychological factors are influencing position and movements of the body. Degenerative changes on vertebrates, discs and vertebral joints are the consequence, causing typical complaints in form of a radicular syndrome, pseudoradicular symptoms and the referred pain syndrome as well as spinal cord deficits and cauda symptoms. The spondylogenic cervical myelopathy is a sequence of a vertebrostenosis in the cervical spine, often misdiagnosed, Changements of a spondylolisthesis especially in the lumbar spine region are a problem in diagnosis and in treatment. The "whiplash injury" of the cervical spine can be followed by chronic complains based on degenerative changements of the cervical spine.

Special neurological examination including the method of the manual therapy (neuro-orthopaedy), together with X-ray examination, completed by the magnetic resonance method and an electrophysiological examination have to be used for an exact diagnosis and a special treatment programm which has to be carefully prepared and consequently executed by special trained physiotherapists. For the decision to a surgery intervention a careful consultation between the different specialists (neurology, orthopaedic, neuro-surgery) is necessary.

HUMAN ANALOGUE OF MORRIS WATER MAZE IN THE ASSESSMENT OF INDIVIDUALS AT RISK OF ALZHEIMER DISEASE.

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Patients with Alzheimer's Disease (AD) frequently have difficulties with spatial orientation in real life. AD is preceded by amnestic Mild Cognitive Impairment (MCI). However, MCI patients form a very heterogeneous population with a various risk of conversion to AD. We examined the characteristics of spatial navigation impairment in different MCI subgroups.

Seventy-eight patients attending our Memory Disorders Clinic were divided into seven groups: probable mild AD (n=21), MCI, further classified according to Petersen's criteria as amnestic MCI single domain (aMCIsd) (n=11), multiple domain (aMCImd) (n=31), or non-amnestic MCI (naMCI) (n=7), and Subjective Memory Complaints (SMC) (n=8). The aMCI group was also stratified using cued recall according to Dubois's criteria into memory impairment of hippocampal type - hippocampal aMCI (HaMCI) (n=10) and isolated retrieval impairment - non-hippocampal (NHaMCI) (n=32). Furthermore the aMCI group was classified according to ApoE4 status into ApoE4 postitive (ApoE4+) (n=12) and ApoE4 negative (n=30). These patients, together with controls (n=28), were tested in a human variant of the Morris Water Maze requiring them to locate an invisible goal inside a circular arena 2.9 m in diameter. Depending on the subtest, the subjects could use the starting position and/or orientation cues on the wall for navigation.

The subtests were thus focused on egocentric and allocentric (hippocampus-dependent) navigation. The AD and aMCImd groups were impaired in all subtests. The aMCIsd group was significantly impaired in subtests focused on allocentric orientation. The HaMCI group performed poorer than the NHaMCI group and was similar to the AD group on all subtests. The ApoE4+ aMCI group outperformed the ApoE4- aMCI group and resembled the AD group on all subtests. Our results suggest that spatial navigation impairment occurs very early in the development of AD. Spatial memory assessment can be profitable for longitudinal monitoring of the disease progression or evaluation of presymptomatic individuals at risk of AD.

The aMCI individuals form a very heterogeneous population and spatial memory or cued recall examination can add more value to aMCI classification. The better understanding of MCI heterogeneity might help to organize clinical trials in the earlier stages of AD.

MOTOR REHABILITATION: WHAT CAN WE LEARN FROM BASIC SCIENCE?

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Within the last decade there has been the enourmos growth in neurobiological knowledge. Fortunately attempts have been made to make this knowledge applicable to day by day clinical practice. In this talk several examples of a fast transfer from laboratory work to bedside use will be described for the field of motor retraining. Examples will be presented from knowledge transfer about learning by imitation, imagination and avoiding learned non use. Elementary rules derived from animal and human studies of motor learning will be discussed (role of repetition, shaping, feedback etc.). Such a transfer enables a fast and innovative development for the improvement of therapeutic techniques.

Marshalling motor rehabilitation according to such elemantary rules enables a reasonable compromise between concepts of evidence based medicine and an individualized treatment for patients.

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INVESTIGATION OF PLASTICITY TO DEVELOP NOVEL REHABILITATION APPROACHES

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Adaptive reorganisation during recovery after brain injury shares common mechanisms with those processes for motor learning in healthy brain. These processes include plastic changes at the molecular, cellular, regional and at network level. We showed in an MR spectroscopy study that GABA neurotransmission is modulated during motor learning. With a model-free fMRI analysis approach we identified the functional networks that are related to different aspects of skill acquisition. In a consecutive study we mapped the white-matter structural networks that contribute to such learning. Similarly, in stroke patients white matter microstructure can have a predictive value of the successfulness of rehabilitation.

In a next set of investigations we modulated the cortical excitability to improve visuo-motor learning by transcranial direct current stimulation (tDCS). The polarity sensitive modulation of cortical neurotransmitters that share common features with those seen during motor learning were identified by MR spectroscopy. Functional MRI investigations showed that besides the local effect of external electrical stimulation a whole network of areas change activity during tDCS.

These results mark out the pathway to a new set of studies identifying a more effective rehabilitation approaches.

WHY HAVE WE FAILED TO FIND A CURE FOR AD?

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There is widespread recognition in the urgency to understand the causes and mechanisms of senile dementia. Attempts to find cures for Alzheimer's disease (AD) have, however, failed so far, in spite of enormous investments, intellectual and financial. We therefore have to reconsider the problem from new angles. AD is regarded as a disease because of its clinical manifestations and underlying pathology. However, this combination does not define a disease but rather a syndrome, just like hepatic cirrhosis in which liver pathology causes metabolic changes, but which can result from many different etiologies. It is unlikely that attacking a downstream phenomenon, like apoptosis or β - amyloid accumulation, can cure AD, or prevent the progression of the disease.

Epidemiological studies have identified many risk factors for "senile dementia of the Alzheimer type", some genetic but most environmental and therefore modifiable. Thus, it is probable that AD is the result of a combination of several processes, working differently in each person. Therefore a concerted action to fight the dementia epidemic must be made by aggressive action against its risk factors, and this battle must begin in midlife, not in old age.

REAPPROACHING THE CONCEPTS AND REDESIGNING THE CLINICAL TRIALS FOR BRAIN PROTECTION AND RECOVERY

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The old concept that neuroprotection means suppressing pathophysiological processes, the idea that a single mechanism molecule might be effective in clinical practice are obsolete today, and represents the root cause of failure in clinical neuroprotection.

The effects of etiological agents on the brain traditionally are conceived as a linear sum of independent pathophysiological processed (excitotoxicity, inflammation, apoptosis-like, oxidative stress, etc) generating the pathways of pathological cascades (ischemic, traumatic, neurodegenerative).

The pathway approach has produced a very detailed understanding of molecular changes in the postlesional brain but it possesses blind spots that are critically related to the failure of neuroprotection.

This has generated the simplistic way of understanding the concepts and as well, all attempts at clinical neuroprotection. The idea that a system is a linear sum of its component parts is called "superposition", and the associated approach is called "reductionism".

The failure of clinical neuroprotection, recovery and modifying disease therapies in many chronic conditions, is measuring the failure of the reductionistic approach to the problem.

The pathways can and do interact in a variety of fashions, via cross-talk, positive and negative feedback, etc, but the pathway heuristic itself offers no formal means of understanding such interactions.

The expectation of discovering the magic cell death pathway X has affected experimental designs of neuroprotection studies. The causality demonstrated by the application of the plus/minus strategy is ultimately an illusion. To overcome the limits of the pathway view of cell function, a different approach is needed.

Such an approach is provided by network concepts applied to complex systems.

The bistable model based on these assumptions seems to be a better instrument for a successful translational approach in brain lesion and recovery.

Current situation urge for consistent improvement in neuroprotection and neurorecovery clinical trials design.

IMPACT OF THE BLOOD BRAIN BARRIER ALTERATIONS ON NEURODEGENERATION

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Neurodegenerative diseases share a few common altered cellular and molecular mechanisms, such as accumulation of abnormal proteins within and outside the cells, mitochondrial dysfunction and oxidative stress, calcium homeostasis dysregulation, early synaptic disconnection and late apoptotic cell death. In main forms of dementia, a progressive loss of mental capabilities occurs during years and probably these symptoms are caused by much earlier abnormal molecular brain events.

Cognitive deterioration takes place in a mild form in ageing as well, possibly due to a global and progressive decrease of the functional brain reserve. Increased age is a risk factor for neurodegeneration and key pathological features of dementia are sometimes found in aged brains as well. An underexplored brain structure in ageing and dementia is the blood-brain barrier (BBB), which tightly regulates both influx and efflux of molecules reaching the central nervous system. BBB disruption was documented not only in brain vascular disease but also in ageing and various neurodegenerative disorders.

Expression of dementia-associated gene mutations in experimental models leads to change of BBB properties. A two-way pathogenic relationship seems to link brain and BBB in respect to cognition: discontinuation of the BBB can trigger malfunction and death of neurons and neuronal or glial pathology can impair BBB functions.

In this review I will discuss data which provide evidence of BBB alterations in conditions associated with cognitive decline and neurodegeneration.

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RETHINKING THE DESIGN OF CLINICAL TRIALS IN AD

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The methodology of randomized clinical trials in AD should be modified in line with experience from recent clinical trials and biomarker studies. To decrease heterogeneity, studies in mild to moderate dementia should separately analyze outcomes in older and younger patients because of differences in the underlying pathology.

Inclusion criteria in future studies should be restricted to clinical and biomarker profiles consistent with AD and sub-analyses should include quantitative assessments of vascular and Lewy body co-morbidity. Increasingly clinical trials for AD will move to milder stages of illness where arrest of brain pathology and delay in symptom progression may be more likely. In these studies emerging biomarkers will play a pivotal role in establishing target engagement and as surrogate markers of treatment response.

Disease-modifying compounds should be implemented in stages of the illness when they are most likely to succeed in order to fully test leading theories of AD pathogenesis. This approach may significantly modify clinical practice within ten years.

HYPERTENSION, DIABETES OR NANOPARTICLES EXPOSURE AS DISEASE MODIFYING FACTORS EXACERBATE PATHOPHYSIOLOGY OF HYPERTHERMIA INDUCED BRAIN DAMAGE AND ATTENUATE NEUROPROTECTIVE EFFICACY OF THERAPEUTIC AGENTS*

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Brain diseases are leading causes of death since time immemorial1. Healthy brain keeps healthy mind and body2,3. Thus, efforts should be made to understand the basic causes of brain disease while treating patients. In this context, understanding of the influence of patients external environment, e.g., air and water quality, as well as internal environment, viz., nutrition, co-morbidity and other organ deficiency must be considered while prescribing any treatment or medicine1-4. The main work of the physician is to cure the disease of the patient and not to focus on only reducing symptoms by various means. This is the essence of Ayurveda (Ayur = life; veda = knowledge), used to practice health care since 5000 BC3-6. The ideas and concepts were developed and crystallized by Maharishi (a scholar with great wisdom) Charak in Charak Samhita (the 1st Book on Medicine in the World) written in 800 BC2-4.

These words are still true in current medical practices. Thus, we still don't know much about the role of co-morbidity (disturbances in the internal environment, e.g., diabetes or hypertension) in relation to ambient environmental influences (alterations in external environmental, e.g., heat, microfine particles) as disease modifying factors. Furthermore, this is still not known whether these disease-modifying agents could also interfere with the neuropharmacological effects of therapeutic agents used to treat such diseases.

To understand these interactions between external and internal environmental factors in brain disease, we developed animal models of hypertension and diabetes as modulators of internal environment-10. Furthermore, we standardized animal models for heat exposure and/or nanoparticles treatment as modulation of external environments7,8. Using a combination of these factors we found that these disease-modifying agents (external or internal factors) could considerably influence the pathophysiology of brain injury and also affect the neuroprotective ability of therapeutic agents8-10.

