

Neurochemistry News

The Newsletter of the
Community of Neurochemical Societies

International Society for Neurochemistry



American Society for Neurochemistry



European Society for Neurochemistry



Asian Pacific Society for Neurochemistry



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I S N

Editorial Comment

Next year it is again time to make important decisions regarding replacements in Council as well as the Secretary of ISN. ISN can only function and develop if dedicated and enthusiastic neurochemists involve themselves directly in our Society.

The Council Members are engaged in the different Committees of the ISN and as such play important roles in the decision making process e.g. regarding funding of conferences and educational activities. We therefore need Council Members having a solid scientific background. Unfortunately such scientists are extremely busy and therefore often reluctant to engage themselves in this kind of activity. It must be remembered, however, that if one wants excellence in the services of the ISN excellent people must join and carry out the different functions.

At the next ISN Meeting in Hong Kong your current Secretary will retire from this office to, if elected, serve as the next President. My four years as Secretary have been extremely rewarding and interesting and it can be recommended to try to get into this office. However, at the same time it is a rather demanding job in terms of time. Nevertheless, it is mandatory for the future of ISN that a devoted scientist seeks to be nominated and gets elected into this office. Considering the tradition that the Treasurer and Secretary do not reside in the same geographical region it may be desirable if the incoming Secretary is not from the Americas. Either Europe or the Asian/Pacific region would be preferable.

Another important issue is to choose the location of the ISN/ASN Meeting in 2007 which according to the rotation schedule among the geographical regions must take place in the Americas. A close look at the Table summarizing the location of previous Meetings in the Americas shows that Canada, USA and parts of South America have hosted such meetings. On the other hand, Central America including Mexico has never had the meeting. Perhaps this should be kept in mind when a final decision is to be taken at the next Meeting of Council in Hong Kong. Again, it is important that we receive high quality proposals from which the best possible venue can be selected.

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Election of new ISN Officers and Councillors

Election of new ISN Councillors

According to Article 41 of the new Articles of Association of the ISN Members of ISN are invited to nominate candidates for the ISN Council. All Members are eligible candidates.

The composition of the present Council is found on pages 8 of this issue of the ISN News.

Table 1 summarizes the current composition of the Council with an indication of regional distribution of Officers and Council Members and also indicating which Council Members will continue service after 2003.

TABLE 1:
End or continuation of service of elected Officers and present Councillors in 2003

Council Member	Country	Region	Continuation past 2003
V. Adam-Vizi	Hungary	East Europe	+
A. Arutjunyan	Russia	East Europe	-
N. Banik	USA	North America	-
R. Butterworth	Canada	North America	+
P. Dunkley	Australia	Australia	-
A. Garcia	Spain	South Europe	-
F. Garcia de Mello	Brazil	South America	+
F. Hucho	Germany	Central Europe	-
K. Mikoshiba	Japan	East Asia	-
P. Nwoha	Nigeria	Africa	+
S. Pfeiffer	USA	North America	-
P. Roberts	UK	West Europe	+
A. Schousboe	Denmark	North Europe	+
H. Soreq	Israel	Middle East	+
B. Zalc	France	West Europe	+

At the Business Meeting of ISN on August 5, 2003 in Hong Kong six (five elected and one coopted) Councillors will retire after having served 4 years (Article 42 A):

A. Arutjunyan (Russia)
N. Banik (USA)
A. Garcia (Spain)
F. Hucho (Germany)
S. Pfeiffer (USA)
K. Mikoshiba (Japan)

Table 2 indicates the regional distribution of the remaining Council Members as well as a summary of the regional loss of Council Members in 2003.

**TABLE 2:
Regional decreases in the number of Councillors and Officers in 2003**

Region	till 8/2003	past 8/2003	loss in 2003
North America	3	1	2
South America (Latin America)	1	1	0
Western Europe			
– UK	1	1	0
– France	1	1	0
Central Europe	1	0	1
North Europe	1	1	0
South Europe	1	0	1
East Europe	2	1	1
East Asia	1	0	1
South Asia	0	0	0
Middle East	1	1	0
Australia	1	0	1
Africa	1	1	0

According to Article 27 of the new Articles of Association the Council consists of the 3 Officers and of 9 Members. Therefore, five newly elected Councillors will then enter their 4 years' term of service on the Council. The ballot for the election of the new Councillors will take place in the spring of 2003. In making your suggestions of candidates please note that the composition of the Council shall reflect the international representation of the Members of the Society (Article 29).

According to Article 41 each suggestion of a candidate for Council requires: i) the signature of the nominated member, ii) the signatures of 1% of the number of Members, i.e., 16 Members (this normally would include the signature of the proposing member. Please note that not more than one third of the supporting signatures should be from one country. It is suggested that the proposing member and/or the proposed candidate arrange for the supporting signatures to be sent by February 14, 2003, to the ISN Secretary (address see page 9 of this issue).

The Members are invited to nominate candidates for the election of the new ISN Councillors (for nomination form see separate sheet inserted in this issue).

Call for nomination for the election of new ISN Council Members

Members of the ISN are hereby invited to nominate candidates for the ISN Council. All Members of the ISN are eligible candidates.

The following form should be filled in by typewriter (printer) or with CAPITALS. Please state name, address, fax and e-mail number for the candidate nominated.

I suggest the following Member of the ISN as candidate for the ISN Council:

Name of candidate: _____

Address of candidate: _____

Fax number of candidate: _____

E-mail address of candidate: _____

Candidate's consent with the nomination: I agree to be a candidate for ISN Council

Candidate's signature: _____

Name of proposing Member

Signature of proposing Member

Phone: _____

Fax: _____

e-mail: _____

This form should be submitted to the ISN Secretary's office before February 14, 2003:

ISN Secretary, Dr. Arne Schousboe, Dept. Pharmacology, Royal Danish School of Pharmacy, Universitetsparken 2, DK-2100 Copenhagen, Fax No. +45 3530 6021, E-mail: as@dfh.dk

Election of a new ISN Secretary

According to Article 28B of the Articles of Association of the ISN every 4 years the Membership elects a new Secretary. The procedures for nomination and election of candidates are the same as those for the election of Councillors (Article 41; see above "Election of a new ISN Secretary").

At the Business Meeting on August 5, 2003 in Hong Kong the present Secretary, Arne Schousboe, Denmark, will end his four years' period as Secretary and be succeeded by a new Secretary to be elected in the spring 2003. At the Business Meeting 2003 the present Secretary, A. Schousboe, will become President of ISN for two years (Article 28 C and D). Thus far, for the period between the Business Meetings of 2003 and 2005 the ISN Officers will be as follows:

President:	Arne Schousboe	(Denmark)
Treasurer:	Roger Butterworth	(Canada)
Secretary:	to be elected	

If one includes the present 3 Officers P. Dunkley, A. Schousboe and R. Butterworth who till the year 2007 will successively have served as ISN Presidents, till the year 2007 the Presidents (Chairpersons) of ISN will have come from the following 9 countries:

USA:	6	Canada:	2
UK:	4	Australia:	1
Norway:	2	Germany:	1
Denmark:	2	Italy:	1

According to Article 41 each suggestion of a candidate for Secretary requires: i) the signature of the nominated member; ii) the signatures of 1% of the number of Members, i.e., 16 Members (this normally would include the signature of the proposing Member). Please note that not more than one third of the supporting signatures should be from one country. It is suggested that the proposing Member and/or the proposed candidate arrange for the supporting signatures to be sent by February 14, 2003, to the ISN Secretary (address see page 9 of this issue).

The Members are invited to nominate candidates for the election of the new ISN Secretary (for nomination form see separate sheet inserted in this issue).

Call for nominations for the election of the new ISN Secretary

Members of the ISN are hereby invited to nominate candidates for the ISN Secretary. All Members of the ISN are eligible candidates.

The following form should be filled in by typewriter (printer) or with CAPITALS. Please state name, address, fax and e-mail number for the candidate nominated.

I suggest the following Member of the ISN as candidate for the new ISN Secretary:

Name of candidate: _____

Address of candidate: _____

Fax number of candidate: _____

E-mail address of candidate: _____

Candidate's consent with the nomination: I agree to be a candidate for ISN Council

Candidate's signature: _____

Name of proposing Member

Signature of proposing Member

Phone: _____

Fax: _____

e-mail: _____

This form should be submitted to the ISN Secretary's office before February 14, 2003:

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Call for proposals for the 21th ISN Biennial Meeting in 2007

The ISN Officers should like to invite the Membership to make suggestions concerning the location of the Biennial Meeting of ISN for the year 2007. The Biennial International Meetings of ISN have been held in the places shown in Table 1. The evaluation of the lists provides an overview on the number of times the Meeting has been held in the various countries (Table 2) or in the various regions or continents (Table 3). It should be noted that in accordance with the Council decision taken at the Council Meeting in Chicago (see Neurochemistry News Number 1, June 2000) the biennial meetings should be organized in collaboration with the ASN/APSN/ESN on a rotational basis. Since the meetings in 2003 and 2005 are organized jointly with the APSN and ESN, respectively, the meeting in 2007 will be organized jointly with the ASN and therefore will have to take place in the Americas. Proposals of meeting sites within this geographical area should be accompanied by the following information:

1. Name and location of the proposed hotel/institution/congress center meeting site.
2. Possible dates for the meeting which normally shall be in the summer. In the past ISN has held meetings as early as late April to as last as end of September.
3. The anticipated costs for accommodations in the host hotel and/or nearby hotels.
4. The cost and location of lower cost accommodations for students and postdoctoral fellows, e.g. at university dormitories or nearby older but adequate hotels.
5. The possibility of financial supports (or even better the agreement for support from the city, local public organizations, and local companies).
6. A rough estimate of the whole budget and anticipated registration fees (member, non-member and student/emeritus) and what the fees would include.
7. The transportation facilities to and in the host city; proximity of airports, train stations, bus terminals, driving distances from the major cities, distance from hotels to the meeting site (walking distance or transportation required?).
8. The expected weather conditions during the proposed time of the meeting.
9. The local cultural points of interest in the host city.
10. If proposal will be approved of, the proposing persons are supposed to form a Local Committee.

Please send your proposal to the ISN Secretary by March 14, 2003:

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TABLE 1. Locations of the Biennial International Meetings of ISN

Number of Biennial Meeting	Year	City	Country	Continent or Region
1	1967	Strasbourg	France	Europe
2	1969	Milan	Italy	Europe
3	1971	Budapest	Hungary	Europe
4	1973	Tokyo	Japan	East Asia
5	1975	Barcelona	Spain	Europe
6	1977	Copenhagen	Denmark	Europe
7	1979	Jerusalem	Israel	Middle East
8	1981	Nottingham	UK	Europe
9	1983	Vancouver	Canada	North America
10	1985	Riva del Garda	Italy	Europe
11	1987	La Guaira	Venezuela	South America
12	1989	Albufeira	Portugal	Europe
13	1991	Sydney	Australia	Australia
14	1993	Montpellier	France	France
15	1995	Kyoto	Japan	East Asia
16	1997	Boston	USA	North America
17	1999	Berlin	Germany	Europe
18	2001	Buenos Aires	Argentina	South America
19	2003	Hong Kong	China	East Asia
20	2005	Innsbruck	Austria	Europe

TABLE 2. Distribution of Biennial ISN Meetings by countries till 2005

Country	Number of ISN Meetings
France	2
Italy	2
Japan	2
Argentina	1
Australia	1
Canada	1
China	1
Denmark	1
Germany	1
Hungary	1
Israel	1
Portugal	1
Spain	1
UK	1
USA	1
Venezuela	1

TABLE 3. Distribution of Biennial ISN Meetings by continents or regions till 2005

Continent/Region	Number of ISN Meetings
Europe (including Israel)	12
East Asia	3
North America	2
Central America	0
South America	2
Australia	1



ISN



APSN

Joint Meeting of the International Society for Neurochemistry (ISN) and the Asian Pacific Society for Neurochemistry (APSN)

August 3-8, 2003

**Hong Kong Convention and Exhibition Centre
Wanchai, Hong Kong**

Registration fees

Members of ISN/APSN	:	385 USD
Non-members	:	450 USD
Students	:	135 USD
Accompanying persons	:	150 USD

Deadline for submission of abstracts: March 15, 2003

Preliminary Program

Day 1

Plenary Lecture

Masatoshi Takeichi (Japan) – “TITLE”

Colloquia

1. Glial-Neuronal Interactions

Chairs: Arne Schousboe (Denmark) and Farrukh A. Chaudhry (Norway)

- a. Connie R. Jiménez (Netherlands) – “Proteomics of the regenerating rat sciatic nerve after injury”
- b. Helle S. Waagepetersen (Denmark) – “Amino acid exchange between astrocytes and neurons”
- c. Farrukh A. Chaudhry (Norway) – “System A and system N transporters in the glutamine-glutamate/GABA cycle”
- d. Ronald B. Tjalkens (USA) – “Astrocytes – role in the development and disease”

2. Stem Cells, Gene Therapy, and Genomics

Chair: Piu (Bill) Chan (China)

- a. Antonella Consiglio (Italy) – “Lentiviral vectors for *in vivo* gene therapy of metachromatic leukodystrophy: investigation on the mechanisms underlying the therapeutic effect by gene marking studies”
- b. Rodney L. Rietze (Australia) – “Purification of a pluripotent mammalian adult neural stem cell”
- c. Nissim Ben Arie (Israel) – “Generation and differentiation of cerebellar granule neurons is controlled by the transcription factor Math1 via multiple pathways and target genes”
- d. Wei-Yi Ong (Singapore) – “Sequelae of kainite induced neuronal injury”

3. Parkin and Synuclein in Parkinson’s Disease

Chairs: Kenji Ueda (Japan) and Poul Henning Jensen (Denmark)

- a. Kenji Ueda (Japan) – “Pathophysiology of α -synuclein in neurodegenerative diseases”
- b. Poul Henning Jensen (Denmark) – “Novel functions of misfolded α -synuclein”
- c. Nobutaka Hattori (Japan) – “Mutation and function analyses in parkin”
- d. Ryosuke Takahashi (Japan) – “The molecular network responsible for the cytoprotective function of parkin”

4. Physiology and Pathophysiology of KCNQ Potassium Channels: Approach to Cognition Enhancement and Anti-Epilepsy

Chairs: Haruhiro Higashida (Japan) and David A. Brown (United Kingdom)

- a. T.J. Jentsch (Germany) – “Neuronal KCNQ potassium channels: physiology and role in disease”
- b. David A. Brown (United Kingdom) – “Functional characterization of KCNQ channels and diversity of M channels”

- c. Haruhiro Higashida (Japan) – “Scaffold proteins and KCNQ channels: mechanism of channel modulation”
- d. Diomedes Logothetis (USA) – “Regulation of KCNQ channels by PIP2”

Symposia

1. Motors, Cytoskeleton, and Neurodegeneration

Chairs: Irith Ginzburg (Israel) and Eckhard Mandelkow (Germany)

- a. Nobutaka Hirokawa (Japan) – “The mechanism of recognition, binding and transport of receptors and synaptic proteins by molecular motors, KIFs”
- b. Larry Goldstein (USA) – “Linking kinesin-dependent transport pathways to signaling and disease”
- c. Eva-Maria Mandelkow (Germany) – “Neuronal tau protein and dysfunction in Alzheimer disease”
- d. Ralph Nixon (USA) – “TITLE”

2. Cerebrovascular Regulation and Stroke

Chairs: Sookja K. Chung (Hong Kong) and Pak H. Chan (USA)

- a. Michael Moskowitz (USA) – “TITLE”
- b. Michael Chopp (USA) – “TITLE”
- c. Konstantin A. Hossmann (Germany) – “Hemodynamics of stroke evolution”
- d. M. Tohyama (Japan) – “TITLE”

3. Telomerase: From Birth to Death

Chair: Jun-Ping Liu (Australia)

- a. Jun-Ping Liu (Australia) – “Telomerase functions and molecular regulation”
- b. Maria Blasco (Spain) – “Roles of telomerase in brain formation”
- c. Seong-Seng Tan (Australia) – “Expression of telomerase during brain development”
- d. Mark P. Mattson (USA) – “Telomerase and apoptosis in brain development and disease”

4. Oligodendrocytes: Perspectives on Development and Function in Health and Disease

Chairs: Andrew L. Gundlach (Australia) and Wendy Macklin (USA)

- a. Jean-Léon Thomas (France) – “Directional guidance of embryonic oligodendroglial migration: molecular mechanisms and role of semaphorins and netrin-1”
- b. Wendy Macklin (USA) – “Regulation of early differentiation of oligodendrocytes – studies using PLP-EGFP transgenic mice”
- c. Andrew Gundlach (Australia) – “Expression of peptides and their receptors by oligodendrocytes: implications for functional regulation”
- d. Anthony Campagnoni (USA) – “Function of golgi products of the myelin basic protein (MBP) gene in oligodendrocytes, neurons and in lymphoid cells”

Workshops

1. *In Vivo* NMR Spectroscopy in Neurochemistry – Basic and Clinical Applications

Chair: Rolf Gruetter (USA)

2. Stem Cells

Chair: Alex Zhang (China)

Day 2

Plenary Lecture

Martha Constantine-Paton (USA) – “TITLE”

Colloquia

1. Shut-Down of Translation – A Key Stress Response of Neurons

Chair: Wulf Paschen (Germany)

- a. Christopher Proud (United Kingdom) – “Mechanisms for the control of protein synthesis in response to cell stresses”
- b. Maltide Salinas (Spain) – “Mechanisms involved in down-regulation of translation in transient cerebral ischemia”
- c. Bingren Hu (USA) – “Aggregation of proteins in transient ischemia”
- d. Wulf Paschen (Germany) – “Search for common denominators of neuronal cell injury in stroke and degenerative diseases”

2. Neuroinflammation

Chair: Sean Murphy (United Kingdom)

- a. Anne I. Boullerne (USA) – “Multiplex role of nitric oxide in demyelination”
- b. Miguel A. Contreras (USA) – “Interactions between N-3 and N-6 polyunsaturated fatty acids in brain of chronically N-3 deficient rats”
- c. Daniel C. Anthony (United Kingdom) – “CINC-1 is an early hepatic acute phase protein induced by focal brain injury that causes leukocyte mobilization and liver injury”
- d. Sandra J. Hewett (USA) – “Regulation of arachidonic acid release from neural tissue following pathological insults”

3. Functional Role of Neuronal Calcium Sensor (NCS)

Chair: Karl-Heinz Braunewell (Germany)

- a. Robert Burgoyne (United Kingdom) – “NCS-1: a regulator of voltage-gated calcium channels”
- b. Jose Naranjo (Spain) – “DREAM, a calcium-dependent transcriptional repressor”
- c. Kenneth Rhodes (USA) – “KChIPs in the regulation of A-type K channels”
- d. Karl-Heinz Braunewell (Germany) – “VILIP-1 as a regulator of guanylyl cyclase”

4. Nongenomic Mechanisms of Ion Channel Plasticity

Chairs: A. Leslie Morrow (USA) and Lawrence Judson Chandler (USA)

- a. Stephen Moss (United Kingdom) – “Modulation of GABAA receptor function and cell surface expression involves multiple roles of protein kinases”
- b. Sandeep Kumar (India/USA) – “Chronic ethanol consumption alters GABAA receptor endocytosis and recycling via PKC-dependent mechanisms *in vivo*”
- c. Michael Ehlers (USA) – “Activity, mRNA splicing, and surface delivery of NMDA receptors”

- d. Lawrence Judson Chandler (USA) – “Ethanol-induced synaptic targeting of NMDA receptors”

Symposia

1. Ca Signaling at the Synapse

Chairs: Teruo Abe (Japan) and Katsuhiko Mikoshiba (Japan)

- a. Teruo Abe (Japan) – “Synaptic vesicle binding to microtubules and its possible role in synaptic vesicle distribution”
- b. Giampietro Schiavo (United Kingdom) – “Glycerotoxin stimulates neurosecretion by up-regulating N-type Ca²⁺ channel activity”
- c. Anna Greka (USA) – “TRP channels and calcium-dependent cytoskeletal remodeling”
- d. Katsuhiko Mikoshiba (Japan) – “Role of IP3 receptor in neuronal function”

2. Cell Transplants and Gene Therapy: Insights in Neural Repair

Chairs: Araceli Espinosa-Jeffrey (USA) and Hideyuki Okano (Japan)

- a. Evan Snyder (USA) – “Neural stem cells: Developmental insights might teach therapeutic lessons”
- b. Angelo Vescovi (Italy) – “Therapeutic effects of neural stem cell transplantation in multiple sclerosis”
- c. Araceli Espinosa-Jeffrey (USA) – “Repairing dys- and demyelinating disorders by oligodendrocyte progenitor cell transplants”
- d. Hideyuki Okano (Japan) – “Stem cell transplantation into spinal cord dysfunctions”

3. Aquaporins in the Central Nervous System: Structure, Regulation and Functions

Chair: Bernd Hamprecht (Germany)

- a. Peter Agre (USA) – “Aquaporins in health and disease”
- b. Yoshinori Fujiyoshi (Japan) – “Protein structure of aquaporins”
- c. Alan S. Verkman (USA) – “CNS effects of aquaporin deletions”
- d. Soeren Nielsen (Denmark) – “Ontogeny of water transport channels in brain”

4. Alternative Medicine (APSN)

Workshops

1. Regulation of Neurotransmitter Transporters in Health and Disease: Revealing Novel Molecular and Mechanistic Targets

Chair: Georgi Gegelashvili (Denmark)

2. Informatics for Neurochemists

Chair: Neil Smalheiser (USA)

3. Therapeutic Strategies for Neurodegeneration

Chair: Piu (Bill) Chan (China)

Day 3

Plenary Lecture

Mu Ming Poo (USA) – “TITLE”

Colloquia

1. Signaling Mechanisms in Neurons

Chair: Mikoshiba Katsuhiko (Japan)

- a. Sadashiva Pai (USA) – “Heat shock proteins and thrombin signaling in brain”
- b. János P. Kiss (Hungary) – “Effect of nitric oxide on monoamine transporters: a novel form of interneuronal communication”
- c. Yogesh Dwivedi (USA) – “Trophic factors and their mediated extracellular signal-regulated kinase pathway in postmortem brain of suicide victims”
- d. Weihai Ying (USA) – “Mechanisms of poly(ADP-ribose) polymerase- and poly(ADP-ribose) glycohydrolase-mediated ischemic neuronal death”

2. Dietary and Aging Influences on Neuronal Function

Chair: James A. Joseph (USA)

- a. Donald Ingram (USA) – “Slowing brain aging by calorie restriction and calorie restriction mimetics”
- b. James A. Joseph (USA) – “Polyphenolics in successful aging”
- c. HyJung Chung (Korea) – “Molecular inflammation hypothesis of aging based on the anti-aging mechanism of caloric restriction”
- d. Isao Shimokawa (Japan) – “Effect of leptin on gene expression in hypothalamus of calorie restricted rats”

3. Protein Phosphatases in Neuronal Death

Chairs: Susanne Klumpp (Germany) and Josef Krieglstein (Germany)

- a. Claude Klee (USA) – “Calcineurin: a protein phosphatase under the control of Ca^{2+} and reactive oxygen species”
- b. Shinri Tamura (Japan) – “Regulation of SAPK signaling pathways by PP2C – implication in the neuronal differentiation”
- c. Josef Krieglstein (Germany) – “Does PP2C play a role in neuronal apoptosis”
- d. Marc Mumby (USA) – “Targeting proteins dictate a role for PP2A in apoptosis”

4. New Aspects of Glial Function

Chair: Kazuhiro Ikenaka (Japan)

- a. Helmut Kettermann (Germany) – “TITLE”
- b. Yoshihisa Kudo (Japan) – “TITLE”
- c. Erik M. Ullian (USA) – “TITLE”
- d. Shinichi Kohsaka (Japan) – “TITLE”

Day 4

Plenary Lecture

Yoshikuni Mizuno (Japan) – “TITLE”

Colloquia

1. Molecular Aspects of Neurodegenerative Disease

Chair: Yoo-Hun Suh (Korea)

- a. Weiming Xia (USA) – “Presenilinase, γ -secretase, BACE2: up- and down-stream events of A β generation”
- b. Marco A.M. Prado (Brazil) – “Intracellular trafficking of the cellular prion protein”
- c. Xiongwei Zhu (USA) – “Abnormal activation of MAPK pathways in Alzheimer disease”
- d. Hye-Sun Kim (Korea) – “C-terminal fragments of APP exert neurotoxicity by GSK-3 β dependent mechanisms through forming ternary complex with Fe65 and CP2/LSF/LBP1 transcription factor in the nucleus”

2. Extrinsic and Intrinsic Factors Controlling Neuronal Identity

Chair: Melitta Schachner (Germany)

- a. Ismael Galve-Roperh (Spain) – “Cannabinoid inhibition of neuronal development”
- b. Constanze I. Seidenbecher (Germany) – “Functional consequences of the deficiency in extracellular matrix proteoglycans in the mouse brain”
- c. Andrew J. Lawrence (Australia) – “Alcohol and reward: behavioural and neurochemical approaches to study craving and addiction”
- d. Matthias R. Evers (Germany) – “Impairment of L-type Ca²⁺ channel dependent forms of hippocampal synaptic plasticity in mice deficient in the extracellular matrix glycoprotein tenascin-C”

3. New Frontiers in Functional Roles for Lipids in the Nervous System

Chair: Rashmi Bansal (USA) and Klaus-Armin Nave (Germany)

- a. Steve E. Pfeiffer (USA) – “Oligodendrocyte signal transduction mediated by lipid rafts”
- b. Klaus-Armin Nave (Germany) – “The role of proteolipids and cholesterol in myelination”
- c. Ronald Schnaar (USA) – “Function of gangliosides in myelin stabilization and nerve regeneration”
- d. Tony Futerman (Israel) – “Neuronal dysfunction in sphingolipidoses”

4. From Genes to Therapy for Psychiatric Disorders (Sponsored by Novartis)

Chair: John P. Quinn (United Kingdom)

- a. John P. Quinn (United Kingdom) – “Characterization of the function of transcriptional regulatory polymorphisms in monoamine transporters that potentially predispose to psychiatric disorders”
- b. Stephen Hunt (United Kingdom) – “Substance P in depression and addiction”
- c. Mike Bannon (USA) – “Cocaine-responsive genes in human brain: This and ‘DAT’”
- d. Graeme Bilbe (Switzerland) – “Prenatal stress produces behavioural and molecular phenotypic changes that mimic aspects of schizophrenia”

Symposia

1. Regulation of Neuroprogenitor Cells

Chair: Fulton T. Crews (USA)

- a. Perry F. Bartlett (Australia) – “Suppressor of cytokine signalling (SOCS) in regulating neuronal stem cell differentiation”
- b. Gerd Kempermann (Germany) – “How can ‘activity’ regulate adult hippocampal neurogenesis?”
- c. Elizabeth Gould (USA) – “Stress, deprivation and neurogenesis”
- d. Jeffrey D. Macklis (USA) – “Cellular repair of complex cortical circuitry by neural precursors”

2. Neuroimmunology

Chair: Raymond Chuen-Chung Chang (Hong Kong)

- a. V. Hugh Perry (United Kingdom) – “The contribution of systemic infection to CNS inflammation”
- b. Wolfgang Streit (USA) – “The role of microglia in neuroprotection”
- c. Michael J. Mullan (USA) – “The role of CD40 in AD pathogenesis”
- d. Narayan R. Bhat (USA) – “Glial cell signaling in neuroinflammation: the kinase connection”

3. Signaling Through Proteolysis

Chairs: Leszek Kaczmarek (Poland) and Fred van Leuven (Belgium)

- a. Leszek Kaczmarek (Poland) – “Matrix metalloproteinase-9 in neuronal plasticity”
- b. Fred van Leuven (Belgium) – “Presenilin-1: more than a proteinase!?”
- c. Takaomi C. Saido (Japan) – “A β metabolism and Alzheimer’s disease”
- d. Denis Monard (Switzerland) – “Control of endogenous serine protease activity: from culture to behavior”

4. Synaptogenesis and Synaptic Plasticity

Chair: Eunjoon Kim (Korea)

- a. Craig C. Garner (USA) – “Nascent assembly of CNS synapses”
- b. Eunjoon Kim (Korea) – “Involvement of synaptic proteins in neuronal transport”
- c. Christoph M. Schuster (Germany) – “Molecular mechanisms of experience-dependent plasticity in *Drosophila*”
- d. Edward B. Ziff (USA) – “Protein interactions and the control of AMPA receptor trafficking”

Workshops

1. Neurotransmitter Systems in Schizophrenia

Chair: Gavin Reynolds (United Kingdom)

2. Proteomics

Chairs: Peter Hojrup (Denmark) and Wei-Ping Gai (Australia)

Day 5

Plenary Lecture

Heinrich Betz (Germany) – “TITLE”

Colloquia

1. Neurotransmitters and Receptors

Chair: Roger Butterworth (Canada)

- a. Ying Qu (USA) – “*In vivo* imaging serotonergic neurotransmission mediated phospholipase A2 signal transduction on different pharmacological and transgenic rodent models”
- b. Tue G. Banke (USA) – “Activation of individual NMDA receptor channels reveals multiple subunit-associated gates”
- c. Zuhang Sheng (USA) – “Neurotransmitter release and assembly of the active zone”
- d. Shigeo Okabe (Japan) – “Dynamic behavior of the postsynaptic density proteins and its regulation by neuronal activity”

2. Normal and Aberrant Function of Reactive Oxygen Species: From Synaptic Plasticity to Alzheimer’s Disease

Chair: Eric Klann (USA)

- a. Eric Klann (USA) – “Reactive oxygen species: cellular signaling molecules critical for hippocampal LTP and memory”
- b. Menahem Segal (Israel) – “Reactive oxygen species in plasticity: pharmacological and genetic manipulations”
- c. Marina Lynch (Ireland) – “The interaction between the proinflammatory cytokine IL-1 and reactive oxygen species in the hippocampus and their effect on LTP”
- d. Paul Chapman (United Kingdom) – “Inflammation and oxidation in transgenic models of AD: physiology and behaviour”

3. Cellular Mechanisms and Molecular Signaling in Cell Death/Survival in CNS Injuries

Chair: Pak H. Chan (USA)

- a. Chung Y. Hsu (Taiwan/USA) – “Transcriptional factors in ischemic cell death”
- b. Jun Chen (USA) – “DNA damage and repair in stroke”
- c. Bingren Hu (USA) – “Synaptic damage after global cerebral ischemia”
- d. Pak H. Chan (USA) – “Oxidative signaling as a molecular switch in cell survival/death in CNS injuries”

4. Neuroactive Steroids: Functions, Mechanisms of Action and Physiologic Relevance

Chair: Synthia Mellon (USA)

- a. A. Leslie Morrow (USA) – “TITLE”
- b. Rainier Rupprecht (Germany) – “TITLE”
- c. Giovanni Biggio (Italy) – “TITLE”
- d. Delia Belelli (United Kingdom) – “TITLE”

Symposia

1. Mechanisms for Cell Migration and Growth Cone Guidance

Chair: Fujio Murakami (Japan)

- a. Andre Goffinet (Belgium) – “Genetic control of neuronal migration”
- b. Susan McConnell (USA) – “Genetic analysis of telencephalic development”
- c. Tatsumi Hirata (Japan) – “Ventral tangential migration of neurons in the telencephalon”
- d. Fujio Murakami (Japan) – “Common mechanisms of commissural axon guidance and transmedian migration of neurons”

2. Ten Years of Knocking Out Genes in the Brain

Chairs: Marcelo Rubenstein (Argentina) and Malcolm Low (USA)

- a. Malcolm Low (USA) – “Genetic manipulation of the proopiomelanocortin gene reveals multiple roles in neuroendocrine homeostasis”
- b. Brigitte Kieffer (France) – “Knocking out the opioid system: what’s new?”
- c. Marina Picciotto (USA) – “Nicotine, addiction and learning: studies using constitutive and conditional knockout of the high affinity nicotinic acetylcholine receptors”
- d. Alcino Silva (USA) – “Molecular and cellular cognition. Lessons from mutant mice”

3. Pre-mRNA Processing in Neuronal Malfunctioning (Sponsored by Promega)

Chair: Jeffrey Rothstein (USA)

- a. Jeffrey Rothstein (USA) – “Glutamate transporters – alternate spliced and aberrant spliced transporters participate in normal and abnormal synaptic transmission”
- b. Hermona Soreq (Israel) – “Concerted modulations in transcription and alternative splicing under stress”
- c. Douglas L. Black (USA) – “Alternative splicing and the regulation of neuronal gene expression”
- d. Peter H. Seeburg (Germany) – “Synaptic activity-induced conversion of intronic to exonic sequences”

4. Molecular Pathogenesis of Alzheimer’s Disease

Chairs: Yoo-Hun Suh (Korea) and Virginia M.Y. Lee (USA)

- a. Yasuo Ihara (Japan) – “Relationship between gamma and epsilon cleavage of APP”
- b. Yoo-Hun Suh (Korea) – “C-terminal fragments of APP; its neurotoxic mechanisms and involvement in gene transcription”
- c. Virginia M.Y. Lee (USA) – “Separate and linked neurodegeneration between tauopathies and synucleinopathies”
- d. Colin Masters (Australia) – “New therapeutic strategies for Alzheimer’s disease”

	Plenary	Colloquia	Symposia	Workshop
Monday	Masatoshi Takeichi	Glial-neuronal interactions Stem cells, gene therapy, and genomics Parkin and synuclein in Parkinson's disease Physiology and pathophysiology of KCNQ	Motors, cytoskeleton, and neuro-degeneration Cerebrovascular regulation and stroke Telomerase: from birth to death Oligodendrocytes: perspectives on development	In vivo NMR spectroscopy in neurochemistry Stem cells
Tuesday	Martha Constantine-Paton	Shut-down of translation - a key stress response Neuroinflammation Functional role of neuronal calcium sensor (NCS) Nongenomic mechanisms of ion channel plasticity	Ca signaling at the synapse Cell transplants and gene therapy Aquaporins in the central nervous system Alternate medicine (APSN)	Regulation of neurotransmitter transporters Informatics for neurochemists Therapeutics strategies for neurodegeneration
Wednesday	Mu Ming Poo	Signaling mechanisms in neurons Dietary and aging influences on neuronal function Protein phosphatases in neuronal death New aspects of glial function	FREE AFTERNOON	
Thursday	Yoshikuni Mizuno	Molecular aspects of neurodegenerative disease Extrinsic and intrinsic factors controlling neuronal New frontiers in functional roles for lipids From genes to therapy for psychiatric disorders	Regulation of neuroprogenitor cells Neuroimmunology Signaling through proteolysis Synaptogenesis and synaptic plasticity	Neurotransmitter systems in schizophrenia Proteomics
Friday	Heinrich Betz	Neurotransmitters and receptors Normal and aberrant function of reactive oxygen Cellular mechanisms and molecular signaling in Neuroactive steroids: functions, mechanisms of	Mechanisms for cell migration and growth cone Ten years of knocking out genes in the brain Pre-mRNA processing in neuronal malfunctioning Molecular pathogenesis of Alzheimer's disease	FREE TIME
Please note that Posters will be displayed during lunch breaks on all days except Wednesday!				