Thus, we exposed rats to engineered nanoparticles from metals (Cu, Ag or Al, 50-60 nm, 50 mg/kg, i.p. once daily for 7 days) in normal, hypertensive or diabetic rats and examined pathophysiology of brain damage on the 8th day7. In addition, we exposed normal, hypertensive or diabetic rats to 4 h heat stress (38°C for 4 h) and examined brain pathology. Furthermore, a group of nanoparticles treated animals were also exposed to heat stress in identical manner8,9. Our observations show that nanoparticles or heat stress given alone induced neuronal. Glial cell and endothelial cell damage leading to blood-brain barrier (BBB= breakdown and edema formation in many parts of the brain. These pathological reactions in the brain were much more aggravated if they are combined with nanoparticles or heat exposure, or heat exposure in hypertensive or diabetic rats as well as nanoparticles exposure in hypertensive or diabetic rats at room temperature. These observations suggest that

brain pathology depends on both the intrinsic (hypertension or diabetes) as well as on extrinsic (nanoparticles exposure or heat). Furthermore, when we treated rats with cerebrolysin, growth hormone, antioxidant compounds (H-290/51 or EGB-761) their neuroprotective ability was more than 50 % reduced when the nanoparticles exposure was combined with heat stress, diabetes or hypertension. In addition, almost double dose of these compounds are need to achieve substantial neuroprotection in hypertensive or diabetic rats subjected to heat stress. Taken together our observation suggest that co-morbidity and environmental factor play important roles in the pathophysiology of brain diseases and the magnitude of neuroprotective efficacy by standard doses of the drugs. These observations open a new avenue of research that will have important bearings in clinical research and/or drug trials in future.

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PRODROMAL COGNITIVE IMPAIRMENT (PCI) – A KEY NOTION FOR DEMENTIA MANAGEMENT. CUTTING-EDGE INSIGHTS.

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Background: Actually, there is no dementia cure. Early detection of prodromal cognitive impairment (PCI) is crucial. It allows sufficient time for designing the most suitable protocol of drug and non-drug interventions when disease outcomes are still susceptible to interventions. Tailoring of this protocol close to the individual's clinical phenotype is also crucial, hence joining the Predictive, Preventive and Personalized Medicine (3P-Medicine) principles is imperative.

Method:We overview the cutting-edge insights into PCI early diagnosis, prognosis and personalized attempts, based on the analysis of recent scientific reports and on the outcomes of our studies and participation to international debates.

Results: Dementia (especially Alzheimer's) is a plurifactorial disease in which challenging environmental factors trigger its onset by acting on inner, predisposing abnormalities. This feature must be considered in PCI early diagnosis, prognosis, and drug and non-drug therapy. We present our NERO-MIND research paradigm dealing with this feature. The huge technological progress supports an unprecedented development of early detection markers, especially molecular (Abeta 41-42, tau, ACE, B12 Vitamin etc.) and imagistic (PET scan, fMRI). As partner in the EADC, we overview the main results of DESCRIPA study related to CSF markers prognostic value (Visser PJ et al., 2009), and to the development of screening guidelines and criteria (Visser PJ et al., 2008). The ongoing effervescence regarding PCI epigenetic definition, markers and epigenetic drugs design are also pointed out. Another imperative required by the huge interpersonal variability of symptoms bulk is the definition of phenotypical clusters, able to facilitate PCI personalized approach. The adjunctive therapy (e.g. ChEIs and memantine) also requires personalization.

Conclusions: Multifactorial approach is crucial for PCI attempt. Early PCI detection is crucial for symptoms improvement and slowing of their worsening rate. The actual scientific and technological progresses, as well as 3P medicine principles allow an optimistic outlook for PCI management.

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ARE S100B AND GFAP GOOD BIOMARKERS OF MODERATE AND SEVERE TRAUMATIC BRAIN INJURY?

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Background: Moderate and severe traumatic brain injury(TBI) are characterize by a high rates of case fatality (15-20% in moderate TBI and 40% in severe TBI) and disability in survivors (30-40% in moderate and 50-60% in severe TBI). Accurate determination of the initial severity of the primary brain damage is imperative in establishing a prognosis and to weigh risks and benefits of specific treatment options. Biomarker levels in blood after (TBI), may offer an objective diagnostic and prognostic tool in addition to clinical indices. In this study we aim to validate glial fibrillary acidic protein (GFAP) and S100B concentrations in blood as outcome predictors of TBI using cut off levels of 1.5 µg/l for GFAP and 1.13 µg/l for S100B from a previous study.

Methods: In 79 TBI patients (GCS score ≤ 12) serum, taken at hospital admission, was analyzed for GFAP and S100B. Data collected: injury mechanism, age, gender, mass lesion on CT, GCS, pupillary reactions, Injury Severity Score (ISS), presence of hypoxia and hypotension. Outcome was assessed, using the Glasgow Outcome Scale Extended (dichotomized in death versus alive and unfavorable versus favorable), 6 months post injury.

Results: In patients who died compared to alive, median serum levels were increased: GFAP 33.4 fold and S100B 2.1 fold. In unfavorable compared to favorable outcome, GFAP was increased 19.8 fold and S100B 2.1 fold. Univariate logistic regression analysis revealed that mass lesion, GFAP, absent pupils, age and ISS but not the GCS, hypotension or hypoxia predicted death and unfavorable outcome. Multivariate analysis showed that models containing mass lesion, pupils, GFAP and S100B were the strongest outcome predictors of death and unfavorable outcome. S100B was the strongest single predictor of unfavorable outcome with 100% discrimination.

Conclusion: This small study confirms that GFAP and S100B levels in serum are powerful adjuncts to the clinical assessment of brain damage and powerful predictors of outcome after TBI.

Keywords: severe head injury; outcome; Glasgow Outcome Scale; CT; severe traumatic brain injury; prognostic factor; S100B ; GFAP.

HRQOL, HEALTH RELATED QUALITY OF LIFE, FOLLOWING TBI IN ADULTS. THE NEUROSURGEONS PERSPECTIVE.

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Objective: Today, increasingly more patients with brain lesions survive the acute stage after the impact but suffering from severe impairment of higher cerebral functioning (WHO-ICF) in the long run. Mental- cognitive and behavioural disabilities are more persistent and constitute more of a handicap than do focal neurological signs. Physical and mental disabilities combine to produce a social or overall outcome regarding the patients' reintegration. Within the Spectrum of Neurorehabilitation acute trauma care and early neurorehabilitation have been shown to significantly reduce secondary and tertiary brain damage and to improve impaired functioning, while out patients treatment and community based continuous care and legal and social support will help social reentry over many years..

Methods: Functional impairments after TBI refer to loss of structures and functions. Disabilities refer to limitations or participating restrictions. Functioning is an umbrella term encompassing all body functions, activities and participation. Acute neurosurgical care and neurorehabilitation need a multidisciplinary team approach. This treatment is be based on neuroprotection, brain plasticity and various medical, physical, neuropsychological and social health care concepts. The new QOLIBRI tool can asses HRQOL by self report.

Results: The essential aspect of neurotrtauma care following TBI is the integration of all disciplines involved and a consistent goal setting to protect he brain from any secondary insult to regard individual patients' needs. This is to preserve brain plasticity and to support all processes of physical and mental cognitive recuperation in the follow up including surgical decompressive procedures and central acting drugs Good structural organization and cooperation, notice of basic communication rules, conflict management, and a definite decision making increase productive interdisciplinary working.
The QOLIBRI was demonstrated to be the first TBI-specific measure of HRQOL. It provides information about patients' subjective views of their own lives and complements traditional measures of disability and recovery (SF 36). It captures life satisfaction rather than health function being sensitive to disability and mental health, demographic and socioeconomic factors.Main predictors for HRQOL are Depression, Help needed, Health complaints, Anxiety, Disability

Discussion: Obviously the impairment of mental-cognitive and neurobehavioral f u n c - tioning and not loss of physical skills will cause patients' loss of life transactions and final outcome. Neurosurgeons started to play a major role within the multidisciplinary spectrum of neurorehabilitation after TBI right from the beginning as to improve HRQOL. The new assessment tool QOLIBRI can be applied across different populations- and cultures. It allows the identification of personal needs, the prioritization of therapeutic goals, and the evaluation of individual progress. It can be useful in clinical trials and in longitudinal studies of TBI recov-

ery, where neuroprotection, brain plasticity, functional assessment and the individuals medical and social legal care over many years have been shown to be crucial. .

Conclusion: Acute neurotrama care and functional rehabilitation is a process whereby patients who suffer from impaired higher cerebral functions after TBI regain their former abilities or, if full recovery is not possible, achieve their optimum physical, mental, social and vocational capacity. It aims at patients' social full reintegration. In order to facilitate such goals neurosurgeons should start with a multidisciplinary team approach as early as possible.
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References:

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WHY IS NO WIN FOR ALZHEIMER'S AND PARKINSON'S SYNDROMES AND THE NEED FOR MULTIMODAL DRUGS

MOUSSA B.H. YOUDIM

TECHNION-RAPPAPORT FACULTY OF MEDICINE, EVE TOPF AND NPF CENTERS OF EXCELLENCE EFRON HAIFA, ISRAEL

The mechanism(s) of neurodegeneration in Alzheinmer's disease (AD) and Parkinson's disease (PD), Huntington disease (HD) , amyotrophic lateral sclerosis (ALS) and multiple Sclerosis (MS) are not known, but they may be initiated by cascade of neurotoxic events, that includes oxidative stress, brain iron dysregulation, glutamate excitotoxicity, nitric oxide, inflammatory process, neurotoxic processing misfolding and aggregation of abeta-peptide, alpha-synuclein resulting from the possible demise of ubiquitin-proteasome system (UPS) as demonstrated neurochemically and by transcriptomics and proteomic profiling. AD and PD subjects are benefiting from symptomatic effects of cholinesterase inhibitors, memantine, monoamine oxidase A and B inhibitors, L-dopa, dopamine receptor agonists, COMT inhibitors and dopamine agonists, glutamate antagonist (memantine), anti viral drug (amantadine) developed to act on a single molecular target. For HD and ALS there are no effective drugs.

Such drugs have limited symptomatic activities and current pharmacological approaches have sever limitation in their ability to modify the course of the disease, offering incomplete and transient benefit to patients. However, the new therapeutic strategies for neurodegenerative diseases are those in which drug candidates are designed expressly to act on multiple neural and biochemical targets involved in the neurodegenerative process and to possesses neuroprotective and neurorestorative activities.

Thus, we have hypothesized and developed a series of innovative novel multifunctional drugs from our successful neuroprotective-neurorestorative, with possible disease modifying, anti Parkinson drug, rasagiline (Azilect, called, ladostigil (phase II clinical control study) and multimodal iron chelators M30 and HLA-20 series derived from iron chelator VK-28. These multimodal drugs possess neuroprotective (anti apoptotic) and neurorestorative activities for AD, PD and AIS animal models.

These were achieved with incorporation of specific functional moieties that target an array of pathological pathways, each of which is believed to contribute to the cascade that ultimately leads to neuronal cell death. These moieties include iron chel;ating–radical scavenging , cholinesterase and monoamine oxidase inhibitory activities and neuroprotective propargylamine moiety. have the ability to process APP to non amyloigenic neuroprotective-neurotrophic, soluble APP alpha and inductions of GDNF, BDNF and HIF (hypoxia inducing factor),VEGF (vascular endothelial growth factor) and prevention entry into cell cycle arrest via inhibition of Go / G1 and cycline D, resulting neuronal differentiation.