Satellite Meetings

Premeeting Satellites

Frontiers of Myelin Biology and Demyelinating Diseases

Rashmi Bansal (USA) and Steven Pfeiffer (USA)

Exact dates and location to be decided

Neurobiology of Glycolipids and Glycoproteins

Robert K. Yu (USA)

July 30 to August 1, Taipei, Taiwan

Environmental Factors in Aging and Neurodegenerative Disorders

Albert Y Sun (USA)

July 30 to August 2, Taipei, Taiwan

Cellular and Molecular Mechanisms of Drug Abuse

Syed F. Ali (USA)

July 30 to August 1, Nagoya/Kyoto, Japan

Postmeeting Satellites

Metal-induced Neurodegeneration: from Global Exposure to Individual Susceptibility

Michael Aschner (USA), Wei Zheng (USA) and Julian Chen (China)

August 9-10, Xi'an, China

Sixth International Meeting on Brain Energy Metabolism

Albert Yu (China)

August 10-12, Beijing, China

Mechanisms of Oxidative Stress in Neurodegenerative Disorders

Bill Piu Chan (China)

August 10-13, Beijing, China

Sixth International Meeting for Brain Energy Metabolism

Transporters, mitochondria and neurodegeneration

August 9 - 12, 2003

Beijing, China



An official satellite meeting of the
19th Biennial Meeting of the ESN/APSIN Joint Meeting - 2003 Hong Kong
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Deadline for Abstracts :

1 June 2003

<http://www.brainenergy2003.org>

Proposed Scientific Program

1. Monocarboxylate transport in the brain
2. Amino acid transport in the brain
3. Mitochondrial heterogeneity
4. Mitochondrial permeability transition
5. Neurodegeneration
6. Neuroprotection
7. Microglia and oligodendrocyte metabolism
8. Iron metabolism in the brain
9. Neuronal inhibition and excitation
10. Measuring brain bioenergetics
11. Mitochondrial stress

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Sixth International Meeting for Brain Energy Metabolism

9-12 August 2003 – Beijing, China

9 August 2003 (1/2 day)

Opening ceremony & Keynote speech

10 August 2003

Session 1: Measuring brain bioenergetics

(Chair: Ursula Sonnewald)

Peter Morris, Nottingham, UK

Rolf Gruetter, Minneapolis, USA

Albert Gjedde, Aarhus, Denmark

Session 2: Neurodegeneration and nitrosative stress

(Chair: John Clark, Roger F. Butterworth)

Juan P. Bolaños, Salamanca, Spain

Guy C. Brown, Cambridge, UK

Agustina Garcia, Barcelona, Spain

Roger F. Butterworth, Montreal, Canada

Session 3: Functional implications of mitochondrial heterogeneity

(Chair: TBA)

Garry K. Brown, Oxford, UK

Nicholas J. Hoogenraad, Bundoora, Australia

Helle Waagepetersen, Copenhagen, Denmark

Michele Merle, Bordeaux, France

Gary Fiskum, Baltimore, USA

Session 4: The mitochondrial permeability transition

(Chair: Michael D. Norenberg)

John J. Lemasters, Chapel Hill, USA

Michael D. Norenberg, Miami, USA

Tadeusz Wieloch, Lund, Sweden

Patrick Sullivan, Lexington, USA

11 August 2003

Session 5: Monocarboxylate transporters in brain: what, where and why?

(Chair: Leif Hertz)

Mary C. McKenna, Baltimore, USA
Luc Pellerin, Lausanne, Switzerland
Lester R. Drewes, Duluth, USA
Ian A. Simpson, Hershey, USA

[N.B. Subject to speaker availability]

Session 6: The role of glutamine and amino acid transport in inhibition and excitation

(Chair: Arne Schousboe, Jon Storm-Mathisen)

Jon Storm-Mathisen, Oslo, Norway
Douglas L. Rothman, New Haven, USA
Andreas Plaitakis, Heraklion, Greece
Susan M. Hutson, Winston-Salem, USA

Session 7: Metabolism of microglia and oligodendrocytes

(Chair: Ralf Dringen)

Gerald Münch, Leipzig, Germany
Gary E. Gibson, White Plains, USA
Ralf Dringen, Tübingen, Germany
José M. Medina, Salamanca, Spain

12 August 2003 (1/2 day)

Session 8: Iron metabolism of the brain in health and disease

(Chair: James Connor)

James Connor, Pennsylvania, USA
Moussa B.H. Youdim, Haifa, Israel
Barry Halliwell, Singapore
Stephen R. Robinson, Melbourne, Australia

Session 9: Strategies and targets for neuroprotection

(Chair: Simon J.R. Heales)

Simon J. R. Heales, London, UK
Chris E. Cooper, Reading, UK
Douglas L Feinstein, Chicago, USA
Feng-Yan Sun, Shanghai, China

Closing ceremony

Also featuring

Poster sessions
Short presentation sessions

See the meeting website for details on registration, accommodation, abstract and poster submission.

www.brainenergy2003.org

Sixth International Meeting for Brain Energy Metabolism Transporters, Mitochondria and Neurodegeneration

Beijing, China – 9-12 August 2003

Meeting Registration Offices

- * Overseas participants (Attn: Dr. Richard Collins)
Hong Kong DNA Chips Ltd, 1805-6, Lu Plaza, 2 Wing Yip St, Kowloon, Hong Kong, China
Tel: (852) 2111 2123
Fax: (852) 2111 9762
E-mail: rcollins@dnachip.com.hk

- * Mainland China participants only (Attn: Dr. Albert C. H. Yu)
Neuroscience Research Institute, Peking University, 38 Xue Yuan Rd, Beijing 100083, China
E-mail: achy@dnachip.com.hk

MEETING REGISTRATION FORM

Deadline for advance registration is 1 June 2003

Complete one form for each participant and mail or fax with appropriate payment.

Title:

First name:

Family name:

Institute/Company:

Building/Street:

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Status

(tick/check the box that applies)

- | | |
|---|--------|
| <input type="checkbox"/> Committee member | waived |
| <input type="checkbox"/> Invited speaker | waived |
| <input type="checkbox"/> Registrant | 200 |
| <input type="checkbox"/> Student | 125 |
| <input type="checkbox"/> Guest | 100 |

Fee (USD)

N.B. Guests are not allowed to enter the scientific sessions.

Late registration (after 1 June 2003)

Registrant: USD 225 / Student: USD 135 / Guest: USD 110

- Special needs (tick/check this box if you have a special need and provide details, e.g. mobility impaired, hearing impaired, vegetarian, etc):

I will submit an abstract YES NO

I will present a poster YES NO
Abstract/Poster subject category*

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* See the Abstract/Poster Submission page for details

** Applies only to those who submit an abstract

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**Sixth International Meeting for Brain Energy Metabolism
Transporters, Mitochondria and Neurodegeneration
Beijing, China – 9-12 August 2003**

Hotel registration form

Deadline for advance registration is 1 June 2003

Complete this form and send it by fax to (852) 2111 9762 attention to Dr. Richard Collins. For security reasons, do not E-mail this information.

The conference organisers have reserved a block of rooms at the XiangShan (Fragrant Hill) Hotel, Beijing for the duration of the meeting. Registrants may reserve a room at the resort using this form. A discounted rate will apply provided reservations are received before 1 June 2003. After this date, or after the block of reserved rooms has been fully booked, a different rate may apply.

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(Double room
plus extra bed)
RMB 420
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|--|--|--|

N.B. Rates are per room per night, inclusive of breakfast, service charges and relevant taxes.

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Number of nights: _____

- Smoking
 Non-smoking
 Special needs (please describe, e.g. mobility impaired): _____

Title: _____ First name: _____ Family name: _____

Institute/Company: _____

Building/Street: _____

City/State/Province: _____

Country: _____

Zip/Postal Code: _____

Tel: _____ Fax: _____

E-mail: _____

I will share my room with:

Title: _____ First name: _____ Family name: _____

Institute/Company: _____

Building/Street: _____

City/State/Province: _____

Country: _____

Zip/Postal Code: _____

Tel: _____ Fax: _____

E-mail: _____

Payment information

We only accept credit card payment. *Do not send cash by mail.*

Amount: US \$ _____

Card type: VISA Mastercard

Name as it appears on card: _____

Card number: _____ - _____ - _____ - _____ Expiry date: ____ / ____

Signature: _____

N.B. Payment by personal cheque and direct bank transfer will be available soon.
Please check the meeting website www.brainenergy2003.org for regular updates.

2003 ISN/APSN Travel Award Application

The ISN/APSN proposes to offer travel awards to young investigators in order to allow them to attend the 19th Biennial Meeting, to be held in Hong Kong from August 3-8, 2003, as a joint meeting of the ISN/APSN.

Investigators who plan to submit abstracts as the first author are eligible to compete for awards for partial support to attend the meeting. The actual amount awarded to any individual will depend on projected travel costs and is not expected to cover the full expenses of the recipient. In addition, awardees will be reimbursed at the meeting for the registration fee they have paid. Preference will be given to younger investigators. This is to be interpreted as less than eight (8) years from award of first doctoral degree. Priority will be given to persons who have not received support previously and who do not hold a permanent senior position.

**Applications should be sent to the Chair of the Travel Award Committee:
Professor Vera Adam-Vizi, Dept. of Biochemistry, Semmelweis University of
Medicine, Puskin St 9, P.O. Box 262, H-1444 Budapest
E-mail: av@puskin.sote.hu or Fax: +36 1 267 0031.**

Applications should preferably be sent by E-mail [an attachment from a standard Microsoft (Windows or Macintosh), or WordPerfect program can probably be handled – you will be informed if there is a problem]. A plain text file in ASCII format with lines of less than 80 characters is the safest option. Application may also be sent by fax or postal mail.

DEADLINE FOR RECEIPT: JANUARY 31, 2003

Receipt will be confirmed only if an E-mail address is included.



2003 ISN/ APSN TRAVEL AWARD APPLICATION

FAMILY NAME: _____ AGE: _____

FORENAMES: _____

DOCTORAL DEGREE(S) OR HIGHEST DEGREE, DATE(S) OBTAINED, FROM
WHAT INSTITUTION(S): _____

CURRENT POSITION: _____

DEPARTMENT/UNIT: _____

INSTITUTION: _____

STREET: _____

CITY: _____ STATE: _____

ZIP CODE: _____ COUNTRY: _____

• COST OF TRAVEL US\$ _____

• ACCOMODATION US\$ _____

• SUBSISTENCE US\$ _____

• TOTAL REQUESTED US\$ _____

PLEASE INDICATE IF YOU HAVE PREVIOUSLY RECEIVED TRAVEL SUPPORT FROM THE SOCIETY TO ATTEND AN ISN MEETING

PLEASE WRITE YES OR NO: _____

INCLUDE A HALF-TO-ONE PAGE DESCRIPTION OF THE PROJECT WHICH WILL BE SUBMITTED AS AN ABSTRACT FOR THE MEETING (IF ALREADY SUBMITTED, A COPY OF THE ABSTRACT IS ADEQUATE).

ALSO LIST UP TO 5 PUBLICATIONS IN NEUROCHEMISTRY (AUTHORS, TITLE AND REFERENCE, INCLUDING IN PRESS).



6TH BIENNIAL ADVANCED SCHOOL OF NEUROCHEMISTRY OF THE ISN

JULY 30 – AUGUST 3, 2003

Responses to Trauma in the CNS: Genes to Ethics

**Hong Kong University of Science and
Technology**

The International Society for Neurochemistry (ISN) is sponsoring an educational program focused on the application of molecular and cellular techniques to understanding the signaling cascades that determine outcomes after chronic or acute trauma to the nervous system. Young investigators are invited to apply for the The Sixth Advanced School of Neurochemistry to be held at the Hong Kong University of Science and Technology in the outskirts of Hong Kong, China, immediately prior to the '03 ISN/APSN Meeting in Hong Kong to be held in August, 2003. We expect that the tradition of international camaraderie and joyful learning in a relaxed environment surrounded by a spectacular setting of the ISN School will make this a memorable event for all involved. At the '01 "School" in Argentina there were faculty and young scientists from 30 countries.

Faculty (all * have accepted): Drs. Regino Perez-Polo, Director (USA), Eckart Gundelfinger (Germany), Polycarp Nwoha (Nigeria), Peter Roberts (UK), Alfreda Stadlin (Hong Kong), Jean DeVellis (USA) – will coordinate the course. Some invited Faculty are Ping Wu* (USA) ~ Hermona Soreq* (Israel) ~ Steffen Rossner* (Germany) ~ David Shine* (USA) ~ John Steeves* (Canada) ~ Ronald Carson* (USA) ~ Freda Miller (Canada) ~ Moses Chao (USA) ~ Luis Parada (USA) ~ Alain Privat (France) ~ Perry Bartlett (Australia) ~ Hideyuki Okano (Japan). Through a series of lectures and informal discussions with an expert faculty the course will acquaint participants with our present understanding as to the role of inflammatory and cell death promoting signal cascades in the nervous system in various experimental paradigms, with an emphasis on basic and clinical applications. Participants will also have the opportunity to present and discuss with faculty their work in poster form. All faculty and young scientists will share meals and accommodations during the 4-5 days of the "School". Postgraduates (up to 5 years after Ph.D. or equivalent) and graduate students are encouraged to apply for fellowships, which will include registration, room and board and travel assistance. All attendees will be issued complimentary registration at the ISN/APSN Meeting that follows. To apply kindly submit a curriculum vitae (two page maximum), a letter of intent stating why you wish to attend, list of publications (if appropriate) and two letters of recommendation from

faculty at an academic institution or research institute, or biotechnology company. Only electronic submissions will be considered. In order to maintain an atmosphere of informal exchanges the number of participants will be restricted.

Applications must be received via email by January 15, 2003 to be considered. Because of the obvious risk involved in receiving attachments from unknown sources, kindly paste the information to the email submission. All materials are to be sent to:

Professor Regino Perez-Polo, Ph.D.
301 University Blvd., UTMB
Galveston, Texas, 77555-0652, USA
Tel: 01-409-772-3667; Fax: 01-409-772-8028
Email: Regino.Perez-Polo@UTMB.EDU

Preliminary Program & Plan

**ISN Advanced Neurochemistry School
Responses to Trauma in the CNS
Hong Kong University of Science and Technology**

Committee:

Eckart Gundelfinger (Germany) – Synaptic Plasticity
Polycarp Nwoha (Nigeria)
Regino Perez-Polo, (USA) Chair – Genomics
Peter Roberts (UK)
Alfreda Stadlin (Hong Kong) – Addiction Responses
Jean DeVellis (USA) – Neuron-glia Interactions

Faculty

Ping Wu* (USA) – Stem Cell Transplants
Hermona Soreq* (Israel) – Antisense Strategies
Steffen Rossner* (Germany) – Transgenic Models
David Shine* (USA) – Viral Transfections
John Steeves* (Canada)
Ronald Carson* (USA) – Bioethics
Jackie Bresnahan (USA) – Inflammation in Trauma
Freda Miller (Canada) – Signaling Cascades
Moses Chao (USA) – Signaling Cascades
Luis Parada (USA) – Transgenic Models
Alain Privat (France) – Gene Therapy
Perry Bartlett (Australia) – Stem Cells
Hideyuki Okano (Japan)

Tentative Schedule

July 30th Arrivals and welcome in afternoon
18:00-20:00 Reception and dinner
20:00-21:00 Evening Lecture ~ 1 hour

July 31st
8:45- 9:00 Welcome
9:00-12:00 5 Talks ~ 4 hours
12:00-14:00 Lunch
14:00-16:00 Student Presentations ~ 2 hours
16:00-17:00 Discussion
17:00-19:00 Free Time
19:00-21:00 Dinner
21:00-22:00 1 Talk ~ 1 hour

August 1st

9:00-13:00 5 Talks ~ 4 hours
13:00-14:00 Lunch
14:00-16:00 Student Presentations ~ 2 hours
16:00-21:00 Excursion
21:00-22:00 Evening Discussion ~ 1 hour

August 2nd

9:00-13:00 5 Talks ~ 4 hours
13:00-14:00 Lunch
14:00-18:00 Discussions & Summaries ~ 4 hours* *
18:00-20:00 Dinner
20:00-22:00 Evening Discussions & Farewells ~ 2 hours

August 3rd Departure in morning for ISN meeting

* Have accepted

** During discussions we will break out into smaller groups of students and faculty to get better representation of views. There will be 4 groups. At the end each group will elect a representative to propose views and conclusions and even further questions to be considered.

We expect about 40-50 students

Students and faculty receive free room and board.

Accompanying members must pay their way.

Some students will receive travel funds.

All students will be required to apply for funding from ISN.

All students and faculty receive registration waivers at the ISN meeting

Some faculty will receive partial travel funds if they are not covered by ISN

Faculty are encouraged to stay for the whole school

Students are encouraged to submit an abstract to be presented as a poster, which may be the same as the one being presented at the meeting.

All faculty must send a 1-2 page plan lesson or outline early enough to assemble in Program Book.

In every talk we will emphasize approaches and methodology and use our own research for an example.

Evening sessions will end when all are ready for bed, they may go beyond allotted times.

We will strive for geographical diversity and gender balance

Faculty and students eat all meals together

Hong Kong University has separate male and female sleeping quarters. We have 15 rooms for 30 females and 15 rooms for 30 males. We have 10 single and 4 twin rooms for faculty. We will need a few more rooms for faculty.

**19th Biennial Meeting of The International Society For Neurochemistry
Joint meeting with the Asian Pacific Society for Neurochemistry
Hong Kong, China, August 3-8, 2003.**

ISN YOUNG SCIENTIST LECTURESHIP AWARDS

These ISN awards are to recognize research achievements of promising young scientists involved in neurochemistry research. Up to two young scientists, 38 years of age or younger at the time of proposal, may be given the ISN award consisting of a paid trip to the joint ISN/APSNe Meeting in Hong Kong, China, where they will present a 30 minute lecture.

Candidates can be proposed either by the head of a department or a senior scientist. Nominating letters should be accompanied by a curriculum vitae and a short description (with references) of the research accomplishments of the candidate. Proposals should be sent to the Chairman of the ISN Conference Committee.

Dr. Agustina Garcia

University Autònoma de Barcelona
Institut de Biologia Fundamental
V. Villar Palasi
Bellaterra
08193 Barcelona
SPAIN
Phone +34 3 581 2802
Fax +34 3 581 2011
E-mail ibftina@blues.uab.es

**DEADLINE FOR RECEIPT OF YOUNG SCIENTIST LECTURESHIP AWARD
NOMINATION**

January 31, 2003

Please note the extended deadline.

General Business Meeting of ISN 2003 Preliminary Agenda

**Hong Kong, China
Tuesday, August 5, 2003 (in the evening)**

1. Opening of the Meeting
2. Treasurer's Report
3. Secretary's Report
4. Results of elections for new Secretary and Councillors
5. Report on the activities of ISN Committees
6. Matters arising from the First ISN Council Meeting
7. Status of the Innsbruck Meeting 2005
8. Proposals for the 21th ISN Meeting in 2007
9. Membership dues
10. Any other business

The Business Meeting will be held in connection with the joint ISN/APSIN Meeting in Hong Kong, August 3-8, 2003.

Committee Reports

Reports from CC Sponsored Meetings

Meeting Report

***2nd International Conference on Heme Oxygenase (HO/CO) and Cellular Stress Response. Held in Catania, Italy; 6- June 9 June 2002.**

Heme Oxygenase, Carbon monoxide and Cellular stress Response in the Nervous System: The good and the enigmatic.

V. Calabrese (a), G. Scapagnini (b), S. Latteri (a), Butterfield DA (c) and U. Scapagnini (d)

(a) Dept. of Chemistry, University of Catania, Italy; (b) Blanchette Rockefeller Neurosciences Institute, West Virginia University, Rockville, MD 20850, USA; (c) Dept. Chemistry, University of Kentucky, Lexington, KY 40506, USA; (d) Dept. of Pharmacology, University of Catania, Italy.

Heme oxygenase regulates cellular heme which is required for mitochondrial function, catalase, drug metabolism and cytochrome P450 activity, cyclooxygenase and prostaglandin synthesis, nitric oxide synthase and nitric oxide formation, soluble guanylate cyclase and cGMP. The heme oxygenase (HO) system in animals provides the first and rate-limiting step in heme degradation. HO cleaves the heme ring via oxidation at the alpha methene bridge to give biliverdin, gaseous carbon monoxide and free iron. Biliverdin is subsequently converted to bilirubin by biliverdin reductase, and both these molecules can act as intracellular antioxidants. All the byproducts of HO activity play a significant role in physiological cell functions, which can explain the extensive presence of HO isoforms in several tissues and cellular types. Although the biological role of HO proteins remains to be fully elucidated, their relevance in cellular stress response has been widely demonstrated in a variety of tissues. In the CNS, the heme oxygenase pathway has been shown to act as a fundamental defensive mechanism for neurons exposed to an oxidant challenge. Deregulation of the HO system has been associated with the pathogenesis of several neurodegenerative disorders. Furthermore, HO activity is implicated in the brain production of carbon monoxide(CO), a putative neurotransmitter, which, similarly to nitric oxide, is able to activate guanylate cyclase performing a variety of physiological functions. An understanding of the biology and pathobiology of the HO system in the nervous system is therefore important, and is likely to underpin the development of more effective therapeutic strategies for treating injury and disease. With the breathtaking view of the Etna volcano as backdrop, HO/CO and related

topics were the focus of a recent pioneering meeting* that brought together an international contingent of researchers from the fields of heme oxygenase and cellular stress response research.

To date three isozymes of heme oxygenase have been identified: the inducible HO-1, also known as hsp32, and the constitutive HO-2 and HO-3, which are products of individual genes. While HO-2 and HO-3 share a very high level of homology, their sequences consistently diverge from HO-1. The most relevant similarity between HO-1 and HO-2 consists in a common 24 AA domain (differing in just one residue) called the "HO signature", that render both proteins extremely active in their ability to catabolize heme. HO-3, cloned only in rat to date, presents several mismatches in the heme signature motif, resulting in a lower heme catalytic activity. As highlighted by Giovanni Scapagnini (Rockville, MD) both HO-2 and HO-3, but not HO-1, are endowed with two Cys-Pro residues considered the core of the heme-responsive motif (HRM), a domain critical for heme binding but not for its catalysis. HO-3 transcript brain distribution showed higher levels of expression in the cerebellum, the hippocampus and also in some specific cortical districts. Hippocampal gene expression profiles within energetic molecular cascades during learning and memory were presented by Daniel Alkon ((Rockville, MD). Using real time PCR and in situ hybridization he provided compelling evidence implicating CO as a putative neurotransmitter which can influence neuroendocrine regulation, as well as long-term synaptic changes (LTP, LTT). Characterization of the regional and cellular distribution of HO isoforms is fundamental to understand their functional role in the CNS. Previous works have shown that in the rat brain HO-1 is poorly expressed, being constitutively present only in few cellular subtypes, but it is quickly and highly inducible by its substrate heme and by other various stress-associated agents. As highlighted by Vittorio Calabrese (Catania, Italy), this process is complex and involves multiple factors that are poorly understood. HO-3 mRNA was detectable in type I astrocytes and not in neurons. Analysis of HO-3 mRNA sequence revealed that it corresponds in the 5' portion to the sequence of a L-1 retrotransposon a member of a family of retrotransposons recently involved in evolutionary mechanisms.

Up-regulation of HO-1 promotes mitochondrial sequestration of non-transferrin-derived iron in cultured rat astroglial cells and also HO-1 protein is highly over-expressed in neurons and astrocytes residing within Alzheimer (AD)-affected hippocampus and temporal cortex. As shown by Hyman Scipper (Montreal, CAN) these observations suggest that HO-1 over-expression may contribute to the pathological iron deposition and mitochondrial damage documented in AD and other age-related diseases.

A series of presentations highlighted the dynamic and broad scope of HO induction or repression as defense strategy in mammalian cells. Shigeki Shibahara (Sendai, Japan) discussed the implications of HO-1 up-regulation in hypercholesterolemic rabbits, where accumulation of bilirubin IX α in foam cells of atherosclerotic lesions may exert an antioxidant anti-atherogenic effect. Data also were presented regarding the implication of down-regulation of HO-1 expression in human cells.

Fritz Bach (Boston, MA, USA) provided a perspective of the role of HO-1 in transplantation. Allo- and xenotransplantation, in addition to their intrinsic importance, are models for studies of cell mediated immunity and vascular biology, respectively. Hearts from HO-1 deficient mice were rapidly rejected when transplanted into rats treated with cobra venom factor and cyclosporin A; normal hearts survived indefinitely. If donor and recipient were treated with CO, the HO-1 deficient hearts survived indefinitely. CO also substitutes for HO-1 by suppressing development of post-transplant arteriosclerosis and ischemia-reperfusion injury. These findings emphasize the importance of all HO products in the regulation of allo-immune response.

Cyclooxygenase (COX) and soluble guanylate cyclase are heme proteins that generate prostaglandin and cGMP, which participate in the regulation of vascular function in normal and pathological conditions. Chronic expression of HO-1, by limiting cellular heme, decreases COX-2 activity, thereby decreasing inflammatory response, as reported by Nader G. Abraham (NY, USA). To identify target genes involved in HO-1 mediated regulation of inflammation and cell cycle, endothelial cells transduced with human HO-1 gene using retroviral vector, and gene array as well as real time PCR were employed to identify, among 2400 genes, known and novel differentially expressed genes. Major findings include up-regulation of VEGF, transcriptional factors, signalling molecules, prostaglandin transporter and ubiquitous proteasome.

HO-1 is a redox-sensitive inducible protein that provides efficient cytoprotection against oxidative stress. Roberto Motterlini (Middlesex, UK) highlighted the potency of plant polyphenolic compounds as novel inducers of HO-1. Caffeic acid phenethyl ester (CAPE) a plant-derived phenolic agent markedly increases HO-1 activity and protein in astrocytes. Similar results were obtained with curcumin-95, a mixture of curcuminoids commonly used as dietary supplements. The potency of curcumin and CAPE appears to be much higher compared to other well known stimulators of the HO-1 gene. This study identifies a new class of natural substances that could be used therapeutically as inducers of HO-1 in protection of vulnerable tissues against inflammatory and neurodegenerative disorders.

The presence of abnormal, aggregated proteins is a common feature of neurodegenerative diseases, ranging from the plaques and tangles of Alzheimer's disease to the Lewy body of Parkinson's disease (PD). Sometimes the abnormal proteins may be products of mutated genes or faulty mRNA splicing or translation, examples being the parkin and α -synuclein mutations associated with some cases of familial PD. Barry Halliwell (Singapore) provided elegant studies tracing a perspective on the role of defective protein clearance, via proteasome, as a major cause of neuronal cell loss in the neurodegenerative diseases. Nitration may play a key role in protein aggregation, and experiments measuring oxidative damage, protein nitration, protein aggregates and cell death in the presence of proteasome inhibitors, abnormal proteins (α -synuclein, parkin and SOD mutations) or oxidative stress were presented in support of this hypothesis.

In Alzheimer's disease abnormal proteolytic processing of the amyloid precursor protein (APP) by a putative enzyme, γ -secretase, favours the increased formation of the amyloidogenic and potentially toxic amyloid β -peptide. The role of oxidative stress in AD brain and models to investigate roles of α 1 (1-42), apoe allele, and HO-1 was discussed by Allan Butterfield (Lexington, KY, USA), and proteomic identification of specifically oxidized proteins (creatine kinase, glutamine synthetase, and ubiquitin C-terminal hydrolase) shown. The implications of these findings in AD pathogenesis, for decreased energy utilization, increased excitotoxicity (especially when coupled to HNE modification of Glt-1), and a decreased ubiquitin pool availability (with consequent decreased proteasome degradation of proteins), were presented in elegant studies. Finally, initial studies showed that neurons exposed to A β (1-42) have increased expression of HO-1, consistent with a cellular response to oxidative stress.

Clearly, much remains to be learned about the role of HO/CO in health and diseased nervous system, but there is also good reason to believe that we can use these informations to improve treatments of a variety of CNS disorders.

The ISN money was used to cover Prof. Barry Halliwell travel expenses.

Prof. Barry Halliwell, distinguished invited speaker, travelled from Singapore to Catania and back to Singapore on Singapore Airlines flights SQ322 and SQ339. The cost of the ticket was 8706.00 Singapore Dollars (8,706 Singapore Dollar = 4,923.09 US Dollar). Thanks to the ISN grant we can pay the majority of prof. Halliwell expenses and the remaining part will be paid within the meeting budget.

Meeting Report

VI Workshop on Apoptosis in Biology and Medicine,

Porto Pirogros, Parghelia, Calabria, Italy, 25 – 29 May, 2002

Giacinto Bagetta¹, M. Tiziana Corasaniti² and Bernhard Brüne³

1. University of Calabria, Department of Pharmacobiology, Arcavacata di Rende (CS), Italy
2. University of Catanzaro “Magna Graecia”, Catanzaro, Italy
3. University of Kaiserslautern, Faculty of Biology, Department of Cell Biology, Germany

Address of correspondence: B. Brüne, University of Kaiserslautern, Faculty of Biology, Erwin Schrödinger Strasse 13, 67663 Kaiserslautern, Germany

Topics selected for the VI Workshop on apoptosis in biology and medicine ranged from basic mechanisms of neuronal cell death, immune-modulation in the nervous system, development of experimental models of neurodegenerative diseases to novel pharmacological targets and therapeutical interventions. Organized as a PhD training course on Pharmacology and Biochemistry of Cell Death the meeting brought together around 30 international scientists and 30 PhD students at Parghelia (Vibo Valentia), one of the loveliest spots at the Mediterranean coast. Lively discussions during the morning secessions and intense interactions with young scientists presenting posters during the early afternoon made this workshop an outstanding example in gathering top scientific contributions from leading experts providing latest results, general concepts and overviews as well as fascinating data and experimental details from ongoing PhD programs. The relaxed atmosphere of the meeting in combination with enough time to discuss on controversial aspects with contributions from various disciplines guaranteed the high success of this VI workshop on apoptosis.

The following major topics have been grouped for oral presentations:

Neuroimmune regulation in normal and pathological brain:

Over the past years much effort has been expanded in attempts to inhibit the damaging actions of pro-inflammatory cytokines. At present, promising candidates for development as therapeutics include the peptide hormone thymulin, inhibitors of phosphodiesterases type 4 as well as drugs developed from thalidomide. Their efficacy during sepsis or ischemia/reperfusion will be tested based on the ability to affect the pro- versus anti-inflammatory cytokine balance (Stephen Poole, UK). Emerging concepts show that caspase-3 is active in healthy glial cells and could regulate the expression of the IL-1 family cytokines by modulating proIL-1 β and/or

caspace-1 processing, thus pointing to a non-apoptotic but inflammatory-like role of this enzyme (Karen Palin, UK). In addition, inflammation and recruitment of immune cells that cause the progressive destruction of myelin is noticed in response to several chemokines that are involved in CNS pathologies, among others in tumor progression (Gennaro Schettini, Italy). Moreover, pro-inflammatory conditions such as IL-1 β or β -amyloid peptides operate in astroglial cells to maintain the level of cGMP low when exposed to agents that induce a high NO output (Augustina Garcia, Spain). It will be the goal to understand the signalling network of these factors that contribute to neuroimmune regulation with the intention to attenuate damaging actions of pro-inflammatory cytokines.