These drugs convert more than 85% of human embryonic stem cells in to neurons and differentiate several other cells into neurons in culture. And in three animal models of PD (MPTP, 6-hydroxydopamine and lactacystin) when given post lesion, they completely restore nigrostriatal dopamine neurons and reverse

plaque formation cognitive deficit in double transgenic model of AD. Multifunctional drugs have advantage over the problem of so called monomodal "Magic Bullets" that act on one neurotoxic and neuropathology modalities of neurodegenerative diseases, where there are cascade of neurotoxic event and pathologies (Youdim and Van Der Schyf, 2009).

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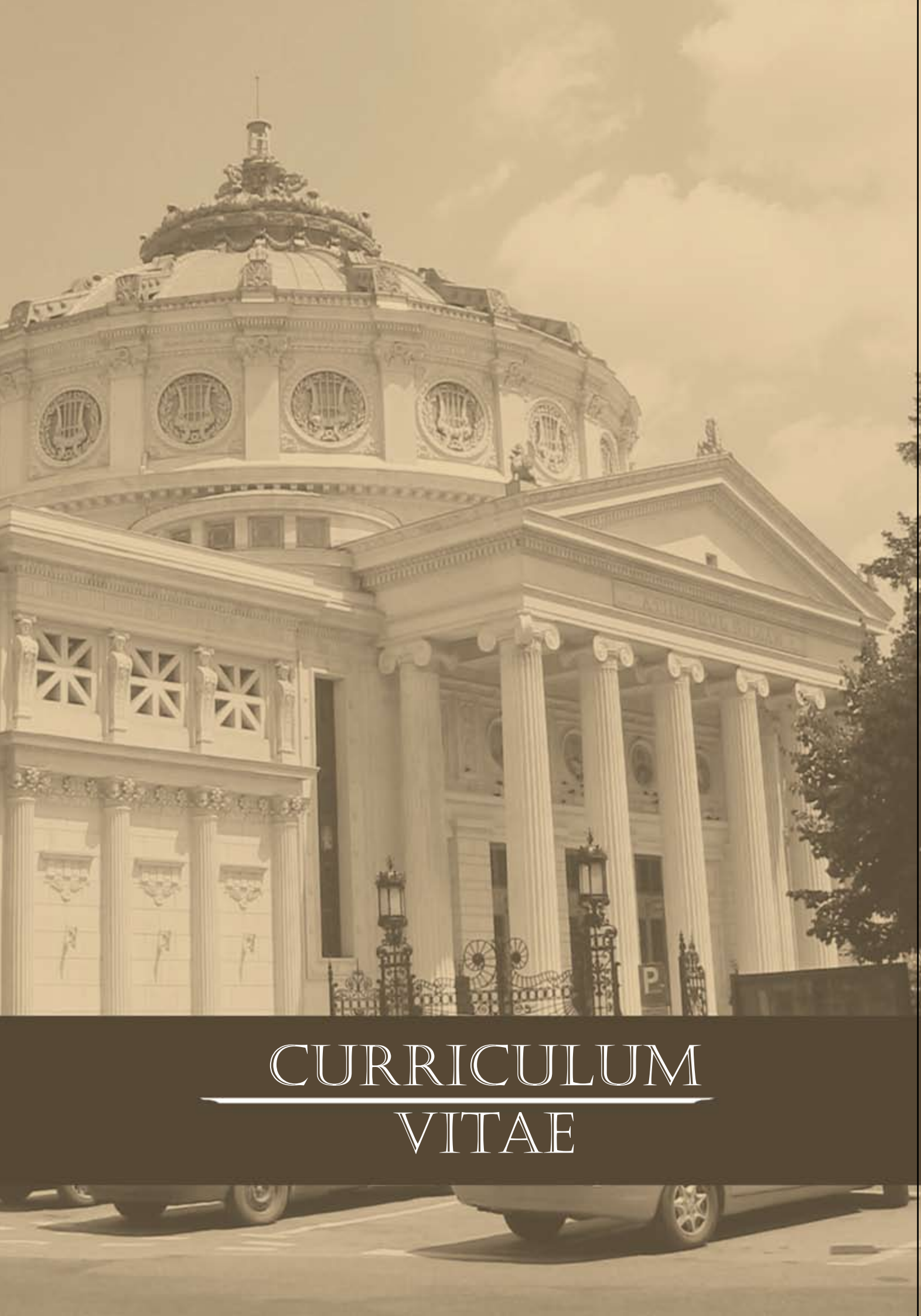

CURRICULUM VITAE

ANTÓN ÁLVAREZ (SPAIN)

Medical Doctor (M.D.), University of Santiago de Compostela (1987)
Diploma of Specialist in Neuroendocrinology, University of Santiago de Compostela (1988)
Graduate in Psychology, University of Santiago de Compostela (1988)
Doctorate in Psychiatry, University of Santiago de Compostela (1988-1990)
Resident Research Fellow of the Ministry of Education and Science (1988-1992)
Department of Psychiatry, Santiago University (1988-1991)
Madrid Complutense University (1992)
Psychiatry Doctor (PhD), Department of Psychiatry, Madrid Complutense University (1997)

Dr. Alvarez has 22 years experience in Basic and Clinical Research on Alzheimer's disease. He was involved in more than 150 research projects, including projects funded by Public Institutions, pharmaceutical R&D studies, industrial and R+D+I projects, epidemiological studies and two projects funded by the European Comunity: (1) MimoVax: Alzheimer's disease treatment targeting truncated AB40/42 by active immunisation (an STREP -Specific Targeted Research Projects- Project approved through the Six Framework Programme of the European Community to develop and test a vaccine for Alzheimer's disease). Period: 2006-2010. (2) BIOMED-PL-950523-European Concerted Action on Pick's Disease. Period: 1995-1998.

As a result of the research activity developed during this period, Dr. Alvarez published more than 120 scientific articles in national and international journals and books. In addition, Dr. Alvarez is actively involved in several scientific forums of his specialty (Congresses, Research Groups, Scientific Journals and Associations).

RAÚL LUCIANO ARIZAGA (ARGENTINA)

Raúl Luciano Arizaga, MD
1970 (School of Medicine, University of Buenos Aires)
Neurologist
1975 (Health Ministry, Argentine College of Clinical Neurologists)
Certified: National Academy of Medicine

• Chief. Cognitive Neurology Unit. Neuraxis Institute. Neurological Foundation. Buenos Aires. Argentina.
• Chairman. Research Group on Dementia. World Federation of Neurology.
• Member. Research Group on Neuroepidemiology. World Federation of Neurology.
• Principal Investigator, Argentina. 10/66 Dementia Research Project. Alzheimer's Disease International.
• Corresponding Fellow. American Academy of Neurology
• Member. Neuroepidemiology Section. American Academy of Neurology.
• Member Behavioural Neurology Section. American Academy of Neurology.
• Member. International Psychogeriatric Association.
• Member. International Federation of Ageing.
• Member International Working Group on Harmonisation of Dementia Drugs Guidelines.
• Member of the Board. Psychogeriatric Commission. Psychiatrists Argentine Association
• Member of the Board. Argentina Stroke Society
• Member of the Editorial Board: Psychogeriatrics
• Professor of Neurology. UCES. (Universidad de Ciencias Socieles y Empresariales). Buenos Aires. Argentina

Published books

• Demencia: enfoque multidisciplinario. Mangone CA, Allegri RF, Arizaga RL, Ollari JA. Editorial Polemos. Buenos Aires, 2005.
• Demencia: enfoque multidisciplinario. Mangone CA, Allegri RF, Arizaga RL, Ollari JA. Ediciones Sagitario. Buenos Aires, 1997.
• Enfermedad de Alzheimer: enfoque actual. Mangone CA, Allegri RF, Arizaga RL y cols. Argentum Editora. Buenos Aires, 1995.

STAVROS J. BALOYANNIS (GREECE)

Studies:
- Medicine, Aristotelian University of Thessaloniki, Greece,
- Theology, Aristoelian University Thessaloniki Greece.
- Neurology, Institute of Neurology, Queen Square, London.
- Catholic University of Louvain, Belgium
- Neuropathology, University of Pennsylvania USA,
- Yale University New Haven USA,
- Neurotopathology Harvard. University, Boston

Specialities:
- Neurology, Psychiatry,
- Neuropathology

Postdoctoral Studies abroad
- Neuropathology and Electron Microscopy. In Institute of Neurology, Queen Square, London
- Neuropathology and experimental neurology Catholic University of Louvain, Belgium
- Neuropathology and Experimental Neurology, University of Pennsylvania, USA
- Neuroimmunology, Yale University, New Haven
- Neurootology and Neurootopathology, Harvard. University, Boston

Academic Position
- Professor of Neurology
- Head of the Department of Neurology,
- Aristotelian University, Thessaloniki, Greece

Visiting Professor
- Tufts University
- Democretian University
- Aristotelian University, School of Theology
- Aristotelian University, School of Philosophy

Reaserch
- Blood Brain Barrier in dementias
- Blood Brain Barrier in Demyelinating diseases
- Mitochondria in Alzheimer's disease
- Synaptogenesis in vivo and in vitro
- Neuronal apoptosis in dementias and demyelinating diseases

Special interest
- Neuroethics, Neurophilosophy
- Participation in Citizen ambassador Programs In 1987 to China

Books
- 28 books on Neurology, Neuropathology, Neuropsychology, Neurology and the Arts, History of Neurology, Neuroethics

Papers
- 700 papers in Greek and International Journals on Neurology, Neuropathology, Neuroimmunology and Neuroethics

Scientific societies
- Member of 60 Greek and International Scientific Societies
- Honorary Member of the Academy of Hellenic Air Forces
- Corresponding Member of the American

Neurological Association

- President of the Society for the amelioration of the quality of life in chronic neurological diseases
- President of the Orthodox Medical Association for support of health and medical education in Asia and Africa.

Honorary Distinctions

- 12 Gold Medals from the Church of Greece, the Hellenic Red Cross and Academic Institutions
- Archon Actuarius of the Ecumenical Patriarchate of Constantinople.

OVIDIU BĂJENARU

(ROMANIA)

He has been presently working as professor at Neurology Department of Universitary Hospital, Bucharest Rumania. He was dean of Medicine Faculty "Carol Davila" (200-2004) and medical vice-director of the Universitary Hospital for ten years since 1993. In present he is coordinator of clinical department of Excellency Centre for Neuroscience from Medicine University "Carol Davila" Bucharest.

Professor Bajenaru has served as expert advisor and evaluator for a number of research councils and institutions in Europe and USA and is currently president of Rumanian Neurology Society, founder member of Rumanian Association for Pain Study, member of American Academy for Advancement in Science and New York Academy of Sciences.

He is also representative of Rumania in World Neurology Federation. Professor Ovidiu Bajenaru has been involved in multiple pivotal clinical trials and his current special research interests are many department of medicine including Parkinson disease, migraine disease, pharmacy-clinical researches in pathology of central nervous system, cerebral ischemia, sleeping disorders.