Immune and nervous system; a dangerous liaison:

Cell death-inducing ligands such as TNF α , FasL or TRAIL transduce apoptotic signals by binding to their cognate receptors with the notion that tumor cells are sensitive to TRAIL-mediated apoptosis and normal tissue resists (Marion MacFarlane, UK). It is suggested that other factors such as TRAIL-induced NF- κ B activation or inhibitors of apoptosis determine differential sensitivity to TRAIL (Maurizio Memo, Italy). In studies of murine prion disease it is suggested that synapses and axon degenerate, i.e. undergo apoptosis, that evokes an anti-inflammatory profile in the resident microglia indicative of phagocytosis of cellular material (Hugh V. Perry, UK). This process will contribute to balance perpetuation of inflammation and cell demise. Histological damage associated with prion disease is absent in mice that are deficient in the normal prion protein. It is now concluded that certain components of the complement system play important roles in pathogenesis and that transgenic expression of an anti-PrP antibody heavy chain suffices to confer to mice antiprion protection which may open avenues to the development of vaccines (Adriano Aguzzi, Switzerland).

Modulators of tissue function and survival in brain and immune cells:

During the developmental period of sympathetic neuron the life versus death balance is determined by the ratio of p53 and truncated p73. A model is proposed wherein the p53 family members provides a major apoptotic checkpoint in neurons, with life versus death being determined by the balance of full-length, proapoptotic versus N-terminal truncated antiapoptotic family members (Freda Miller, Canada). P53 and p73 transcriptionally regulate N-terminal truncated p73, which in turn functionally inactivates p53 and p73 to attenuate the proapoptotic function of these proteins. This pathway creates a regulatory dominant negative feedback loop to regulated cell demise (Gennaro Melino, Italy). The apoptotic function of p53 is regulated by p53-binding proteins (ASPP; apoptosis stimulated protein of p53) as well. ASPP acts by stimulating the transactivation function of p53 specifically on the promoters of apoptosis-related genes such as Bax and PIG-3, but not on promoters of Mdm2 or cyclins (Xin Lu, UK). Among various agonists that stabilize p53, NO emerged recently (Bernhard Bruene, Germany). NO stabilized a transcriptionally active p53, that interacts with Mdm2, is ubiquitinated and apparently is not exported from the nucleus. Modulation of p53 stability regulation but also the interaction with different regulators such as p73 adds to the life and death decision in cells.

Mechanism of neuronal apoptosis and brain injury:

Apoptosis and necrosis are distinct modes of cell death with the important consideration that switches between both forms of cell demise may be of potential relevance for therapy of neurodegenerative conditions. It is known that the cellular ATP level is critical for deciding the mode of death and that the cleavage of SNARE proteins can initiate a caspase-independent program of neurite self-destruction that is sensitive to neurotrophic stimulation, and a caspase-mediated execution program (Pierluigi Nicotera, UK). Along this line, PARP inhibitors attenuated ischemia or excitotoxic-induced necrosis but not apoptosis (Flavio Moroni, Italy). An new concept in producing NMDA-receptor antagonists postulates to use low-affinity agents instead of high affinity blockers as done in the past, that block the NMDA-receptor associated ion channel only when it is excessively activated. Emerging from initial studies on NMDA-receptor S-nitrosylation one may envision to use this posttranslational protein modification to selectively down-regulate receptor activity. NitroMemantines, drugs that target the NO group to the NMDA-receptor are leading compounds (Stuart A. Lipton).

Modulators of cell signaling in apoptosis and injury:

Superoxide (O_2^-) is a rather unreactive radical but therefore suits as a messenger molecule. NO can act as an antagonist under physiological conditions. O_2^- combines with NO under formation of peroxynitrite, a species found to tyrosine nitrate and attenuate activity of prostacyclin synthase with consequences in vascular tone. Peroxynitrite upon reaction with excess NO generates an efficient nitrosating species. The upcoming concept proposes that nitration can be compensated on the expense of nitrosation when the flux of NO overrides that of O_2^- . Thus, relative rates of O_2^- versus NO formation act as key players in redox-regulation and thus in affecting cell demise (Volker Ullrich, Germany). Along that line intracellular processing of caspases are subjected to redox regulation (Lucio Annunziato, Naples). Glutathione depletion still allowed formation of the DISC (death-inducing signaling complex) while caspase-8 activation at the DISC was blocked. This predicts a new function of glutathione as a critical intracellular switch to control receptor mediated apoptosis and may suggest a redox-controlled protein to be involved in DISC formation/ caspase activation. Whereas glutathione depletion may attenuate caspase-8 activation one needs to consider that a continuous and long-lasting oxidative stress may induce caspase-3 activation with initiation of apoptosis and that formation of oxidized glutathione turned out to be a valid antiproliferative agent, particular in those cases where chemotherapy approaches are counteracted by pharmaco-resistance induced via intracellular glutathione increase (Albrecht Wendel, Germany). This implies that any redox-changes applied for therapeutical approaches need to be addressed with great caution.

Cellular and molecular targets for neuroimmune regulation:

Reports are accumulating to suggest that molecular and biochemical pathways of apoptosis are involved in dopaminergic cell death in Parkinson's disease and in patients suffering from HIV-associated dementia (HAD). It is becoming clear that for full functional recovery of i.e. dopaminergic neurons the combination of an anti-apoptotic together with a neurorestorative therapy is advised (Jorge B. Schultz,

Germany). Thus, attacking proapoptotic pathways only, may attenuate cell demise but generates a dysfunctional neuron. HAD, Alzheimer and Parkinson disease point to an important role for IL-1 β , most likely generated by microglia. Rat models have been developed for the characterization of the neuroprotective profile of drugs which interfere with mediators of neuroinflammation and crucial steps involved in the activation of the death program (Giacinto Bagetta, Italy). Mediators identified by this strategy are prostanoids derived from cyclooxygenase-2. These and other models such as monocular deprivation (Carlo Nucci, Italy) point to the action and cross-talk between excitotoxic-, radical- (O₂·, NO), cytokine- and prostanoid-mediators in affecting the balance between life and death.

Although we significantly increased our knowledge on the formation and action of individual messenger, we still lack sufficient and detailed insights into the cross talk of mediators to predict consequences for therapy. However, the VI workshop on apoptosis in biology and medicine which focused on the role of proinflammatory and chemotactic cytokines in normal and pathological brain helped to update current information, to exchange ideas and certainly will stimulate further experiments that will advance the field in the future.

Participants supported by ISN

Name and working place	Type of grant	arrival	departure	
1. Palin Karen (Southampton, UK)	Air tiket and full board hotel accomodation	25/5	28/5	997,53
2. Baltrons Maria (Barcelona, Spain)	Air tiket and full board hotel accomodation	25/5	29/5	787,43
3. Nucci Carlo (Rome, Italy)	Air tiket and full board hotel accomodation	25/5	29/5	877,97
4. Silvia Piccirilli (Rome, Italy)	Full board hotel accomodation	25/5	29/5	620
5. Robert Nisticò (Cosenza, Italy)	Full board hotel accomodation	25/5	29/5	620
6. Leta Aida (Rome, Italy)	Full board hotel accomodation	25/5	29/5	620
7. Viviani Barbara (Milan, Italy)	Full board hotel accomodation	25/5	29/5	620
8. Rombolà Laura (Cosenza, Italy)	Full board hotel accomodation	25/5	29/5	620
9. Bellizzi Caterina (Cosenza, Italy)	Partial hotel accomodation support	25/5	29/5	170
10. Russo Rossella (Catanzaro, Italy)	Partila hotel accomodation support	25/5	29/5	170
Total				6102,93

Meeting report

14th Biennial Meeting of the International Society for Developmental Neuroscience Sydney, 31 January – 4 February, 2002

Program Committee: Ralph Bradshaw (Chair), Perry Bartlett, Lynn Beasley, Ira Black, Ian Hendry, Caryl Hill, Kazuhiro Ikenaka, Paul Pilowsky, Michael Sendtner, Giulio Tagliatela, Phil Waite.

The meeting was held before the annual meeting of the Australian Neuroscience Society with an overlapping day between the two meetings. This provided an added benefit and attraction, both scientific and social, as the Australian Society for Neuroscience Meeting is a major neuroscience society in its own right. One of the treats at ISDN and ANS also was the large number of neuroscientists who made their first visit to down under and ISDN both. Our society benefits from this increase in participation of outstanding scientists, which was also the result of the labour of the program chair Ralph Bradshaw. There were over 300 registrants at the meeting and over 30 registered exhibitors. Sessions were well attended and in spite of great concerns following 9/11, there was broad international participation. The venue on Darling Harbour provided a spacious and modern setting in the midst of glamorous Sydney. Professor Bashir, the first woman to be appointed Governor of New South Wales, a Psychiatry Professor at the Universities of Sydney and New South Wales opened the meeting most graciously, a scientist herself, who delighted us with an opening talk such as one expects not from a successful politician but from a colleague from the laboratory down the hall. There was much interest by the National Press in the Proceedings of the meeting. Through the auspices of the International Society for Neurochemistry and the Institute for Developmental Neuroscience and Aging over 20 travel awards were given to students and young postdoctoral fellows. The ISDN is very grateful for the support it received from ISN for this meeting. There were 4 plenary lectures, 11 symposia and 4 sessions of short talks. In addition 81 posters were presented at this meeting.

ISN provided support for a symposium on Altered Stress Response Effects on Neurotransmission in the Aged Brain organised by Regino Perez-Polo who also gave his thoughts on aging-associated changes in transcriptional responses to stress.. The speakers supported by ISN were Stephen Rossner who talked on expression of the Alzheimer's disease b-secretase (BACE) during aging of APP transgenic mice with amyloid plaque pathology. Hermona Soreq talking on the stress-induced "readthrough" acetylcholinesterase variant and its cleavable c-terminal peptide exert morphogenic activities in blood and brain. Jim Joseph who discussed fruit polyphenolics and brain aging. Don Ingram talked on stress responses to caloric restriction: key to anti-aging effects on the brain.

The other symposia held at the meeting were:

The 5-HT_{2B} Receptor: A Candidate for Serotonin Mediated Morphogenesis inside and outside the Brain organised by Jean Lauder.

Neuron-Glia Signalling during Development: A view from inside and outside the Synapse organised by Vittorio Gallo.

Trophic Regulation of Synaptic Plasticity organised by Ira Black.

Neuronal migration in the developing brain organised by Kazunori Nakajima and Norbert Koenig.

Axonal Development and Regeneration at the Molecular Level organised by Lyn Beazley.

Development of olfactory system organised by Charles Greer and Brian Key

Retrograde axonal Transport and signalling organised by Ian Hendry.

Regulation of cell death organised by Ralph Bradshaw.

The roles of protease-activated receptors in neurodegeneration organised by Barry Festoff.

Regulating stem cells to produce neurons organised by Perry Bartlett.

The first two plenary lectures exemplified the scope of science presented at the meetings of the ISDN. The first was given by Lloyd Greene of the Department of Pathology, Columbia University New York who talked on Gene regulation and neural development: ATF's and beyond. Lloyd used serial analysis of gene expression (SAGE) technology to obtain a comprehensive view of gene expression associated with responses to Nerve Growth Factor (NGF). One example of these includes the transcription factor ATF5, which undergoes 26-fold down-regulation in response to long-term NGF treatment. Functional and developmental studies of this factor reveal that it may play a crucial role in the transition between dividing precursor cells and post-mitotic neurons. A second example is the transcription factor MAFK which responds to NGF as an immediate early gene and which functional studies indicate plays a significant role in neuritogenesis.

The second was by Lesley Rogers from School of Biological Sciences, University of New England, Armidale on Linking brain development and behaviour. Leslie enthralled the audience with her studies on lateralization chick behaviour. Experience-dependent development of the visual pathways of the chick begins prior to hatching. During the final stages of incubation, and under the influence of specific gene expression, the embryo is positioned within the egg so that it occludes its left eye but its right eye is exposed to light stimulation (unpatterned light passing through the shell). This lateralized stimulation leads to asymmetrical expression of *c-fos* in the Wulst region of the forebrain and to asymmetrical development of the thalamofugal visual projections. Hence light experience leads to a lateralized visual responses in chicks after hatching, shown as left-right eye differences in responding to food, conspecifics and predators.

David Kaplan from the Brain Tumor Research Centre, Montreal Neurological Institute, McGill University gave a comprehensive talk on the regulation of neuronal survival and growth by TRK receptors. Neurotrophins regulate the survival, axonal growth, and regeneration of neurons by interacting with their receptors, the Trk tyrosine kinases and the p75NTR. These two receptors can either collaborate or inhibit each others actions. He discussed recent advances identifying the signalling pathways used by these receptors in peripheral neurons, including Akt, MAPK, and

p73-induced signalling via Trk, and JNK and p53 signalling via p75NTR. Survival signals mediated by Trk primarily use the PI-3 kinase/Akt signalling pathway, with the MEK/MAPK pathway playing a secondary role. These survival pathways function to suppress apoptotic signals induced by the p75NTR, including the JNK/p53/BAX pathway.

The fourth plenary lecture was held on the overlapping and was the first plenary lecture for the Australian Neuroscience Society. It was given by Mu-ming Poo of the Division of Neurobiology, Department of Molecular and Cell Biology, University of California, Berkeley on neuronal plasticity at growth cones and synapses. He summarized recent findings on the cellular mechanisms underlying the guidance of growth cones and refinement of developing synapses. The formation of intricate neural networks in the nervous system depends on the pathfinding of axonal growth cones to reach their correct target cells as well as activity-driven refinement of neuronal connections after initial synaptic contacts have been made. He discussed the cytoplasmic events associated with the growth cone turning responses induced by gradients of extracellular guidance cues and addressed the critical role of cyclic nucleotides and calcium ions in determining the attractive and repulsive turning responses of the growth cone. The growth cone may amplify the gradient signal provided by the guidance cue, and may readjust its sensitivity toward the guidance cue as it moves up the gradient. He went on to describe how the temporal pattern of electrical activity may determine the nature of synaptic modification, how multiple converging synapses on the postsynaptic cell cooperate with or compete against one another in achieving the refinement of developing connections, and how visual experience may shape the development of receptive field properties of tectal neurons. Here, the pattern of postsynaptic elevation of calcium ions plays a critical role in setting the polarity (potentiation or depression) of synaptic modification. These studies of growth cone guidance and synaptic modifications illustrate a common theme in the cellular mechanism underlying neuronal plasticity: Environmental (epigenetic) factors set a cytoplasmic pattern of second messengers, which in turn shift the balance of antagonistic cellular signaling events, leading to alternative cellular responses.

Meeting report

9th International Symposium on Pharmacology of Cerebral Ischemia 21-24 July, Marburg, Germany

Summary

The 9th International Symposium on Pharmacology of Cerebral Ischemia was a gathering of more than 200 scientists from all over the world at the Philipps-University Marburg, Germany. Organized by Josef Kriegelstein with the support of an international advisory board the meeting was again a great success combining international contributions of outstanding scientists and clinicians in the field of ischemia and neuroscience research. In the conference basic research on mechanisms of neuron death and therapeutic strategies explored in experimental models of cerebral ischemia, as well as clinical trials of known drugs and novel agents under development were presented in 84 poster presentations and 57 oral presentations divided in 11 sessions and 2 plenary lectures. Prominent areas discussed were newly discovered mechanisms of cell death involving matrix-metalloproteinases and death signals from the nucleus to the mitochondria, neuroprotective drugs including PAF antagonist, bradykinin inhibitors, PARP-inhibitors and p53-inhibitors as well as new strategies to enhance brain recovery by Nogo-A antibodies, and stem cell or bone marrow stromal cell transplants. Moreover, a formerly unknown protein histidine phosphatase which is expressed at high levels in brain tissue was presented on the conference. Another highlight was the presentation of a clinical safety study on erythropoietin which revealed a pronounced benefit of erythropoietin in stroke patients.