HEINRICH BINDER

(AUSTRIA)

EDUCATION:

1953 - 1957 Primary School „Piaristen“ in Vienna, Austria

1957 - 1965 Secondary School Bundesgymnasium VIII, Vienna

1965 - 1972 Faculty of Medicine at the University Vienna MD since (promotion on) 1972, June 6th

1972 - 1978 University Hospital for Neurology, graduated in Medical Specialist for Neurology and Psychiatry

9/1982 Docent for neurology, a title corresponding to PhD since 1988 Professor for Neurology, University Vienna founding member of the Austrian Society for Neurorehabilitation

5/1989 Head of the Neurological Hospital “Maria Theresien-Schlössel”)

1994-2007 Head of Ludwig Boltzmann Insitute for Restorative Neurology and Neuromodulation

2010 Organization of 6th WCNR in Vienna (Congress president)

Since 2008 Deputy Head of Landsteiner Institute for Neurorehabilitation and Space Medicine

Since 2002 Head of the Neurological Center, Otto Wagner Hospital, Vienna. Main focus: Patients with severe neurological/neuropsychological deficits and invasive neurorehabilitation methods

Currently President of Austrian Society for Neurorehabilitation
Member of the Management Committee of WFNR
President of EFNRS

Main topic of research: Neurorehabilitation, brain injury, spinal cord injury, vegetative state/ apallic syndrome (more than 140 publications)

ANGEL CEDAZO MINGUEZ (SWEDEN)

Dr. Angel Cedazo-Minguez received a M.Sc. in Biological Sciences from Salamanca University (Spain). He spent 4 years working in a collaborative project between Pharmaltalia and Karolinska Institutet, to identify modulators of beta-amyloid production. He joined Karolinska Institutet as a Ph.D. student in 1998, accomplishing the degree in Medicine in 2002.

Recently in 2010, he has been appointed as an Associate Professor. Currently, he is Deputy Head of the KI-Alzheimer's disease research center, and Vice-Head of the Department of Neurobiology, Care Sciences and Society at Karolinska Institutet. Dr. Cedazo-Minguez was the recipient of awards from Lundbeck in 2003, the Swedish Brain Foundation in 2003 and 2004, and the Bank of Sweden (Erik Rönnberg's award 2006-2009).

The focus of Dr. Cedazo-Minguez 's research involves investigating the pathological mechanisms behind some known risk factors for Alzheimer's disease. Current projects are directed towards 1) fundamental alterations in antioxidant systems; 2) homeostatic dysregulation of cholesterol metabolism; and 3) signal transduction alterations.

ALEXANDRU-VLAD CIUREA (ROMANIA)

Education

1958-1964
• Medical School (IMPh - Institute of Medicine and Pharmacy) of Bucharest,

1972
• Specialist in neurosurgery, Neurosurgical Clinic, Hospital "Dr. Gh. Marinescu",

1976
• "Doctor in Medical Sciences" (Ph.D.), Neurosurgical speciality

1979
• Senior Neurosurgeon, M.D., in Neurosurgical Clinic Hospital "Dr. Gh. Marinesco"

1989
• Chairman of 1st Neurosurgical Department, Neurosurgical Clinic, "Prof. Dr. D. Bagdasar" Hospital, Bucharest.

1993
• Associate Professor UMPh (University of Medicine and Pharmacy), "Carol Davila" Bucureşti,
• Neurosurgical Clinic 1, "Prof. Dr. D. Bagdasar" Hospital, Bucharest.

1997-2005
• Director of "Bagdasar-Arseni" Clinical Hospital, Bucharest

1997-present
• Professor of Neurosurgery UMPh (University of Medicine and Pharmacy), "Carol Davila" Bucureşti, Head & Chairman Neurosurgical Clinic 1, Hosp. "Prof. Dr. D. Bagdasar - Arseni" Hospital, Bucharest.

Professional Affiliations

1980
• Member I.S.P.N. (International Society of Pediatric Neurosurgery)

1982
• Member E.S.P.N. (European Society of Pediatric Neurosurgery)

1994
• New York, New York Academy of Sciencies. Member I.D. 383884 - 4.

1995
• Member International Society for Pediatric Skull Base.

1997
• Munster, EMN (Euroacademia Multidiciplinaria Neurotraumatologica)

1998
• Marsilia, Member SNCLF (French Society of Neurosurgery).

1999
• Neurorehabilitation Committee of the WFNS Memmber CNS (Congress of Neurological Surgeons)
• President of the Romanian Society of Neurosurgery (RSN)

2001
• General Secretary of the EMN (Euroacademia Multidisciplinaria Neurotraumatologica)
• International Honorary Member of Executive Committee of ACNS (Asian Congress of Neurological Surgeons)

2002
• CNS (Congress of Neurological Surgeons) Ambassador.

2003
• Secretary, Founder Member AMN (Academia Multidisciplinaria Neurotraumatologica)

2005
• Vice-President WFNS "at large" (World Federation of Neurosurgical Societies).
• President, Society of Neuro-Oncology from Romania (SNOR).

2007
• Corespondent Member of Romanian Scientists Academy

2008
• Vice-President AMN (Academia Multidisciplinaria Neurotraumatologica)
• Doctor Honoris Causa Medical University "N. Testimitianu", Chisinau, Republic of Moldavia
• Honorary President of the Romanian Society of Neurosurgery (RSN)

2009
• Member of Romanian Academy of Medical Science
• Vicepresident of EMN (Euroacademia Multidiciplinaria Neurotraumatologica)

2010
• Chairman of WFNS Nominating Committee

ANTONIO FEDERICO

(ITALY)

Prof. Antonio Federico, born in Polla (Sa) on the 25.08.48, from 1990 is full professor of Neurology at the University of Siena , Director of the Unit Neurometabolic Disease and of the Research Center for diagnosis, therapy and prevention of Neurohandicap and Rare Neurological Diseases.

He was Director of the Department of Neurological and Behavioural Sciences, University of Siena (2002-2008).

He received the degree in Medicine and specialization in Nervous and Mental Diseases, summa cum laude, at the University of Naples in 1972 and 1975.

His biological training was in the Centre de Neurochimie of CNRS, in Strasbourg, directed by prof. Mandel where he worked in the years 1973-75. He after collaborated with many international research groups, in different countries where he spent in the past years some times: in Montreal (Prof. Andermann, Karpati and Shoudgbridge), in London (dr A. Harding and prof. Morgan-Hughes), in Toronto (dr.Robinson), in Bonn (prof. von Bergmann) , in Paris (dr.Baumann), in Baltimore (proff. Moser and Naidu), in Oxford (prof. Matthews), etc.

His clinical formation was made at the Medical School of the University of Naples, in the Dept, Neurology, and after in Siena, where he moved on 1980. Associated professor in Neurology in 1982, since 1990 he is full professor of Neurology, Medical School, University of Siena.

His present positions are:

- Full professor of Neurology, University of Siena, Medical School
- Director of the Section Neurological Diseases of the Department of Neurological and Behavioural Sciences of the University of Siena,
- Chairman of the Panel of Neurometabolic Diseases of the European Federation of Neurological Societies, where he was also member of the Scientific Committee.
- Italian Delegate to the World Federation of Neurology, and member of the by-low and Constitution Committee and of the Nominating Committee of WFN
- President of the Italian Society of Neurology
- He is associated editor of the Neurological Sciences, Springer-Verlag, Editor-in chief of the Italian Edition of Continuum ; he is also in the editorial board of several other italian and foreign neurological journals
- From 1995 he is Director of a PhD Programme on Applied Neurological Sciences at University of Siena, from 2004 of the European PhD Programme and European School of Doctorate of Applied Neurological Sciences.
- He is Italian member of European Union of Medical Specialists, in the section Neurology.

In the years 1990-96 he was Secretary of the Italian Society of Neurology.

He coordinated the Study Group on Clinical Neurogenetics of the Italian Society of Neurology.

He has been referee for projects evaluation in the area of Orphan drugs and Orphan diseases

for Biomed Projects from EU, for MURST, CNR and Istituto Superiore di Sanità, and other national and international funding agencies, etc. He is member of the Second Opinion Group of the American Leucodistrophy Association.

He is author of more than 500 article (more than 300 of them quoted by Pubmed). He is author of a chapter on Cerebrotendinous Xanthomatosis, Vinken and Bruyn Edts, Handbook of Clincal Neurology, vol 49, Neurodystrophies and Neurolipidoses. On the book McKusick's Mendelian Inheritance in Man,. Ed.1992, Catalog of Autosomal Dominant and Recessive Phenotypes he is cited for 3 different diseases. He was editor of the book Late Onset Neurometabolic diseases (A.Federico, K. Suzuki and N.Baumann Edts), Karger 1991, and many others book from Italian and international Publishing Companies.

His main field of interest is related to neurometabolic, neurodegenerative and rare diseases, investigated from a genetic, metabolic, neuroimaging and clinical point of vue.

FRANZ GERSTENBRAND (AUSTRIA)

Born September 6 .9.1924 in Hof, North Moravia
Primary School in Untertannowitz, South Moravia
High school in Nikolsburg, South Moravia, graduation 1942
Military service (air force) July 1942 to May 1945
Study of Medicine at the University of Vienna April 1946 to July 1950

Graduation M.D. July 14th1950
Resident physician in Neurology and Psychiatry 1st October 1950 to 31st March 1959, senior physician from 1959 onwards at the Psychiatric-Neurological, University Clinic of Vienna

Associate Professor of Neurology and Psychiatry (venia legend) July 10th 1967
Professor of Neurology April 25th 1973
Head of the 2nd Department of the Neurological Hospital, Rosenhügel, Vienna, 1st March 1975 to 31st January 1976

Head of the Neurological University Clinic of Innsbruck, February 1st 1976
September 30th 1994, emeritus from the University of Innsbruck

Co-Director of the Luwig Boltzmann Institute for Restorative Neurology and Neuromodulation, Vienna, since January 1st 1995

Honorary doctorate of the Charles University, Prague, June 4th 1997
Honorary Senator of the Danube University Krems (Austria), 16th November 2001
Honorary doctorate of Aristotle University of Thessaloniki (Greece), 27th June 2003

Professional affiliations:
• Founding member and Past President of the European Federation of Neurological Societies (EFNS)
• Founding member and Honorary President of the Danube Symposia for Neurosciences and Education (Central and East European Neurological Association)
• Founding member and member of the Executive Board of the European Society of Neuropharmacology (ESCNP)

• Chairman of the Research Group on Neuroethics of the EFNS
• Chairman of the EFNS – SIG on Ethics in Neurology
• Chairman of the WFN Research Group on Space and Underwater Neurology

• Member of the Executive Board of the World Federation for Neurological Rehabilitation
• Austrian Society for Tropical Medicine and Parasitology
• International Neuropsychiatric-Pula-Symposia
• WFN Research Group for Extrapyramidal Diseases
• Austrian Society of Coma Vigile (Wachkoma)
• German Society of Coma Vigile (Wachkoma)

• Member of the WFN Research Group for Intensive Care Neurology

• WFN Research Group for Neurological Services
• WFN Research Group on Neuro-Imaging
• Myanmar Neurological Society
• American Academy of Neurology
• Member of the Royal Society of Medicine, London

• Honorary President of the Austrian Parkinson Society
• Honorary President of the Austrian Society of Neurology and Psychiatry
• Honorary President of the Austrian Advocacy Group for Apallic patients
• President of the Austrian-Myanmar Society

• Honorary member of the European Federation of Neurological Societies (EFNS)
• All Russian Scientific Society of Neurologists
• Polish Medical Society
• Neurological Society of the Czech Republic
• Medical Society J.E. Purkynje
• Hungarian Neurological Society

• Candidate for President of the World Federation of Neurology (WFN), 1997

Main research activities in:
Neurotraumatology (spinal cord injury, brain injury), Apallic Syndrome/Vegetative State, Development and Paediatric Neurology, Neurology of the Brain Stem and the Limbic System, Neurology of the Extrapyramidal System (Parkinson's Disease, Huntington's Disease etc.) Biochemistry of Neurotransmitter and Neuropeptides, Neurodegenerative Diseases (olivo-ponto-cerebellar-atrophy, Myotonia etc.), Space and Underwater Neurology, Hyperbaric Oxygenation Therapy, Tropical Neurology (parasitic diseases, food intoxications, malnutrition, snake-bite injury, infection diseases) Neuroaids, Neuroorthopedia (vertebral spine dysfunction), Neuroimaging (magnetic resonance tomography, functional MRI), Cerebrovascular Diseases, Dementia, Neurorehabilitation, Restorative Neurology and Neuromodulation, Neuroethics.