Intracellular signaling after ischemia

After cerebral ischemia, glutamate-mediated excitotoxicity and the extensive production of reactive oxygen species mediate, in large part, the neurotoxic effect of an ischemic insult in the brain. However, the intracellular signaling cascades involved in ischemic neuron death have not been fully unraveled. While most neurons in the core of an ischemic infarct are damaged in a passive manner and die by necrosis due to the lack of energy and oxygen supply, neurons in the penumbra area often expose hallmarks of an active biochemical cell death cascade called apoptosis. This cascade is triggered by oxidative stress and disruption of cellular calcium homeostasis, and involves mitochondrial dysfunction, cytochrom c release and activation of caspases. As suggested by Perluigi Nicotera (MRC Toxicology Unit, Leicester, UK) ischemic neuron death may occur as a continuum of apoptosis and necrosis. In the ischemic brain tissue, neuron death may involve elements of both forms of cell death such as an early activation of caspases typical for the induction of apoptosis, which could switch to necrosis depending ATP levels in the damaged cell. Nicotera demonstrated in addition that caspase-mediated cleavage of plasma membrane calcium pumps may also lead to Ca²⁺ overload of neurons thereby inducing necrosis. Therefore, caspase inhibitors could prevent both, the initiation of an apoptotic program and necrotic neuronal death after stroke.

The activation of the poly (ADP-ribose) polymerase (PARP) due to DNA strand breaks is another apoptotic mechanism that can rapidly lead to cell death. Valina Dawson (Johns Hopkins University School of Medicine, Baltimore, USA) reported that peroxynitrite-induced DNA damage and the activation of PARP was substantially involved in ischemic neuron death. The excessive activation of PARP and the resulting depletion of cellular energy stores preceeded neuronal cell death, while pharmacological inhibition or genetic disruption of PARP provided significant protection against excitotoxic or ischemic insults. Dawson further demonstrated that apoptosis inducing factor (AIF) is a downstream effector of PARP, which translocated from mitochondria to the nucleus following N-methyl-d-aspartate (NMDA) exposure. The critical role of AIF in excitotoxic neuron death exposed AIF as an intriguing therapeutic target to improve the outcome of cerebral ischemia.

The important role of PARP in ischemic neuronal death was also exposed by contributions of Richard Traystman (Johns Hopkins University School of Medicine, Baltimore, USA) and Raymond A. Swanson (Department of Neurology, University of California at San Francisco, USA). In a mouse model of global ischemia induced by cardiac arrest Traystman and co-workers found a significant reduction of neuronal reperfusion injury in mice lacking PARP expression as compared to wildtype animals. Swanson emphasized an additional role for PARP activation in impaired glutamate uptake of astrocytes which may thereby enhance neuronal injury after brain ischemia. During ischemia, neurons release Zn^{2+} or NO which can initiate both, DNA damage and activation of PARP. In cultured astrocytes inhibition of PARP by benzamide prevented the impairment of glutamate uptake after exposure to Zn^{2+} or NO, demonstrating a pivotal role of PARP in dysfunction of astrocytes after stroke.

In addition to the intracellular death cascades in ischemic neurons, endogenous survival signaling pathways may be activated to prevent further damage to neurons after cerebral ischemia. Pak Chan (Stanford University School of Medicine, Stanford, USA) reported that Akt phosphorylation was enhanced in the cortical penumbra region of the infarct 4 h after middle cerebral artery occlusion, while it was decreased in the infarct core. Enhanced Akt phosphorylation was not observed in neurons with damaged DNA, and inhibition of Akt phosphorylation by the PI3-kinase inhibitor LY294002 further facilitated DNA damage in the ischemic brain tissue. In mice overexpressing superoxide dismutase-1 (SOD1), which exposed reduced brain damage after ischemia, phosphorylation, hence activation of Akt was increased as compared to wildtype controls. Chan concluded that enhanced phosphorylation of Akt was involved in promoting cell survival after cerebral ischemia.

Nicholas G. Bazan (Neuroscience Center of Excellence, Louisiana State University Health Sciences Center, New Orleans, USA) presented effects of new antagonists of the platelet-activated factor (PAF). PAF is a potent bioactive phospholipid involved in excitotoxic neuron death after cerebral ischemia, most likely by enhancing glutamate release in presynaptic terminals. In addition PAF-mediated excitotoxicity involved mitochondrial dysfunction as demonstrated in measurements of isolated mitochondria where PAF induced permeability transition and release of cytochrome c. Among a

new series of PAF antagonists termed LAU, the compound LAU-901 was identified as a potent neuroprotectant which protected neurons from ischemic damage by a mechanism involving the up-regulation of bcl-2. Moreover, LAU-901 prevented the activation of Jun-kinase and NF-kappa B, suggesting that in addition to their neuroprotective effects PAF antagonists could block inflammatory responses contributing to the infarct development after cerebral ischemia. In a model of transient middle cerebral artery occlusion in mice PAF antagonists protected brain tissue against ischemic damage and improved functional parameters even when applied 2 hours after reperfusion.

In addition to well established models of neurodegeneration in cultured neurons, in slice preparations or in rodent ischemia models, synaptosome preparations have been established as a useful model to study the mechanisms of neuron cell death. After cerebral ischemia and in various neurodegenerative diseases including Alzheimer's disease and Parkinson's disease synaptic terminals at dendrites and axons are the main structures exposed to various death-inducing stress factors. Mark P. Mattson (Laboratories of Neurosciences, National Institute on Aging, Baltimore, USA) demonstrated that after exposure to apoptotic stimuli the most pivotal mechanisms involved in programmed cell death, such as up-regulation of prostate apoptosis response factor-4 (Par-4) and activation of the tumor suppressor p53 were also regulated in synaptosomal preparations which lack the nucleus. In particular, Mattson presented data demonstrating the existence of mRNA of Par-4 in synaptosomes which allowed the post-transcriptional regulation of this pro-apoptotic factor by various apoptotic stimuli including staurosporine, and oxidative stress induced by iron-ions or amyloid-beta peptide. In synaptosomal preparations and in ischemic brain tissue Par-4 was rapidly up-regulated and induced the activation of a pro-apoptotic cascade involving the activation of caspase-8 followed by mitochondrial dysfunction and activation of caspases. Antisense oligonucleotides suppressed Par-4 up-regulation and protected brain tissue from ischemic damage.

Diazoxide, an activator ATP-dependent K⁺-channels acts as a vasodilator and has been established in the treatment of hypertensive crisis. As now shown by Mattson diazoxide exhibited a pronounced cerebroprotective effect in a model of permanent cerebral ischemia in mice at doses that did neither effect mean arterial blood pressure nor cerebral blood flow. Diazoxide increased Bcl-2 levels and inhibited the association of Bax with mitochondria in cultured neurons exposed to apoptotic insults. Moreover, diazoxide depolarized mitochondria and prevented cytochrome c release, suggesting that activation of mitochondrial ATP-sensitive potassium channels stabilized mitochondrial function thereby protecting neurons from apoptotic insults.

Mitochondrial dysfunction is a critical component in ischemic neuron death and treatments that preserve mitochondrial function are neuroprotective in models of cerebral ischemia. As suggested by Laura Dugan (Washington University School of Medicine, St. Louis, USA) mitochondrial uncoupling proteins may play a crucial role in modifying mitochondrial function and mitochondrial generation of reactive oxygen species (ROS). According to Dugan, the uncoupling protein UCP-5 was decreased in the ischemic penumbra after middle cerebral artery occlusion, which could increase mitochondrial ROS production. By contrast, UCP-5 expression was

enhanced in preconditioned cortical tissue most likely providing more resistance to following ischemic insults. Overall the data suggested a protective role for mitochondrial uncoupling proteins in neurons.

The up-regulation of chaperones such as heat shock protein-70 (Hsp-70) may be another promising strategy to prevent ischemic brain damage. Frank R. Sharp (Department of Neurology, University of Cincinnati, USA) demonstrated that geldanamycin, a benzoquinone ansamycin which binds Hsp-90 protected brain tissue in a model of transient cerebral ischemia in rats when applied at concentrations of 1 µg/kg into the lateral cerebral ventricles 24 h before ischemia. This protective effect was accompanied by the induction of Hsp-70 in neurons and Hsp-25 in glia and endothelial cells throughout the brain. Transfection of brain tissue with a TAT-linked vector containing Hsp-70 exerted similar protective effects suggesting that the up-regulation of Hsp-70 played a pivotal role in cerebroprotection by geldanamycin. Moreover, activation of NF-kappa B was inhibited after Hsp-70 overexpression indicating a potential for Hsp-70 to suppress detrimental inflammatory responses after stroke. The studies by Sharp and co-workers pointed at Hsp-70 as a promising target for the therapy of stroke as it ideally combines neuroprotective potential and suppression of immune reactions in ischemic brain tissue.

Protein histidine phosphatase:

Susanne Klumpp (Department of Pharmaceutical Chemistry, Wilhelms-University of Münster, Germany) reported about the expression of a new protein histidine phosphatase (PHP) in brain tissue. Klumpp emphasized that phosphorylation and dephosphorylation is a major principle of protein regulation in the cell. So far, only phosphorylation at tyrosine or serine/threonine residues regulated by the corresponding kinases and phosphatases were identified in eucaryotic cells. Therefore, PHP represents a new class of phosphatases which is ubiquitously expressed in vertebrates and affects phosphorylation at nitrogen-residues as in arginine or histidine. Klumpp demonstrated the high expression of PHP in various areas of the rat brain, and already identified the ATP-citrate-lyase as one substrate of the new phosphatase. Interestingly, PHP was exclusively expressed in the nervous system in *Caenorhabditis elegans*, suggesting that PHP is a highly conserved protein phosphatase with an important, yet unknown function in neurons.

Stem cell therapy:

The injection of exogenous neuronal progenitor cells or other pluripotent stem cells into ischemic brain tissue could be a future strategy for the treatment of stroke. Michael Chopp (Department of Neurology, Henry Ford Health Sciences Center, Detroit, USA) presented the beneficial effects of bone marrow stromal cells implanted up to 1 d after an ischemic insult in mice. According to his data these cells provided a protective environment of growth factors and gave rise to new brain cells including neurons and glial cells. Chopp introduced NO-donors such as SNAP as a class of substances that enhanced the percentage of neurons generated from bone marrow stromal cells in ischemic brain tissue. While enhanced NO levels may be contraproductive within the first 24 h after ischemia, NO-donors applied 1 d after

onset of ischemia significantly improved the recovery of brain function. Similar effects on functional outcome after ischemic brain damage were obtained with the phosphodiesterase type 5 inhibitor sildenafil (Viagra™), which mimicked the beneficial effects of NO-donors by increasing intracellular cGMP levels. In addition, sildenafil given at doses of 2 mg/kg/day induced neurogenesis and angiogenesis in the ischemic brain. Chopp's data suggested that a combination of bone marrow stromal cells with NO-donors or sildenafil did not affect the total area of brain damage but enhanced endogenous repair mechanisms involving neurogenesis, thereby enhancing neuronal plasticity and improving brain function.

Osamu Honmou (Department of Neurosurgery, Sapporo Medical University School of Medicine, Japan) assessed the value of stem cells obtained from either brain tissue, bone marrow or embryonic tissue which all could be applied for the replacement of injured brain tissue. He demonstrated that from the different sources all major kinds of brain cells, i.e. oligodendrocytes, astroglia and neurons could be derived, and these new brain cells appeared to be functionally active. When applied into the ischemic brain, neuronal stem cells obtained from adult brain tissue migrated into the lesioned area and differentiated into neurons and glial cells. Concomitantly, the application of neuronal stem cells resulted in smaller infarctions and functional improvement. Similar effects were obtained after application of embryonic stem cells. In addition to the effects observed with neuronal stem cells, embryonic cells may also contribute to angiogenesis after stroke. A major disadvantage of embryonic stem cells was the generation of teratoma in at least 20% of the animals which indicating a high risk in using such tissue for transplantation. Bone marrow stem cells appeared to be safe but may give rise to a large variety of different cells which are normally not found in brain tissue. Overall, Osamu Honmu judged bone marrow cells as the best choice so far for stem cell therapy as these cells hold a sufficient potential for the differentiation into brain cells, they are easier accessible than brain stem cells and they appeared safer in comparison to embryonic stem cells. Moreover, due to ethical reasons the use of embryonic stem cells is still restricted in most countries.

Recombinant tissue plasminogen activator

Recombinant tissue plasminogen activator (rtPA) is the only available therapy for thromboembolic stroke. However, side effects such as haemorrhagic insults and neurotoxic effects of rtPA may counteract the beneficial effect of thrombolysis in stroke patients. Therefore, the time window of administration (intravenous within 3 h and intraarterial within 6 h after onset of symptoms) and the application protocol should be further improved as suggested by presentations of Tobias Back (Department of Neurology, Philipps-University Marburg, Germany) and Alastair M Buchan (Department of Clinical Neurosciences, University of Calgary, Canada). Back presented a study where he compared early (1.5 h) versus late (3.5 h) thrombolytic treatment with rtPA in a thromboembolic model of middle cerebral artery occlusion in rats. Successful reperfusion was observed in 54% and 64% of the animals after early and late rtPA treatment, respectively. Animals with successful early or delayed reperfusion exhibited a similar reduction of infarct volume and a 3-fold reduction of

periinfarct depolarisations (PID). It was concluded that in addition to reperfusion the reduction of PID contributed to the protective effect of rtPA. However, in successfully reperfused rats the percentage of scattered brain injury, most likely induced by floating small thrombi, was increased.

In stroke patients, neurotoxic effects of rtPA became evident as some patients treated within the proposed time windows were not profiting from the treatment but exposed seizures and exacerbation of the infarct as demonstrated by Buchan. Neurotoxic effects of rtPA may involve propagation of postischemic excitotoxicity, generation of free radicals and breakdown of blood-brain-barrier followed by exacerbated inflammatory processes. Data obtained from patients and experimental models suggested that such toxic effects appeared to be enhanced when rtPA reached the 'wrong side of the thrombus', ie the ischemic tissue distal of the occlusion. According to Buchan rtPA exacerbated the infarction if reperfusion was not achieved within 1 to 2 h, if rtPA was applied by a catheter through the thromboembolic clot to the distal ischemic side, or if the initial ischemic area was very big. In such cases he suggested to stop the treatment immediately to prevent the following exacerbation of the damage. Buchan also listed drugs such as NMDA-antagonists, proteasome inhibitors, atenolol, matrix-metalloproteinase-inhibitors, radical scavengers and albumin which may be combined with rtPA to further enhance therapeutic benefit in stroke patients.

Plenary lectures

The plenary lectures held by Bo K. Siesjö (University of Lund, Sweden) on pharmacology of post-treatment in cerebral ischemia and Christoph Wiessner (Novartis, Basel, Switzerland) on Nogo-A antibody therapy after experimental ischemia reflected the two major approaches for the treatment of stroke presented in the conference: While the overview given by Bo Siesjö introduced possible neuroprotective strategies with the aim to reduce ischemic brain damage, Wiessner pointed at a possible strategy to enhance neuronal plasticity thereby promoting functional recovery of the injured brain.

Siesjö reviewed the major mechanisms of ischemic neuron death which include increased oxidative stress, disruption of cellular calcium homeostasis and activation of a death program called apoptosis. Neuronal apoptosis involves mitochondrial ion permeability changes, cytochrome c release and activation of caspases. An important regulatory step in apoptosis occurs at mitochondrial membranes where members of the Bcl-2 family of proteins either promote (Bax, Bad) or prevent (Bcl-2, Bcl-xl) membrane permeability transition. Dephosphorylation of the pro-apoptotic Bcl-2 family member Bad by Ca^{2+} -regulated protein phosphatase-2b/calcineurin is a major event in ischemic neuron death. Therefore, inhibitors of calcineurin such as the immunosuppressant FK-506 showed protective effects in models of cerebral ischemia, acting upstream of mitochondrial damage. Mitochondrial protection was also found with another immunosuppressant, cyclosporin A, which prevented neuron death after cerebral ischemia even more effectively than FK506. This superior effect of cyclosporine A may reflect its blockade of the mitochondrial permeability transition pore and the preservation of protein kinase-B/Akt-levels after ischemia. Siesjö

further presented data on protective effects of radical scavengers such as the spin trap compound alpha-phenyl-N-tert-butyl nitron (PBN), which reduced brain damage even if applied 1 to 2 h after ischemia.