Publications
• Author of 721 scientific papers
• Author of a monograph on the traumatic Apallic Syndrome
• Editor of 12 books on different neurological topics

Organizer and President of various congresses, symposia, workshops, teaching courses.

Lecturer in Clinical Neurology and in special neurological topics.

JAKUB HORT (CZECH REPUBLIC)

Education: Undergraduate: 2nd Medical School, Charles University (1988-1994), Postgraduate: 1st degree specialisation in Neurology (April 1997), Postdoctoral studies in Biomedicine – Neurosciences. (1994 - 1998), State examination in Neurosciences (March 31, 1998), 2nd degree specialisation in Neurology (May 16, 2000)

PhD thesis: Cognitive Memory Impairment and Models of Epilepsy in Rats (Charles University, 2001)

Employment: Medical appointments: Resident at Department of Neurology, 2nd Medical School, Charles University (1994 – 1999), Head of Outpatient Department, Dept. of Neurology, 2nd Medical School, Charles University (since 2000-2006), Head of Memory clinic, Dept. of Neurology, 2nd Medical School, Charles University (since 2001), Chairman of the Section on Cognitive Neurology, Czech Neurological Society (since 2004, re-elected 2009) Teaching appointments: Assistant professor at Dept. of Physiology, 2nd Medical School, Charles University (part time job since 1998), Assistant professor at Department of Neurology, 2nd Medical School, Charles University (2002-2008), Associated professor in Neurology (January 17, 2008)

Publications, lectures, and presentations: over 20 full-length papers in impacted journals, up to date over 90 citations, over fifty posters and presentations to foreign and Czech conferences.

Current research activities: biomarkers of Alzheimer´s disease, spatial memory, Mild cognitive impairment

International Memberships: vice- chair of the European Federation of Neurological Societies (EFNS) scientific panel on dementia, in charge of EFNS task force on cerebrospinal fluid biomarkers., leading the EFNS guideline process on Alzheimer disease. Member of the EFNS task force on Neuropsychology and a member of a team preparing EFNS guidelines on Non-AD dementias. Member of organizing committee of ADPD 2009 conference, Scientific advisory board for ADPD 2011,
Clinical Fellowship and courses:

- Nevill Hall Hospital, Great Britain, internal medicine (1994)
- Salzburg Cornell Seminars in Neurology (Austria, 1996)
- Memory clinic at Queen Square Institute of Neurology, London, (2001)
- Evidence Based Medicine in Dementia (Skodsborg, Danmark, 2002)
- Memory disorders unit, Massachusetts General Hospital, Harvard Medical School, Boston, USA (2003)
- University of South Florida, Tampa, USA (december 2007).

Important publications

Hort J., O´Brien J., Gainotti G., Pirtila T., Popescu B., Rektorova I., Sorbi S., Scheltens P. EFNS guidelines for the diagnosis and management of Alzheimer's disease - to be published by the end of 2009 in EUR J NEUROLOGY

HORT J., BARTOS A., PIRTTILA T and SCHELTENS P. se of cerebrospinal fluid biomarkers in diagnosis of dementia across Europe, EUR J NEUROLOGY, Aug 2009 – in press

LACZÓ J, VLCEK K, VYHNALEK M, VAJNEROVA O, ORT M, HOLMEROVA I, TOLAR M, ANDEL R, BOJAR M, HORT J. Spatial navigation testing discriminates two types of amnestic mild cognitive impairment. BEHAV BRAIN RES 2009 Sep 14;202(2):252-9.

HORT, Jakub, LACZO, Jan, VYHNÁLEK, Martin et. al. Spatial navigation deficit in amnestic Mild cognitive impairment. PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA 104, MAR 6 2007, No.10, p. 4042-4047

SAKKA, Paraskevi, TSOLAKI, Magda, HORT, Jakub et al. Effectiveness of donepezil in patients with Alzheimer's disease discontinuing memantine monotherapy. CURRENT MEDICAL RESEARCH AND OPINION, DEC 2007; 23(12):3153-3165

HOŘÍNEK, Daniel, PETROVICKÝ, Pavel, HORT, Jakub et. al. Amygdalar volume and psychiatric symptoms in Alzheimer´s disease: an MRI analysis. ACTA NEUROLOGICA SCANDINAVICA 113, 2006, No.1., 40-45

HOŘÍNEK, Daniel, VARJASSYOVÁ, Alexandra and HORT, Jakub. Magnetic Resonance analysis of Amygdalar volume in Alzheimer's Disease. CURRENT OPINION IN PSYCHIATRY, 20, 2007, No. 3, 273-277

Rektorova I., Rusina R., Hort J., Matěj R. The degenerative dementias. In: Robert P Lisak, Daniel D Truong, William M Carroll and Roongroj Bhidayasiri. International Neurology, Clinical approach, p. 126- 150, Wiley Blackwell, 2009

VOLKER HÖMBERG

(GERMANY)

Medical Director:
• St. Mauritius Therapy Hospital Meerbusch

Personal Data
• Born 25 July 1954
• Married to Priv.-Doz. Dr. Kristina Müller, paediatric neurologist

Medical Career
1973 - 1980
• Medical School, Universities of Düsseldorf and Freiburg;
• Elective in Neurology at Boston City Hospital, Boston, Mass.;
• National Hospital for Nervous Diseases, London

since 1975
• Junior researcher in the Department of Neuropsychology at the C. & O. Vogt Institute for Brain Research, Düsseldorf and the Department of Neurology, Freiburg (Prof. R. Jung)

1980 - 1981
• Research fellow in the Department of Neuropsychology (Prof. G. Grünewald) at the C. & O. Vogt Institute for Brain Research, Düsseldorf

since 1981
• Clinical training in the Department of Neurology (Prof. H.-J. Freund), Heinrich-Heine-University Düsseldorf

since 1985
• Senior registrar in the Department of Neurology, Heinrich-Heine-University Düsseldorf

since 1987
• Senior investigator for the German Research Council Special Task Force in Neurology at Heinrich-Heine-University (SFB 200 and SFB 194)

1987-2005
• Medical director of the Neurological Therapy Center (NTC), Heinrich-Heine-University Düsseldorf

since 1988
• Board examiner for Neurology at the local examination board (Ärztekammer Nordrhein)

1989-1997
• Vice president of the German Society for Neurological Rehabilitation
1993
• Habilitation in Neurology, Heinrich-Heine-University Düsseldorf

since 1995
• Board examiner for physical medicine and rehabilitation (Ärztekammer Nordrhein)

1997-2005
• Medical director of the Neurological Therapy Center, Cologne

1998-2004
• President of the German Society for Neurological Rehabilitation

since 2000
• Medical director and head of neurology, St. Mauritius Therapy Hospital, Meerbusch

since 2003
• Secretary General World Federation for NeuroRehabilitation (WFNR)

since 10/2004
• Vice president of the German Society for Neurological Rehabilitation

since 2005
• Panel-Chairman Neurorehabilitation for European Federation Neurological Societies (EFNS)

ZSIGMOND TAMAS KINCSES (HUNGARY)

Qualifications MD
University of Szeged, Faculty of General Medicine, Hungary, 2001

Corticostriatal circuitry mediates fast-track visual categorization

PhD
University of Szeged, Faculty of General Medicine, Hungary, 2004

Cortico-striatal circuitry in visual perception
Board certicate in Neurology
May 2010

Educations

1995-2001
• Faculty of General Medicine, University of Szeged, Hungary

2000-(3 semesters)
• Faculty of Programming and Mathematical Sciences, Univ. of Szeged, Hungary

2001-2003
• Ph.D in Experimental and Clinical Neuroscience, Univ. of Szeged, Hungary

Experience
• Dept. of Physiology, University of Szeged, Hungary.

1997-2001
• Student Research Assistant Dept. of Physiology, University of Szeged, Hungary

1999-2001
• Teaching Assistant
Dept. of Clinical Neurophysiology, University of Gottingen, Germany

2001-2003
• Research Fellow
Dept. of Neurology, University of Szeged, Hungary

2003-2005
• Resident of Neurology
FMRIB,University of Oxford, UK

2005-2007
• Post Doctoral Research Fellow
Dept. of Neurology, University of Szeged, Hungary

2007-2010
• Registrar in Neurology
Dept. of Neurology, University of Szeged, Hungary

2008
• Head of Neuroimaging Research Group
Dept. of Neurology, University of Szeged, Hungary

2010
• Consultant Neurologist

AMOS KORCZYN (ISRAEL)

Professor Amos D. Korczyn is the Sieratzki Professor of Neurology at Tel-Aviv University. Professor Korczyn graduated from the Hebrew University – Hadassah Medical School in Jerusalem in 1966 (MD), where he also received an MSc degree in pharmacology (cum laude) in 1966. He trained in neurology at Beilinson Hospital and at the National Hospital for Nervous Diseases in Queen Square, London. He was the Chairman of the Department of Neurology at the Tel-Aviv Medical Center since 1981 until 2002. Professor Korczyn has a particular interest in dementia. He has authored or co-authored over 600 articles in peer-reviewed journals, as well as chapters in books, etc. Professor Korczyn is or has been an Editorial Board member of 15 international journals, and organized several neurological conferences, mainly in the field of dementia, Parkinson's disease and other degenerative brain disorders.

STEVEN LAUREYS

(BELGIUM)

Present post
• Senior Research Associate (tenure) at the Belgian National Fund for Scientific Research (FNRS),
• Head of the Coma Science Group, Cyclotron Research Center
• Clinical Professor of Neurology (tenure), University Hospital, University of Liège

Percent of time spent in: teaching (20%), research (50%), clinical (30%), private (0%)

University qualifications
• 1993: Medical Doctor, Vrije Universiteit Brussel, Brussels, Belgium : magna laude
• 1997: Master in Science (Medical and Pharmaceutical Research, Specialization in Pharmaceutical Medicine), Vrije Universiteit Brussel : magna laude
• 1998:Qualified in Neurology, Ministry of Health, Belgium
• 2000: Ph.D. in Biomedical Sciences, University of Liège, Liège, Belgium: summa cum laude
• 2004: Qualified in Palliative Care (Université Libre de Bruxelles, Université de Liège, Université Catholique de Louvain) : magna laude
• 2007: Agrégation de l'enseignement supérieur (ULg)
• 2008: Clinical Professor of Neurology (ULg)

Current h-index 37 Cumulated impact factor >1300 (first or last author: >900)

Representative publications
• Detecting awareness in the vegetative state Owen AM, Coleman MR, Boly M, Davis MH, Laureys S, Pickard J Science 313 (2006) 1402
• Death, unconsciousness and the brain Laureys S Nature Reviews Neuroscience, 11 (2005) 899-909
• Willful modulation of brain activity in disorders of consciousness Monti MM & Vanhaudenhuyse A, Coleman MR, Boly M, Pickard JD, Tshibanda JF, Owen AM, Laureys S New England Journal of Medicine 362 (2010) 579-89
• Restoration of thalamocortical connectivity after recovery from persistent vegetative state Laureys S, Faymonville ME, Luxen A, Lamy M, Franck G, Maquet P Lancet 355 (2000) 1790-1791
• Brain function in coma, vegetative state, and related disorders Laureys S, Owen A, Schiff N Lancet Neurology 3 (2004) 537–46

Prizes multiple awards including ASSC William James Prize for Contributions to the Study of Consciousness, USA; Tom Slick Research Award in Consciousness, Mind Science Foundation, TX, USA; InBev-Baillet Latour Fund Clinical Research Prize; Society of Cognitive Neuroscience Young Investigator Award, NY, USA