Christoph Wiessner reported about the enhanced functional recovery of rats treated with a Nogo-A antibodies in different models of cerebral ischemia. Nogo-A is expressed in oligodendrocytes building the myelin in the central nervous system (CNS) and prevents axonal outgrowth. Thus, recovery of brain function after stroke may be partially blocked by Nogo-A which prevents the recovery of axons, thereby inhibiting reorganization of neuronal communication and plasticity. In a model of photothrombotic stroke in normotensive rats and in a model of distal middle cerebral artery occlusion in hypertensive rats new monoclonal mouse anti-rat IgG antibodies against recombinant Nogo-A (7B12) were applied intracerebroventricularly for 2 weeks starting the treatment 1 d after onset of ischemia. In both models Nogo-A antibody treatment did not affect infarct size measured 2 weeks after ischemia but enhanced functional outcome in the treated groups. The functional improvement reached significance 5 to 6 weeks after ischemia and correlated with enhanced plasticity as evaluated by determination of midline-crossing of corticospinal tract neurons. The results presented by Wiessner suggested that Nogo-A neutralization could contribute to functional recovery of brain function after acute focal brain injury with a prolonged time-to-treatment window.

Ischemic tolerance

The phenomenon of (ischemic) preconditioning describes the activation of endogenous survival signaling by a sublethal ischemic period which provides brain tolerance to a following prolonged ischemic event. Such preconditioning requires transcriptional activity and new synthesis of pro-survival proteins. Roger P. Simon (Robert S. Dow Neurobiology Laboratories, Legacy Research, Portland, Oregon) described the involvement of CREB in a model of ischemic preconditioning in mixed neuronal-glia cultures. After a short period of oxygen glucose deprivation CREB phosphorylation was mediated by multiple mechanisms including protein kinase A and calmodulin kinase. Upon activation CREB enhanced the synthesis of bcl-2, an antiapoptotic protein that may account for the protective effect of ischemic preconditioning.

Catherine Heurteaux (Institut de Pharmacologie Moléculaire et Cellulaire, Centre National de la Recherche Scientifique, Valbonne, France) reported that activation of NF-kappa B in neurons was involved in preconditioning by 3 min of ischemia, a single dose of 5 mg/kg kainic acid or 500 nmol of linolenic acid which all induced a 3-day window of protection against a following severe ischemic (6 min) or epileptic insult. The results presented by Heurteaux suggested that polyunsaturated fatty acids (PUFA) such as linolenic acid provide a new therapeutic strategy to induce tolerance against ischemic and epileptic brain damage.

Microarray analysis of gene regulation after ischemia

While most contributions in the meeting reported on the investigation of a few factors involved in defined signaling pathways after ischemic brain damage, others attempted

to describe more broadly the regulation of multiple genes in ischemic neuronal death or after ischemic preconditioning by using microarray techniques. Rainald Schmidt-Kastner (University of Miami School of Medicine, Miami, USA) reported on DNA microarray studies of differential gene expression after cerebral ischemia, and Mary P. Stenzel-Poore (Department of Molecular Microbiology and Immunology, Oregon Health Sciences University, Portland, Oregon) and Tihomir P. Obrenovitch (Department of Pharmacology, University of Bradford, UK) also analysed gene expression in models of ischemic tolerance. Stenzel-Poore described profound differences in the regulation of genes after cerebral ischemia in preconditioned brain tissue vs. ischemic tissue without preconditioning. According to her data, preconditioning caused a change in the response of ischemia-induced gene regulation towards a general decrease in transcriptional activity resulting in a reduced expression of metabolic enzymes and other factors involved in cell cycling and apoptosis. In another model of ischemic tolerance using repetitive cortical spreading depression (CSD) as a defined preconditioning stimulus Obrenovitch and co-workers found about 180 genes that had their expression changed by 100 fold or higher within 6 h after CSD. Significant CSD-induced changes in AMPA receptor subunits and nicotinic receptor subunits were also confirmed at protein levels. Overall, the microarray-based analysis of altered gene expression in ischemic brain tissue is an emerging field which will provide a huge amount of data that has to be analyzed carefully in the future to identify new relevant factors and signaling pathways involved in ischemic neuron death.

Angiogenesis during ischemia

In a session on vascular reactivity and angiogenesis during ischemia, Gregory J. del Zoppo (Department of Molecular and Experimental Medicine, The Scripps Research Institute, La Jolla, California, USA) emphasized the emerging role of microvessel-associated signaling in the maturation of the ischemic infarct. Enhanced expression of factors involved in angiogenesis such as integrins, VEGF and the angiopoietin system appeared immediately after the onset of ischemia suggesting a potential for angiopoiesis in ischemic brain tissue. Del Zoppo demonstrated that indeed new vessels were formed after focal cerebral ischemia which most likely hold a beneficial potential for the evolving infarct.

In the same session, Chung Y. Hsu (Center for the Study of Nervous System Injury and Department of Neurology, Washington University School of Medicine, St. Louis, USA) demonstrated evidence for angiogenesis in a model of focal cerebral ischemia in rats. He showed that post-ischemic angiogenesis as a compensation for the metabolic demand of the ischemic tissue evolved up to 2 weeks after the insult. Neovascularization in the cortex was accompanied by enhanced expression of angiogenic factors such as bFGF, VEGF, tie-1/2 and AngPo1/2. In addition, angiostatic factors such as thrombospondin-1/2 were also upregulated, suggesting a balance of angiogenic and angiostatic signaling in ischemic brain tissue.

Matrix metalloproteinases

Contributions by Eng H Lo (Neuroprotection Research Laboratory, Harvard Medical School, Charlestown, USA) and Stuart A Lipton (Center for Neuroscience and Aging, Apoptosis and Cell Death Research Program, Burnham Institute, La Jolla, USA)

suggested matrix-metalloproteinases as an important target for therapeutic strategies in stroke treatment. Lipton demonstrated that enhanced NO levels generated by Ca²⁺-activated nNOS after ischemic insults led to an activation of MMP-2 and MMP-9. Such an activation is initially induced by the reversible binding of NO to cysteine residues in MMPs. NO binding to MMPs then matures and becomes irreversible, resulting in the prolonged, fatal activation of MMPs. The following MMP-induced apoptosis is most likely comparable to anoikis, a form of programmed cell death induced by the disruption of the extracellular matrix which results in a deprivation of the cell from matrix-dependent survival signaling. The importance of MMPs in ischemic cell death was shown by Lo who provided evidence for a cerebroprotective effect of the broad spectrum MMP-inhibitor BB04. Furthermore, Lo demonstrated that MMP-9 played a pivotal role in ischemia-induced brain damage. In MMP-9 knock-out mice but not in MMP-2 knock-outs brain edema and infarct size were reduced after cerebral ischemia as compared to controls. In addition, Lo presented data suggesting that toxic effects of rtPA may involve up-regulation and activation of MMP in astrocytes, thereby promoting the disruption of the blood-brain-barrier and ischemic neuron death. Another involvement of MMPs in neuronal death was reported by Gary A Rosenberg (Department of Neurology, University of New Mexico Health Sciences Center, Albuquerque, USA) who demonstrated that the up-regulation of tissue inhibitor of metalloproteinase-3 (TIMP-3) may be involved in the conductance of programmed cell death. In a model of doxorubicin-induced cell death TIMP-3 prevented MMP cleavage of Fas-receptors which were upregulated and translocated to the cell membrane in response to the apoptotic stimulus.

Clinical trials

In his overview on the difficulties in translation of experimental findings on neuroprotectants to stroke trials John R Marler (National Institute of Neurological Disorders and Stroke, Bethesda, USA) who is involved in the design of clinical stroke trials presented a large list of compounds that demonstrated neuroprotective effects in experimental models of cerebral ischemia but failed in clinical trials. Marler emphasized that a possible reason for these failures was the poor design of clinical studies, in particular the time window of treatment after stroke, but he also pointed at possible flaws in the design of experimental studies. He asked to translate the rules of clinical studies to experimental models, which would mean the performance of randomized, blinded studies of potential neuroprotective drugs, and proper documentation, intention-to-treat protocols and publication of failed approaches. Moreover, he suggested preclinical testing of test compounds in different *in vivo*-models of neurodegeneration, and testing of different routes and timepoints of application. In addition, planning of clinical trials requires data on stability, compatibility, pharmacokinetics and side effects of the drugs, as well as gender differences in the response to a potential neuroprotectant. Marler questioned the design of experimental studies using single-dose treatments of a specific drug and proposed the combination of different neuroprotectants or using compounds that provide more than one mechanism of protection. In particular, combination of the well established rtPA with neuroprotectants was suggested as a basis for the design of future successful clinical studies.

The role of cytokines such as interleukin-1-beta (IL-1 β) in the inflammatory response which significantly contributes to the development of the ischemic infarct has been well established as emphasized by Nancy J. Rothwell, Herve Boutin (School of Biological Sciences, University of Manchester, UK) and Pippa Tyrell (University Department of Stroke Center Hope Hospital, Salford, UK). Previous work by Nancy Rothwell and co-workers showed that the IL-1 β antagonist IL-1ra applied after onset of ischemia reduced brain damage up to 70% in animal models of stroke. Tyrell reported on an ongoing stroke trial of IL-1ra which is not yet completed.

Hannelore Ehrenreich (Max-Planck-Institute for Experimental Medicine, Göttingen, Germany) reported about positive results of a clinical safety study on the effects of erythropoietin in 40 stroke patients. Erythropoietin has been well established as a safe drug widely used in the treatment of anemia. Previous experimental studies revealed profound neuroprotective effects of erythropoietin *in vitro* and in animal models of ischemia. The presented clinical study included patients under the age of 80 with a MRI-confirmed stroke in middle cerebral artery territory and within 8 h after the onset of the symptoms. Patients receiving erythropoietin infusions of 3333 units on 3 consecutive days after stroke significantly profited from the treatment as confirmed by MRI, neurological scoring and laboratory parameters such as S100-beta. These encouraging results could provide a new treatment of acute stroke and will be the basis of a larger follow-up study with a similar design to confirm the data obtained from this initial safety study.

Neuroprotection

Albumin provides a promising potential for the treatment of acute stroke as emphasized by Myron D Ginsberg (Cerebral Vascular Disease Research Center, Department of Neurology, University of Miami School of Medicine, USA) who presented data on the cerebroprotective effect of albumin in various experimental models of cerebral ischemia. Albumin reduces brain edema and infarct size also if applied several hours after the onset of ischemia. Cerebroprotection by albumin after ischemia is most likely mediated by various mechanisms including amelioration of microvascular perfusion and by antioxidant properties of the protein. Ginsberg also provided data showing that after albumin treatment the content of polyunsaturated fatty acids (PUFA) in the ischemic brain tissue was significantly increased. Such an increase in PUFA may provide further radical scavenging capacities, thereby contributing to the cerebroprotective effect of albumin.

A new method for the delivery of neuroprotective proteins into brain tissue *in vivo* was presented by Jun Chen (Department of Neurology and Pittsburgh Institute for Neurodegenerative Disorders, University of Pittsburgh School of Medicine, USA). He demonstrated the construction of a functionally active BCL-xL-fusion protein which contained the protein transduction domain (PTD) of the human immunodeficiency protein TAT. This fusion protein was successfully transduced into cultured neurons which were then protected against staurosporine-induced apoptosis. Excitingly, the fusion protein was also transducing into various brain regions after intraperitoneal injection, and potently protected mouse brain tissue from ischemic damage in a dose-

dependent manner, even if the protein was administered up to 45 min after ischemia. This method of transduction of brain tissue with fusion proteins could be used in the future treatment of neurodegenerative disorders to transport biologically active neuroprotective proteins into brain tissue.

Inhibitors of p53 were presented as a new therapeutic approach for the treatment of acute stroke by Carsten Culmsee (Department of Pharmacology and Toxicology, University of Marburg, Germany). He demonstrated protective effects of the p53 inhibitor pifithrin- α (PFT) and new PFT-derivates against neuronal cell death induced by DNA damage, oxygen-glucose deprivation, glutamate and amyloid-beta peptide. PFT prevented mitochondrial dysfunction and blocked activation of caspases in neurons exposed to glutamate or amyloid-beta peptide, suggesting that activation of p53 is an early event in programmed cell death. Moreover, PFT (2mg/kg) exhibited a profound protective effect in different models of focal cerebral ischemia in mice, reducing the infarct size to 50% of control levels. Cerebro-protective effects of PFT were also observed when the drug was applied up to 3 h after ischemia.

Carine Ali (School of Biological Sciences, University of Manchester, UK) and Yuan Zhu (Department of Pharmacology and Toxicology, University of Marburg, Germany) emphasized the cerebroprotective potential of the cytokine transforming growth factor- β 1 (TGF- β 1) in experimental models of ischemia. Ali revealed the protective role of endogenous TGF- β 1 by demonstrating that soluble TGF- β type II receptors acting as TGF- β 1 antagonists exacerbated brain damage induced by either NMDA infusion or focal ischemia. She further reported on the neuroprotective mechanism of TGF- β 1 which involved the up-regulation of plasminogen activator inhibitor-1 (PAI-1) in astrocytes in mixed hippocampal cultures. Another new mechanism involved in neuroprotection by TGF- β 1 was introduced by Yuan Zhu. She demonstrated that TGF- β 1 enhanced mitogen-activated protein kinase (MAPK) signaling resulting in the phosphorylation, hence inactivation of the proapoptotic oncogene Bad. In cultured neurons exposed to staurosporine, TGF- β 1 blocked the up-regulation and dephosphorylation of Bad by a mechanism involving MAPK. Inhibition of MAPK by UO126 blocked the neuroprotective effect of TGF- β 1 and concomitantly prevented TGF- β 1-induced phosphorylation of Bad. Similar neuro-protective effects of TGF- β 1 were observed in animals overexpressing the cytokine after adenoviral transfection of brain tissue. Transduced animals exhibited increased TGF- β 1 levels in brain tissue and enhanced phosphorylation of MAPK and Bad. Moreover, dephosphorylation and up-regulation of Bad after transient focal ischemia was significantly blocked in mice overexpressing TGF- β 1, which correlated well with the significantly reduced brain damage in these animals.

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Symposium Report

4th International Symposium on Experimental and Clinical Neurobiology

The *4th International Symposium on Experimental and Clinical Neurobiology* took place at the Academia Congress Center, Stara Lesna, Slovakia, 22-25 September 2002. The symposium was organized by the Slovak Academy of Sciences Institute of Neurobiology and the Faculty of Medicine, P. J. Safarik University, Kosice, and brought together over 80 scientists and physicians interested in neurochemistry, neuromorphology, neurosurgery and neuropharmacology. The symposium was opened by Prof. Jozef Marsala, chair of the Scientific Program Committee and by Prof. Dusan Dobrota, chair of the Czech and Slovak Neurochemical Society. The symposium focused on experimental and clinical research in the field of neurobiology. The first day of the symposium was devoted to the session "Brain and spinal cord injury", the other two days to "Stem cells in neural repair" and "Cytotoxicity versus cytoprotection in neural tissue". The scientific program of the symposium included 3 plenary lectures, 14 invited lectures and 17 oral presentations selected from proposals made by individual scientists. Forty-two posters were presented in chaired discussions based on 3-minute presentations beside posters. The first session, chaired by G. Goracci (Italy), covered the molecular basis of CNS traumatic and ischemic injury, focusing on the cellular and metabolic changes of acute brain and spinal injury and chronic neurodegenerative disorders. Leading invited speakers, G. Goracci (Italy), J. Gross (Germany), J. Mourek (Czech Republic), M. Réthelyi (Hungary), J. Marsala (Slovak Republic), J. Strosznajder (Poland), T. Kristian (USA), M. Schwartz (Israel) and other researchers summarized the most recent advances from the biochemical and morphological points of view, combining to create a fresh understanding of neurodegenerative disorders. The second session, chaired by M. Marsala, USA, summarized the latest findings on stem cells and on the therapeutic potential of implanted neuronal progenitor cells. Leading invited experts, M. Marsala (USA), T. Zigova (USA), N. Katsube (Japan) and other researchers covered several key points regarding developmental mechanisms as a basis for neural plasticity, cell signaling in neuronal damage and apoptosis, stem cells as a possible source for neurotransplantation, and transgene expression in regeneration and therapeutic purposes. The final session, chaired by S. Stolc (Slovak Republic), dealt with approaches to the treatment of specific neurological diseases. Well-known invited speakers M. Bentivoglio (Italy) and D. Jezová (Slovak Republic) and other leading researchers covered several key points regarding the pharmacological influence of neuronal damage caused by oxidative stress in normal cells, pathology of inflammatory signaling in CNS, protection against neurodegenerative diseases and targeting of CNS tumoral cells by proliferation-inhibiting agents (mechanisms, methods, drug delivery and transport, vaccines).