Funding 4 European Grants; 2 US grants; multiple national grants

Fellowships and Scientific Memberships
• Chair of the European Neurological Society •Subcommittee on disorders of consciousness (2008-)
• International Brain Injury Association, Board of Directors (2010-)
• Association for the Scientific Study of Con-

sciousness – Board of Directors (2009-)
• Invited Professor College Belgique (2008-)
• Member of the American Academy of Neurology Committee for the Development of Practice Guidelines for the Vegetative and Minimally Conscious State (2007-)
• Member of the European Federation of Neurological Societies Task Force for the Development of Management Guidelines for the Vegetative State (2007-)
• Research Collaborator, Oxford Centre for Neuroethics (2008-)
• Honorary International Fellow at the Institute of Complex Neuro-disability, Royal Hospital of Neuro-disability, London, UK (2004-)

DAFIN F. MURESANU

(ROMANIA)

Affiliation
• "Iuliu Hatieganu" University of Medicine and Pharmacy, Faculty of Medicine, Victor Babes Str.,Nr.8, 4012, Cluj-Napoca, Romania

Occupation or position held
• Professor of Neurology
• Vice-Dean, Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu", Cluj-Napoca, Romania
• Member of the Romanian Academy of Medical Sciences
• President of the Society for the Study of Neuroprotection and Neuroplasticity

Title of qualification awarded
• Specialist in Neurology
• Senior Neurologist
• PhD
• MBA

Awards in the last 2 years
• Romanian Natoinal Television Channel 2 - Best Medical TV Program
• Viata Medicala - (Romanian Medical Journal) 2009 award of excellence for the whole medical activity
• Romanian Academy of Science (2009 Gheorghe Marinescu award) for the contribution to the development of concepts in neuroprotection and neuroplasticity

Main activities and responsibilities
• Head of the Department of Neurology CFR University Hospital (medical, scientific, and administrative responsibilities)
• Chairman of University Department (teaching courses for undergraduate students, post graduate students and PhD students)

Papers published in international journals (indexed in ISI and Pubmed)
• 30 articles

Papers published in other journals, (indexed in other databases)
• 44 articles

Papers published in Romanian journals
• 46 articles

Monographies
• 7 monographies

Chapters in published books
• 5 chapters

Fluent in
• English, Italian

Main affiliations
• World Academy for Multidisciplinary Neurotraumatology (WAMN)- Chairman scientific committee
• Danube Neurological Society- Executive Board
• European Society of Clinical Neuropharmacology- Secretary General
• European Federation of Neurological Societies (EFNS)- Neurotrauma Panel

- Global College for Neuroprotection and Neuroregeneration (GCNN)- Vicepresident, Chair man of Clinical Committee
- The Society for the Study of Neuroprotection and Neuroplasticity (SSNN)- President and Founder
- The Romanian Society of Neurology (RSN)- Executive board
- The Romanian National Stroke Association- Board
- The Romanian Society of Clinical Neurophysiology- Board
- European Stroke Organization (ESO)
- Society for Neuroscience (SfN)
- European Neurological Society (ENS)- Member

Journals

- International Journal of Neuroprotection and Neuroregeneration – Editorial Board
- American Journal of Neuroprotection and Neuroregeneration – Editorial Board
- Journal of Cellular and Molecular Medicine – Guest Editor
- Frontiers in Neuroscience – Guest Editor

BOGDAN O. POPESCU

(ROMANIA)

Academic Education and Appointments
1996
MD, 'Carol Davila' University School of Medicine, Bucharest, Romania

1997- 2002
Resident in Neurology, University Hospital Bucharest

2000 -
Assistant Professor, 'Carol Davila' University School of Medicine

2001
PhD, 'Carol Davila' University School of Medicine - suma cum laudae

2002 -
Neurologist, University Hospital Bucharest

2004
PhD, Karolinska Institute, Stockholm, Sweden

2005 -
Head of Laboratory of Molecular Medicine, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania

2008-
Senior Neurologist, University Hospital Bucharest

2009 -
Lecturer, 'Carol Davila' University School of Medicine

2009 -
Senior Researcher, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania

Awards
1999 - Beaufour-Ipsen prize for the best research study in neurology
2000 - Young histochemist award - International Society of Histochemistry and Cytochemistry
2004 - Diploma of scientific merit – 'Victor Babeş' National Institute of Pathology
2007 - Romanian Academy award for medical research
2010 - 'Science and Art National Foundation Award of Excellence for research in the field of Neuroscience and Neuropathology

Other current activities

- Guest editor for Alzheimer's review series at Journal of Cellular and Molecular Medicine
- Executive editor of Romanian Journal of Neurology
- Secretary General of the Romanian Society of Neurology
- Research director of the Society for the Study of Neuroprotection and Neuroplasticity
- Director, Victor Babeş' National Institute of Pathology, Bucharest, Romania
- Spokesman for University Hospital Bucharest

Selected publications

- Popescu BO, Toescu EC, Popescu LM, Bajenaru O, Muresanu DF, Schultzberg M, Bogdanovic N. Blood-brain barrier alterations in ageing and dementia. J Neurol Sci, 283:99-106, 2009.
- Popescu BO. Still debating a cause and diagnostic criteria for Alzheimer's disease. J Cell Mol Med. 2007;11:1225-6.
- Romanitan MO, Popescu BO, Winblad B, Bajenaru OA, Bogdanovic N. Occludin is overex pressed in Alzheimer's disease and vascular dementia. J Cell Mol Med. 2007;11(3):569-79.
- Cowburn RF, Popescu BO, Ankarcrona M, Dehvari N, Cedazo-Minguez A. Presenilin-mediated signal transduction. Physiol Behav. 2007;92:93-7.
- Hansson CA, Popescu BO, Laudon H, Cedazo-Minguez A, Popescu LM, Winblad B, Ankarcrona M. Caspase cleaved presenilin-1 is part of active gamma-secretase complexes. J Neurochem. 2006;97:356-64.
- Popescu BO, Ankarcrona M. Mechanisms of cell death in Alzheimer's disease: role of presenilins. J Alzheimers Dis. 2004;6:123-8.

• Popescu BO, Cedazo-Minguez A, Benedikz E, Nishimura T, Winblad B, Ankarcrona M, Cowburn RF. Gamma-secretase activity of presenilin 1 regulates acetylcholine muscarinic receptor-mediated signal transduction. J Biol Chem. 2004;279:6455-64.

• Cedazo-Mínguez A, Popescu BO, Blanco-Millán JM, Akterin S, Pei JJ, Winblad B, Cowburn RF. Apolipoprotein E and beta-amyloid (1-42) regulation of glycogen synthase kinase-3beta. J Neurochem. 2003;87:1152-64.

• Popescu BO, Oprica M, Sajin M, Stanciu CL, Bajenaru O, Predescu A, Vidulescu C, Popescu LM. Dantrolene protects neurons against kainic acid induced apoptosis in vitro and in vivo. J Cell Mol Med. 2002;6:555-69.

• Popescu BO, Cedazo-Minguez A, Popescu LM, Winblad B, Cowburn RF, Ankarcrona M. Caspase cleavage of exon 9 deleted presenilin-1 is an early event in apoptosis induced by calcium ionophore A 23187 in SH-SY5Y neuroblastoma cells. J Neurosci Res. 2001;66:122-34.

PHILIP SCHELTENS

(THE NETHERLANDS)

Dr Philip Scheltens studied at the VU University Amsterdam, Netherlands, gaining his MD in 1984, and PhD (Magnetic Resonance Imaging in Alzheimer's disease) in 1993. Clinical residencies in neurosurgery at the Municipal Hospital Slotervaart, and at the Vrije Universiteit Medical Centre, Amsterdam, supported his academic development.

Dr Scheltens is Professor of Cognitive Neurology and Director of the Alzheimer Center at the VU University Medical Center in Amsterdam. His main clinical and research interests are Alzheimer's disease, vascular dementia, frontotemporal dementia, magnetic resonance imaging, PET imaging and biomarkers. He is active in the field of biomarkers and clinical trials and has been the national PI for many studies, including fase 1-3 multicenter clinical trials. He founded and directs the Alzheimer Center since 2000, from which over 19 PhD theses have appeared since then.

Dr Scheltens is an active member of several societies including the Dutch Society for Neurology, the International Psychogeriatric Association, the American Academy of Neurology, the Alzheimer Imaging Consortium, the ISTAART consortium and the European College of Neuropsychopharmacology. He has been instrumental in organising several national and international conferences among which the Imaging symposium attached to ICAD. He chairs the dementia panel of the European Federation of Neurological Societies.

He is associate editor of the Journal of Neurology, Neurosurgery and Psychiatry and book review editor of Alzheimer Disease and Associate Disorders. He is chief editor of the official journal of the Dutch Society of Neurology (Tijdschrift voor Neurologie en Neurochirurgie).
He is also member of the editorial boards of Dementia and Geriatric Cognitive Disorders and International Journal of Geriatric Psychiatry, and acts as an ad hoc reviewer of scientific articles for, amongst others, The Lancet (Neurology); Stroke; Neurology, Annals of Neurology, New England Journal of Medicine, Brain and Science.

He has authored more that 410 peer reviewed papers and > 50 book chapters. Dr Scheltens co-edited the book on Magnetic Resonance in Dementia (Springer) and co-edited the book on Functional Magnetic Resonance Imaging: Clinical Applications (Oxford University Press).. He is Treasurer of the International Society for Vascular Behavioural and Cognitive Disorders (Vas-Cog) since 2003.

HARI SHANKER SHARMA (SWEDEN)

Hari Shanker Sharma (Swedish Citizen), Docent in Neuroanatomy (UU); Professor of Neurobiology (MRC), is currently working in Uppsala University Hospital, Department of Surgical Sciences, Division of Anesthesiology & Intensive Care Medicine, Uppsala University, Sweden.

Career History on Research in Neuroscience
Hari Sharma was born on Jan 15, 1955 in an Industrial town Dalmianagar (Bihar), India in a well-reputed family: Father Shri Ram Rup Sharma, M.Eng. (Cal), and one of the founders of Paper Factory under Rohtas Industries Ltd. Hari Sharma did his Higher Secondary Schooling in 1969 from Dalmianagar and enrolled in Bihar University, Muzaffarpur for higher studies. He did his Bachelor of Science with Honors from the prestigious L S College Muzaffarpur in 1973 and secured 1st position in his batch. He obtained his Master Degree from Bihar University with special expertise in Cell Biology in 1976 and awarded Gold Medal of Bihar University for securing 1st potion in the 1st Class. Having a knowledge in cell biology with special interest in the central nervous system, Hari Sharma joined the group of Professor Prasanta Kumar Dey, a neurophysiologist by training in the Department of Physiology, Institute of Medical; Sciences, Banaras Hindu University, Varanasi in 1977 to obtain Doctor of Philosophy Degree (D. Phil) in Neurosciences. In the lab he conducted experiments on morphine dependence and withdrawal in relation to body temperature regulation, behavioral changes and neurochemistry in rat and mice models. In addition he was trained as neurophysiologist to record electrophysiological activity in relation to stress, hyperthermia and drugs of abuse. Hari Sharma was always fascinated by the role of blood-brain barrier (BBB) in various experimental conditions and wanted to know whether brain disease has any relation with the spontaneous disruption of the BBB. His curiosity about the role of the BBB breakdown in stress condition leading to mental diseases was the basis of his Doctoral studies on "Blood-Brain Barrier in Stress" in which he for the first time showed that long or short term stress can disrupt the BBB and disrupts the EEG activity. These changes can be altered by drugs capable to modulate neurochemical metabolism of serotonin, prostaglandins and opioids in the CNS. On this work, he was awarded Ph D in 1982, that was examined and approved by the renowned team of experts on the BBB, namely: the father of Blood-Brain Barrier Research, Stanley I Rapoport of NIH, Bethesda, Maryland, USA; a pioneer on BBB in hypertension Professor Barbro Johansson, Department of Neurology of Lund University, Lund, Sweden; and noted Neuroanatomist with special regard to BBB Erik Westergaard, University of Copenhagen, Copenhagen, Denmark.

Hari Sharma after carrying out a series of Govt. of India funded Research Projects on the BBB and brain dysfunction (1982-1987), joined the lab of Neuropathology at Uppsala University with Professor Yngve Olsson in 1988 to expand his knowledge on the passage of tracer transport across the BBB in stress caused by

traumatic insults to the Brain and Spinal cord at light and electron microscopy. Dr Sharma awarded the prestigious Alexander von Humboldt Foundation Fellowship of German Govt. (1989-1991) to work on hyperthermia induced BBB dysfunction at the ultrastructural level in the laboratory of Professor Jorge Cervós-Navarro (recognized as living "Legends in Neuropathology in Europe", World Federation of Neuropathology in 1990, Kyoto, Japan, and later awarded with the German Govt. highest Civil Award, Bundestag by German Chancellor in 1996). After that Dr Sharma came back to Uppsala to continue his research on Neurotrauma and established a network of collaboration on "Experimental CNS Injury Research Group" with key collaborators in various parts of Europe, USA, and Australia including his parent Institutions in India that is still continuing. The works carried out by Dr Sharma on the pathophysiology of blood-brain barrier in hyperthermia using immunohistochemistry and electron microscopy in the Neuroanatomy Department of Uppsala University (1995-1999). On his work on hyperthermia Dr Sharma was decorated with prestigious Neuroanatomy award "Rönnows Research prize" of Uppsala University for "best neuroanatomical research of the year 1996" followed by the Award of the Degree of Doctor of Medical Sciences of Uppsala University in Neuroanatomy in 1999 (examined and approved by another legend of Blood-brain barrier Research, Professor David Begley, Kings College London, UK). The Uppsala University Thesis of Dr Sharma was also selected for the Best Thesis Award of the Medical faculty, "The Hwassers Prize" of 1999. Subsequent research of Dr Sharma in Uppsala University on the neurobiology of hyperthermia in relation to the Blood Brain barrier and Brain edema (2000-2003) has earned the prestigious title of Docent in Neuroanatomy of Medical Faculty, Uppsala University (approved and recommended by eminent Neuroanatomist, Professor Ole Petter Ottersen, University of Oslo, Norway) in April 2004.

Research Interest & Publications

Currently his main research interest is Neuroprotection and Neuroregeneration, in relation to the Blood-brain barrier in stress, trauma, and drugs of abuse in health and disease. Dr Sharma on his research on brain pathology and neuroprotection in different model received the prestigious award from The Laerdal Foundation of Acute Medicine, Stavanger, Norway, in 2005. His recent research is aimed to find out the role of nanoparticles in neurodegeneration and Neuroprotection using various treatment strategies supported by European Aerospace Research & Development (EOARD), London, UK and US Air Force Research Laboratory, Wright Patterson Air Force Base, Dayton, Oh, USA.

Hari Sharma has published over 180 peer reviewed research papers, 50 reviews, 8 monographs, 55 international book chapters and edited 6 book volumes. He served as Guest Editor of Curr Pharm Desig (2005, 2007); J Neural Transmiss (2006) and is founding Editor-in-Chief of International J Neuroprotec & Neuroregen (2004-), UK., now renamed as American

Journal of Neuroprotection and Neuroregeneration, American Scientific Publications, (2009-) USA and Journal of Nanoneuroscience (2009-). Dr Sharma is on board of various International Journals including Journal of Neurodegeneration and Regeneration, USA; CNS & Neurological Disorders - Drug Targets, Bentham and is associate editor of Journal of Nanoscience & Nanotechnology (Nanoneuroscience 2006-), USA; Frontiers Series on Aging Neuroscience (2009-); Review editor in Frontiers Series on Neuroengineering (2008-) and Neurorestoratology (2010-).

Dr Sharma has served as an expert evaluator and advisor to various Boards, Councils and Institutions for their Research Grants in Europe, USA and in Australasia. Some of the notable organizations include: Australia and New Zeeland Health Council (2000-); University Commission of Grants, Hong Kong (2002-), Singapore Medical Council, Singapore (2003-); UK Charity Organization "Research on Ageing: Help the Aged" (2003-); Selection committee for faculty members of various Universities as external Expert reviewer: Israel, USA, India, UK.

Other Notable Awards & Distinctions

• SIRI Research Prize, 1986, on Hyperthermia Induced Brain Dysfunction, Indian Association of Biomedical Scientists,

• Shakuntala Amir Chand Research Prize 1988, on "Blood-Brain Barrier and Brain Function", Indian Council of Medical research, Govt of India

• Neuronal Plasticity Award 1991; on "Brain Dysfunction in Heat Stress", International neuroplasticity Conference on Brain Dysfunction, Sicily, Italy

• Career Award 1989, University Grants Commission, New Delhi, Govt. of India on Research on Blood-Brain Barrier and Brain Pathology.

• Distinguished Leadership Award, Neuroscience 1990, by Am Bio Res Inst, North Carolina, USA.

• Elected Fellow, Am Inst of Stress, 1996, New York, USA

• Elected Dy. Governor (Board of Governors) of Am Bio Res Inst, North Carolina, USA, 1988 for Europe to help finding and highlighting eminent researchers in the World whose research has really improved the life of Humans or has the potential to change the current scientific thinking for the benefit of mankind.

• Elected Dy. Director General, Int Bio Soc, Cambridge, UK 1997 for Europe to provide input and awareness about the work of scientists whose research can influence the current status of medicine & healthcare.

• Certificate of Excellence, National Centre for Toxicological Research (NCTR), US Food & Drug Administration (FDA), Jefferson, Ar, USA on Works on Psychostimulants and the Blood-Brain Barrier.

• Distinguished International Scientist Collaboration Award (DISCA) by NIH to work in NIDA, Baltimore, USA on Psychostimulants induced neurotoxicity, 2006, 2007.2008.

• Ambassador of the City of Uppsala 2007- Uppsala County Award for promoting Uppsala, Sweden as International Research Collaboration/Meetings and Conference Destination

• Entered into the "Hall of Fame" 2009, Record for Distinguished services in "Neuroscience" influencing Human Health Factors in November 2009, ABRI, NC, USA.
• Awarded Honorary Doctorate, Doctor of science (D.Sc) by Medical University of Cluj-Napoca, October 2009.

Current research projects
• Molecular mechanisms of blood-brain barrier permeability and cell injury in CNS trauma and neuroprotection
• Effect of Nanoparticles on the Blood-Brain Barrier. An Experimental Study in Rats and Mice
• Drugs of Abuse Induced Neurotoxicity. An Experimental Study in Rats using Physiological and Morphological Approaches
• Pathophysiology of CNS damage in heat stress. Exploration of suitable therapeutic measures.
• Brain pathology induced by cardiac arrest. Possible mechanisms of brain damage and repair.
• Novel peptides and neurotrophic factors in neuroprotection and Neuroregeneration against CNS insults
• Therapeutic role of antibodies in CNS injuries

Some Internationally Acclaimed Book Volumes
1. Nyberg F, Sharma HS, Wissenfeld-Hallin Z. Neuropeptides in the Spinal Cord. Prog Brain Res 104, Elsevier Science BV, Amsterdam, North Holland, 1998.
2. Sharma HS, Westman J Editors. "Brain Function in Hot Environment", Prog Brain Res 115, Elsevier Science BV, Amsterdam, North Holland, 1998.
3. Sharma HS, Westman J Editors "Blood-Spinal Cord and Brain Barriers in Health and Disease, Academic Elsevier, Boston, San Diego, USA, 2004.
4. Sharma HS Editor. "Neurobiology of Hyperthermia", Prog Brain Res 162, Elsevier Science BV, Amsterdam, North Holland, 2007.
5. Sharma HS. 2009. Editor, Psychostimulants induced Neurotoxicity, Int Rev Neurobiology 88, Elsevier, Oxford Publ.
6. Sharma HS. 2009. Editor, Nanoneuroscience & Nanoneuropharmacology, Prog Brain Res 180: 1-270, Elsevier, Oxford, Publ.

LUIZA SPIRU

(ROMANIA)

Luiza Spiru, MD, PhD is Professor of Geriatrics and Old Age Psychiatry at "Carol Davila" University of Medicine and Pharmacy in Bucharest, head of the Geriatric and Geronto-Psychiatry University Department in Elias University Emergency Hospital. After her PhD (1997) she acquired multiple specializations (Molecular medicine, Geriatrics Gerontology and Old age psychiatry) and competences in cell biology, neuro-psycho-pharmacology, abdominal and heart ultrasound investigations, IT in medical services, standardized geriatric evaluation, diagnosis and treatment of memory impairment diseases, differential diagnosis of Alzheimer dementia's and other related dementias. She is graduate in health services management (MBA - British Council), and leadership management - personal productivity (Leadership Management International, Waco Texas, USA).

Prof. Luiza Spiru is founder member of national and international professional organizations (Ana Aslan International Foundation, Ana Aslan International Academy of Aging, Romanian Society of Neurosciences, and member of 22 national and international professional associations (The New York Academy of Sciences, Alzheimer Europe society, International Psychogeriatric Association, Alzheimer's Association International Society to Advance Alzheimer Research and Treatment (ISTAART), Alzheimer's Disease International (ADI), among other. Prof. Luiza Spiru is the coordinator of the Romanian Representatives of the European Association for Predictive, Preventive and Personalized Medicine (EPMA) and Millennia 2015 Community. She is also faculty member of The World Society of Anti-Aging Medicine (WOSAAM), European Organization of Scientific Anti-Aging Medicine (ESAAM), European Masters in Aesthetic and Anti-Aging Medicine (EMAA) and European Movement Disorders Society. Beginning with 2001, she is recognized by the European Alzheimer's Disease Consortium as an international expert in cognitive impairment and dementia.

Prof. Spiru is an active principal investigator and international promoter of Ambient Assistive Living movement as one of the most suitable answers to demographic aging, and since 2010 she joined the Working Group of the Assistive Technology Association (UK).

Prof. Luiza Spiru has been awarded with the Honorary Membership for Women in Science Top (Science Academy New York), Women of the Year (The American Biographical Institute, USA, 2007 and 2009), Order of International Ambassador (The American Biographical Institute, USA), World Lifetime Achievement Award (The American Biographical Institute, USA), The Decree of Excellence - International Biographical Centre, Cambridge, UK, Hall of Fame for Distinguished Accomplishments (The American Biographical Institute, USA), The Director General's Leadership Award (International Biographical Centre, Cambridge, UK), Royal Retreat for Special Scientific Activity, Bangkok, Thailand, Excellence diploma for creating a successful Romanian brand - Ana Aslan International Academy of Aging-

granted by "Saptamana Financiara", Romania and the Special Award, Founder and coordinator of Memory Diseases Center ("Carol Davila" Awards, Romania).

As president of "Ana Aslan" International Foundation (from 2000) and vice-president of its research and education department - the Ana Aslan International Academy of Aging (2001) Prof. Luiza Spiru has founded and developed two clinical research and health centres: The Memory Clinic (in 2003) and Ana Aslan International Clinic for Longevity, Healthy Aging and Brain Aging Prevention (in 2008), both in Bucharest. Like in the Ana Aslan International Academy of Aging, within these two clinical centres Prof. Luiza Spiru sustains managerial, educational, editorial and research activities. She coordinated and actually coordinates Aslan Academy partnership in the

EU funded research projects (Tempus Phare ROMGER, VIASAN, EADC, ICTUS, DESCRIPA, K4CARE, SHARE-it, E-ADNI. Recently, Prof. Luiza Spiru has initiated and actually coordinates the implementation at national level of the FP-7 SOP-HRD BRAINAGING Project that aims to train the specialists in hospitals and ambulatories in the particularities of aging and brain aging pathology and the latest technological achievements in the field. She is the main author of two original, multidisciplinary and integrative basic research concepts: NERO_MIND (Critical neuromes in brain aging pathology) and ASGARD (Critical neuromes in organism- environment relationship during brain aging). She also lead clinical trials in epidemiology of aging related diseases, longevity and brain aging medicine, cognitive disorders, memory impairment and dementia, geriatrics and gerontoprophilaxy, geronto-psychiatry, physiotherapy and occupational therapy, social gerontology, health economy and drugs trials. Since 2005 she is advisory board member for pharma companies addressing the neuropathology field.

Prof. Luiza Spiru develops a sustained education activity. Beside her didactical activity within the Geriatric and Geronto-Psychiatry chair at "Carol Davila" University of Medicine and Pharmacy in Bucharest, since 2000 year she is the organizer and active contributor of the annual, CME credited International Training in Memory Diseases. Beginning with 2010 Prof. Spiru has initiated the CME credited annual conference Brain Aging – Hot Topics, whose first edition will be realized in October, in Bucharest. She is also activating as organizing committee chair in 6 renowned international events and 6 international events on continuing medical education.

Prof. Luiza Spiru is the author of 3 treatises on Geriatric medicine, 6 guides on the diagnosis and treatment of Alzheimer's disease and related dementias and 11 book chapters. She has published 85 studies in international journals and books of proceedings. Within scientific events she has sustained 113 oral communications, most of them as invited speaker, and over 150 posters.

PIETER E. VOS

(THE NETHERLANDS)

Pieter Vos is neurologist at the Institute of Neurology at Radboud University Nijmegen Medical Centre, The Netherlands. His research activities are connected with traumatic brain injury, traumatic spinal cord injury and other acute neurological disorders.

Focus of the research activities consist of studies aiming to unravel the clinical, biochemical and genetic determinants of neuroplasticity and recovery after mild, moderate and severe traumatic brain injury.

Pieter Vos is founder of the Dutch working group on Neurortraumatology. Current international activities are chairman of the scientist panel on neurotraumatology and head of the task force mild traumatic brain injury, both residing under the European Federation of Neurological Societies.

KLAUS VON WILD

(GERMANY)

Present Appointment
Professor (apl) for Neurosurgery Medical Faculty Westphalia Wilhelms- University of Münster,

Professor (apl) for Neurorehabilitation and Re-engineering of Brain and Spinal Cord Lesions, International Neuroscience Institute, INI, Hannover, Institute at Otto-von-Guericke University, Medical Faculty, Magdeburg, Germany
Visiting professor Armed Force and Rheumatic Rehabilitation Hospital EL AGOUZA Military Hospital Centre, Cairo, Egypt; China Rehabilitation Research Centre, CRRC, Beijing, PRCh

Medical Education - Qualifications:

• 1966 Graduation from the Medical Faculty of the J.W.Goethe-University Frankfurt/ Main
• 1968 M.D.
• 1975 Specialist Neurosurgeon, Department of Neurosurgery, Head Prof. Hugo Ruf
• 1977 Postdoctoral lecture qualification (Habilitation), Dr.med. habil., in Neurosurgery
• 1977- 1984 Assistant Professor Med. Faculties of the Universities of Frankfurt and Hanover
Consultant Neurosurgical Department Academic Public Hospital Nordstadt,
Director Prof Madjid Samii, Hanover
• 1982- 2002 Director Neurosurgical Clinic Clemens Academic Hospital, Med. Faculty Muenster
• 1984 Professor Medical Faculty University Münster, North Rhine Westphalia, Germany
• 1993- 2002 Founder & Head Special Department for Early Neurorehabilitation in Neurosurgery,

Licence for education and board examination for neurosurgeons of the medical
association in Neurosurgical intensive care, Clinical laboratory medicine in neuro
surgery, Neuroradiology, Electroencephalography, Treatment of Pain,

Physical Training

Dr von Wild has personally performed more than 5000 major operations of CNS and PNS lesions with special interest in pituitary adenomas & tumours of the sella region & the cavernous sinus , CPA tumours , tumours of the spinal cord, brain stem cavernomas; Intramedullary tumours of the spinal cord; all kind of spinal surgery. Birth traumatic spinal cord and brachial plexus lesions;transdisciplinary neurotraumatology and functional reconstruction in cooperation with Reconstruc tive trauma-, Ear- nose and through-, Head and Neck, Thoracic-, Maxilla facial, and Eye surgeons.

At present:

Functional restoration of locomotion in paraplegics by FES implanted neuroprosthesis and via central nervous system- peripheral nervous system (CNS_PNS) by pass grafting procedure following SCI; Neuromodulation of patients in coma and VS State

Clinical Research:

• Organizer & President of numerous national and international congresses, workshops and courses
• Guidelines On Quality management in neurotraumatology, functional neurorehabilitation, and outcome:
• The German Coma Remission Scale (CRS) In Schmidek, HH (ed) 2000: Operative neurosurgical techniques, 4th edition, Vol. 1, Saunders Comp, Philadelphia, US, pp 45-60
• Guidelines on Early Neurological-Neurosurgical Rehabilitation
See Acta Neurochir Suppl. 79, 11-19, 2001
• Guidelines on Management of Poly -traumatised Patients .
See The German Interdisciplinary Association for Intensive Care Medicine (DIVI) 1998, only in German
• Guidelines on Mild Traumatic Brain Injury,
European J. Neurology, 2002, No 9,207-219)
• Revised Guidelines on MTBI Early Management,
See EFNS MTBI Taskforce in EFNS Hand book of neurology 2006,
• Guidelines on quality management for AS/VS
European Journal of Trauma Emerg Surg. 2007, No3:268-292
• The QOLIBRI : Quality of Life after traumatic brain injury assessment tool
See von Steinbüchel N et al 2005 in Acta Neurochirurgica Supp.93, pp 43-49
• Preset Quality management of multidisciplinary neurotraumatology and brain protection
• Quality management and amelioration of patients in long-lasting coma and AS/VS
• Neuromodulation in paraplegics after SCI; Exernal audit for cell-transplantation
• Neuroethics; Long term outcome, HRQoL, and social re-entry following TBI

Distinction

Professor honoris causa (h..c.) for Neurorehabilitation and Reconstructive
Neurosurgery Faculty of Physical Rehabilitation at Al Azhar University, Cairo, Egypt
Doctor honoris causa (Dr.h.c.) at the Faculty of Medicine and Pharmacology, „Iuliu Hatiegau" University, Cluj- Napoca, Romania Honorary (& founding) President EMN, Euroacademy, and AMN, World Academy of Multidisciplinary neurotraumatology;;Honorary President Romanian Society of Neurorehabilitation RoSNeRa ; Corresponding Fellow The Cuban Society of Neurophysiology (SCNFC); Honorary Chairman WFNS Committee Neurorehabil. & Reconstructive Neurosurgery; Honorary Chairman EFNS Panel Neurotraumatology
Honorary Member (former President)German Soc. Neurotraumatology & Neurorehab.
Honorary Member of the Austrian Society , the Lithunian Society, the Polish, the Romanian Society of Neurosurgery, the Russian Federation of Neurosurgical Societies; The Cuban Neurological Society, Egyptian and Pan Arab Societies for Neurorehabilitation, the Japanese Society for Neural Repair and Neurorehabilitation

Scientific Societies - Present Duties

• Since 2001 WFNR Executive Board , World Federation for Neurorehabilitation,
• Since 2009EBIS 1st Vice-President , European Brain Injury Society,
• IANR Scientific Executive Board, International Association of Neurorestoratology
• Since 2008/9Treasurer (Founding Member) International QOLIBRI Society, CNM, International Society for Clinical Neuromusicology, EFNR Europ. Federation Neurorehabilitation
• Since 2003AMN Secretary General, World Academy of Multidisciplinary Neurotraumatology,
• Director (CEO) kvw neuroscience consulting GmbH Muenster, D
• Founding Member & Member of the Presidium: ISRN International Society of Reconstructive Neurosurgery ;
• MASCIN, Madjid Samii Congress of International Neurosurgeons; ESCRI Europ.
• Spinal Cord Research Institute Giorgio Brunelli Foundation, Brescia, Italy;

Founding Member . DANC/GANS German Academy of Neurosurgery; BDNC, German Social Professional organisation of neurological surgeons.

MOUSSA B.H.YOUDIM

(ISRAEL)

Moussa Youdim was Chairman and Professor of Pharmacology from 1977 to 1994 and for establishing the department and was Finkelstein Professor of Life Sciences at the Technion- Rappaport Family Faculty of Medicine . He is the Director of the Eve Topf and National Parkinson Foundation (USA) Centers of Excellence for Neurodegenerative Diseases at Technion and is now Professor Emeritus.. He holds the position of Honorary Professor and Distinguished Chair Professor at Hong Kong and PolyTechnic Universities in Hong Kong, Distinguished Scientific Professor at Yonsei University in Seoul , South Korea, Honorary Distinguished Professor at Qingdao University and Shanghai University Rujin University as wellas Shanghai University Traditional Chinese Medicine in China.

He is internationally renowned for his research on monoamine oxidases and its identification of two forms in 1965 as part of his Msc. Degree and went on to purify the enzyme in in 1966 and showed that this enzyme contained a covalentlt bound FAD as a cofactor. in drug development for Depressive illness, Parkinson's disease (PD) Alzheimer's disease, and amyotrophic lateral scelrosis and for establishing the importance of monoamine oxidase and brain iron metabolism for brain function and aminergic and cholinergic neurotransmission and neurodegtenration. At Oxford University with his colleagues they discovered the monoamine oxidase B inhibitor, l-deprenyl (selegiline) as an anti Parkinson drug in 1975 .At Technion he discovered and co-developed the anti-Parkinson drug, Rasagiline (Azilect) and hsowed that this drug has neuroprotectiev as well as neurorestoreative activity and which recently has been demonstrated to be the first neuroprotective-disease modifying drug (ADAGIO study) in Parkinson's disease . He developed the multifunctional neuroprotective and neurorestorative anti-Alzheimer drug, Ladostigil (TV 3326, Phase II clinical studies) and M30 series under development for Alzheimer's disease, ALS and Huntington Disease as neurorestorative drugs. He is among the very few academics in neuroscience go from bench to clinic.

He has published more than 800 scientific articles, book chapters and invited reviews and edited 45 books and has been on the Editorial Board of 43 International scientific journals. He has over 100 patents as consequence of his drug development. He has received numerous major prizes, awards and honours from Israel, U.S., England, Germany, China, Hong Kong, Iran, Denmark, Holland and Switzerland, several Honoary Doctorate of Philosophy, Honorary Causa, from universities of Semmelweis University (Hungary) and Pisa (Italy).).

From 1991-1999, he was the prestigious Fogarty International Scholar-in-Residence at the Fogarty International Center for Advanced Study in the Human Health Sciences program of the National Institute of Health in Bethesda (USA). Recently he was elected to Leopoldina German Academy of Sciences and received the The Eruoean College of Neuropsychopharmacology LifeTime Achievement AwardProf.

Youdim like Dr Paul Jenssen has been a member of CINP since 1974 and is also a memebr of American College Neuropsychopharamcology. He has had a very strong ties with CINP since joining this organization and has always supported the CINP through his attendance, as well as organizing numerous symposia. and being an invited speaker.

His supop0rt for CINP stems from the fact CINP is the most important neuropharamcology organization that in compasses all the other similar organization and brings together the clinicians and the basic scientists.


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