The experimental studies presented by well-known speakers were most stimulating and resulted in fruitful, in-depth discussions with clinical workers attending the symposium. In this sense the symposium was helpful in bridging the gap between

experimental and clinical neuroscience, and provided an inspiring forum for the exchange of views concerning the latest research trends and developments in the field of neuroscience. In addition, a number of new contacts between experimental and clinical scientists as well as stimulating proposals for collaboration were made during the symposium. Abstracts submitted and presented in the symposium booklet will be published in January 2003 in *Physiological Research*, and 28 lectures will appear as individual chapters in the book "New Trends in Neuroscience Research and Therapeutic Implications".

Eighty-six scientists from 12 different countries attended the symposium. Of these 29 were PhD students and postdoctoral fellows who were supported by the *ad hoc* Committee nominated by the Organizers, and received special financial assistance from two organizations (ISN-CC and IBRO-CEERC) enabling their attendance. Of the eleven sponsored by ISN-CC (Nepal 1, Poland 3, Russia 2, Slovakia 3 and USA 2), 3 were aged between 35-39 years, but we assisted them because they gave lectures and could not have done so without financial support. Of the eighteen PhD students and postdoctoral fellows sponsored by IBRO-CEERC (Czech Republic 1, Poland 2, Russia 3, Slovakia 12), 3 gave lectures. The meeting organizers listed the recipients of these fellowships in the program brochure. At the meeting the organizers publicized ISN's efforts to recruit new members, and eight new people expressed interest in ISN membership.

We would like to thank ISN-CC and IBRO-CEERC for their generous support, without which the attendance of 29 young scientists at this interesting meeting would not have been possible. Another major source of financial support for the symposium was Amersham Biosciences Trading GmbH, Austria, and other sponsors who made possible the organization of a symposium that was greatly appreciated by the participants for its high scientific standard and a very warm and friendly atmosphere.

Travel grant expenses

4th International Symposium on Experimental and Clinical Neurobiology

The ISN-CC provided 5 500 USD (264 378 SKK) to support young scientists' attendance. Eleven applicants were successful. The grants covered all local symposium expenses (registration, accommodation, full board, social program). Travel expenses were divided among the participants according to the distance they had to travel to the symposium venue, and were paid to them in Slovak currency after their arrival in Stara Lesna. A copy of the amount allocated to each recipient is enclosed. Due to the fact that the symposium was held at the Academia Congress Center, the cost of accommodation for 3 participants from the Slovak Academy of Sciences was considerably reduced, with the result that 27 069.11 SKK was saved. This money was used for covering the accommodation expenses of the invited speakers.

Registration fees:	USD	SKK
Cost for 1 person	80	3 516.00
Cost for 11 persons	880	38 684.80

Traveling expenses:

Sangraula Himal, Nepal	1 300	
Strosznajder Robert, Poland	80	
Czapski Grzegorz, Poland	80	
Chalimoniuk Malgorzata, Poland	80	
Onufriev Michail, Russia	300	
Kerkeshko Gleb, Russia	300	
Sekerkova Gabriela, USA	800	
Cizkova Dasa, USA	800	
Kubovcáková Lucia, Slovakia	30	
Mravec Boris, Slovakia	30	
Babusíková Eva, Slovakia	30	
Subtotal	3 830	168 366.80

Meals (breakfast=140 SKK, lunch=180 SKK, dinner=190 SKK)

Breakfast on Monday (10x)	1 400.00	
Breakfast on Tuesday (11x)	1 540.00	
Breakfast on Wednesday (11x)	1 540.00	
Lunch on Monday, Tuesday, Wednesday, Thursday (11x4)	7 920.00	
Dinner on Monday (11x)	.2 090.00	
Subtotal		14 490.00

Accommodation:

Cost for 11 persons	15 770.00	
Accommodation expenses of invited speakers	27 067.31	

Total Expenses: **264 378.91**

The deadline for submission of travel grant applications was May 31st 2002. The Organizing Committee awarded the young scientists their travel grants on June 30th 2002 and then informed them about the amount of money available for their traveling expenses. The calculation (1 USD = 43.96 SKK) was therefore made as at this date.

Date: November 5th, 2002

*Nadezda Lukacova
Chair of the Organizing Committee*

The report of ISN symposium

ISN symposium titled as “Molecular Basis of Neuronal Signaling” was held on July 17, 2002 during a period of the 45th Annual Meeting of the Japanese Society for Neurochemistry (JSN) in Sapporo, Japan. The number of the participant was about 600 scientists, from Japan, China, USA, Australia, and Switzerland. We summarize the scientific contributions and indicate the program as follows:

2002 International Neurochemistry Symposium “Molecular Basis of Neuronal Signaling”

Chairpersons: Prof. Katsuhiko Mikoshiba (Univ. Tokyo, Japan) & Prof. Peter Dunkley (Univ. Newcastle, Australia)

Prof. Robert Malinow gave a talk on AMPA receptor trafficking during plasticity. He showed that activity driven phosphorylation of GluR4 by PKA is necessary and sufficient for delivery of receptors to synapses during early development. He argued a mechanism that mediates plasticity early in development becomes a gate for plasticity later in development.

Prof. Masayoshi Mishina gave a talk on molecular genetics of the synapse formation and function. Elucidation how the neural network is formed and modulated is essential to understand how the brain functions. To address this issue, he and his colleagues developed a novel strategy that enables visualization and manipulation of developing neurons *in vivo*, and suggested that GSK-3b regulates the arborization field and maturation of RGC axon terminals *in vivo*.

Prof. Nancy Y. Ip gave a talk on Cdk 5: A new player at the neuromuscular synapse. In the regulation of acetylcholine receptors in the neuromuscular junction, she and his colleagues identified a new player, cyclin-dependent kinase 5 (Cdk 5), which is involved in signaling at the neuromuscular junction.

Prof. Brian A. Hemmings gave a talk on Structure, regulation and function of PKB/Akt – a pleiotropic protein kinase involved in multiple signaling pathways. He beautifully showed three-dimensional structure of PKB/Akt playing several important functions in the signaling system of a variety of cells.

Prof. Susan G. Amara gave a talk on excitatory amino acid transporters. She demonstrated some of the novel aspects of neurotransmitter transporter function and the results of molecular genetic, electrophysiological and cell biological approaches aimed at defining the relationships between neurotransmitter transporter structure, substrate transport, inhibitor binding and ion permeation.

The funds were used for travel expense of three persons (\$7,000) and rental fee for a meeting room (\$500).

Yasuyuki Nomura, Ph.D.

Professor & Chair of the Organizing Committee

The 45th Annual Meeting of JSN and 2002 ISN Symposium

2002 International Neurochemistry Symposium

“Molecular Basis of Neuronal Signaling”

Chairpersons: Prof. Katsuhiko Mikoshiba (Univ. Tokyo, Japan) & Prof. Peter Dunkley (Univ. Newcastle, Australia)

1. Prof. Robert Malinow
(Cold Spring Harbor Laboratory, USA)
AMPA receptor trafficking during plasticity
2. Prof. Masayoshi Mishina
(The University of Tokyo, School of Medicine, Japan)
Molecular genetics of the synapse formation and function
3. Prof. Nancy Y. Ip
(Hong Kong Univ. Science and Technology, China)
Cdk 5: A new player at the neuromuscular synapse
4. Prof. Brian A. Hemmings
(Friedrich Miescher-Institute, Switzerland)
Structure, regulation and function of PKB/Akt – a pleiotropic protein kinase involved in multiple signaling pathways
5. Prof. Susan G. Amara
(Vollum Institute, Oregon Health Sciences University, USA)
Excitatory amino acid transporters: New insights into structure, function and regulation



*Chairpersons: Prof. Mikoshiba
and Prof. Dunkley*



Yasuyuki Nomura, Ph.D.

Professor & Chair of the Organizing Committee

The 45th Annual Meeting of JSN and 2002 ISN Symposium

Report from an ISN sponsored Symposium at the ASN Meeting, Palm Beach June 2002

The symposium, entitled “Checks and Balances in the Cholinergic Nervous System” was held at the American Society for Neurochemistry meeting in Palm Beach, Florida in June 2002. The session considered post-synaptic function and was directed to the structure-function relationships and the regulation of expression and responsiveness of cholinergic receptors and enzymes.

Dr. Palmer Taylor of the University of California, San Diego discussed the structure and function of the nicotinic acetylcholine receptor and acetylcholinesterase. He detailed various means by which the molecular basis of specificity of these two proteins could be ascertained through the use of selective toxins, surrogate soluble receptor preparations and spectroscopic methods to examine conformation.

Dr. Edson Albuquerque of the University of Maryland discussed the unusual properties of galanthamine as a cholinesterase inhibitor, but more importantly, as an allosteric modulator of brain nicotinic receptors. He also presented some intriguing results how an indole by product, kynurenic acid, can modify nicotinic receptor function.

Dr. Jon Lindstrom of the University of Pennsylvania discussed the various subunit subunit combinations that give rise to functional receptors in neurons, and in particular, how the alpha5 subunit can influence expression and functional parameters when it combines with other permutations of alpha and beta subunits to form a pentameric receptor.

Dr. Hermona Soreq of Hebrew University in Jerusalem, ISRAEL, presented intriguing results on how stress responses can enhance the expression of various molecular forms of acetylcholinesterase and how soluble or “read through” forms, when expressed in abundance, can lead to deleterious responses in the motor system and the CNS. She was also able to demonstrate directly how expression can influence motor parameters.

Report of expenditures

Edson Albuquerque	\$ 894.09
Jon Lindstrom	\$ 492.96
Hermona Soreq	\$2,355.00
Palmer Taylor	894.15
Total expenditures	\$4,636.20

Neurochemistry Winter Conference, Sölden, Austria, April 6-11, 2002

The EuroConference/Neurochemistry Winter Conference on „Modeling addiction”, held on 6-11 April 2002 in Soelden, Austria*, explored the question of how closely various experimental approaches, ranging from the molecular biological to the human behavioral level, reflect human patterns of drug abuse and dependence and if these various approaches constitute suitable predictive or homologous models to test novel pharmacotherapies. Approx. 100 participants enjoyed the relaxed setting and the high level science, which was made possible by financial contributions of the European Commission, NIDA and ISN.

Behavioral experiments remain the mainstay in addiction research

Interestingly, the enormous spread of gene targeting and other molecular biological methods has lent new credence to a well-established experimental discipline, i.e., behavioral research. James H. Woods (University of Michigan, Ann Arbor, USA) provided an excellent overview of how operant conditioning paradigms, especially drug self-injection studies in nonhuman primates, have again and again proved relevant for human drug dependence of various types and how this experimental approach has demonstrated its predictive validity with respect to the pharmacotherapies of human opioid dependence (i.e., methadone, naltrexone, and buprenorphine) and alcohol dependence (i.e., naltrexone), as Rainer Spanagel (Zentralinstitut für Seelische Gesundheit, Mannheim, Germany) detailed in his overview of rodent behavioral assays.

Dr. Spanagel also focussed on the close ties between molecular biologists and behavioral pharmacologists, reporting that voluntary alcohol consumption in a long-term drinking model has specific effects on the NMDA/NO signalling pathway (e.g., alterations in NMDA receptor subunit composition).

Rafael Maldonado (University of Barcelona, Spain) used the example of nicotine-induced behavior in cannabinoid CB1 receptor knockout mice to demonstrate how intense and complicated systems-cross-talk can be – and showed that drug reinforcement is much more than simple activation of the mesolimbic dopamine system: Nicotine-induced hypolocomotion and antinociception were enhanced in CB1 knockout mice, the moderate reinforcing effects produced by nicotine in the conditioned place preference paradigm were completely blocked in CB1 knockout mice, whereas the severity of mecamylamine-precipitated nicotine withdrawal was similar in wild-type and CB1 knockout mice. Overall, a rather complex picture that should warn us not to oversimplify things, especially not in the treatment of substance abuse and dependence.

This impression was deepened after the contribution of Lars Terenius (Karolinska Institutet, Stockholm, Sweden) who used the endogenous opioid systems to illustrate the degeneracy of the reward system, i.e., its ability to serve the same function through a number of different elements: Of the four endogenous opioid peptide precursor genes, proopiomelanocortin (POMC) with beta-endorphin (and

ACTH), proenkephalin, prodynorphin and pronociceptin give rise to peptides of different behavioral quality, beta-endorphins and the enkephalins being robust reinforcers acting through mu opioid receptors while, on the other hand, dynorphins are – if anything – aversive, and nociceptin behaviorally neutral.

Werner J. Schmidt (University of Tuebingen, Germany) summarized experimental evidence on the glutamatergic involvement in drug seeking and drug taking. He showed that glutamatergic neurons (from the prefrontal and orbitofrontal cortex as well as those from the amygdala and the hippocampus to the ventral tegmentum and to the nucleus accumbens) are the main constituents of the brain reward system, both in the mediation of „primary” (i.e., unconditioned) reward (as reflected in its neurochemical correlate, dopamine overflow in the nucleus accumbens upon the first administration of a drug of abuse) as well as in the mediation of conditioned approach to drug-associated stimuli. These neurons, in connection with the cortical representation of the “addiction memory”, are also thought to mediate cue/context-induced relapse.

Hari Manev (University of Illinois, Chicago, USA) showed to an amazed audience to what species behavioral experiments can be carried to: Dr. Manev’s group uses the common fruit fly, *Drosophila*, to study the effects of selective gene silencing, i.e., genetic knockout in the adult animal. Confronted with double-stranded RNA (dsRNA, which can be injected directly into the *Drosophila*’s abdomen), eukaryotic cells respond by destroying this dsRNA and all the mRNA that share sequence with the double strand

Impulsivity and drug taking

Harriet deWit (University of Chicago, USA), whose wide-ranging background as an animal researcher serves as the basis for her well-known human behavioral research, demonstrated that the same behavioral experiments can be performed – with essentially identical results – in laboratory animals and humans. Dr. deWit’s work reflects the increased attention that impulsivity, i.e., the inability to inhibit or control inappropriate behaviors, as an evolving major determinant of human drug abuse and dependence receives from the research community.

Increasing focus on drug-associated stimuli

Another major factor in human drug abuse and dependence that has received more and more attention from the research community is the role that previously neutral drug-associated stimuli play in drug seeking and drug taking. Barry J. Everitt (Cambridge University, UK), one of the pioneers in this field, emphasized that different forms of associative learning have an impact on addiction. In particular, drug-associated environmental stimuli can evoke powerful drug craving and precipitate relapse to a drug-taking habit. This process can be modeled in rats, which show cue-controlled drug-seeking for prolonged periods of time. Cocaine-seeking measured in this way depends upon interactions between the basolateral amygdala and the nucleus accumbens core and is correlated with both nucleus accumbens and dorsal striatal dopamine under different conditions. He pointed out that functional imaging studies in human addicts exposed to cocaine cues that induce craving also reveal activation of the amygdala and other limbic cortical structures that mediate

emotional learning. Dopamine D3 receptors have a restricted central distribution (amygdala, nucleus accumbens and mesolimbic dopamine neurons). Everitt showed that drugs acting at the D3 receptor and thereby decreasing dopamine transmission in the mesolimbic dopamine system greatly diminish cue-controlled cocaine-seeking, but these drugs have no intrinsic reward – or cocaine reward-altering effects. In addition, the GABA-B receptor agonist baclofen, which profoundly inhibits mesolimbic dopamine neurons, also dose-dependently reduced cocaine-seeking. Both D3 and GABA-B drugs are in clinical trial.

The ecstasy of power: Neuroimaging and behavioral research

Michael A. Nader (Wake Forest University, Winston-Salem, USA) uses functional imaging methods such as positron emission tomography (PET) on brain glucose metabolism, cerebral blood flow and dopamine receptor function to understand drug abuse, relapse, craving and vulnerability to drug abuse. Measures in humans and in animal models of cocaine abuse were described. Of particular interest to Dr. Nader and colleagues is the role of the social environment on vulnerability to drug abuse and dependence. Dr. Nader and colleagues could thus show that [18F]FCP binding potential, a measure of D2 receptor density, was increased in the basal ganglia of cynomolgous monkeys that assumed a dominant role when socially housed. The same animals had expressed much lower D2 receptor levels when singly-housed previously. In contrast, the D2 receptor density of subordinate monkeys remained low regardless of the housing conditions. Accordingly, cocaine served as a reinforcer in intravenous self-administration experiments only in subordinate monkeys but not in dominant monkeys. Being the boss apparently is ecstasy enough.

So what is the new cocaine treatment going to be?

Michael J. Kuhar's lecture (Emory University, Atlanta, USA) on the development of medications against cocaine abuse was, considering how much some of the meeting participants yearned for insider tips on hot lead compounds, the most enigmatic presentation of the meeting: Psychostimulant abuse has no specific treatment medication that is approved clinically. Based on physiologic mechanisms, there have been several ideas such as direct competitive antagonists at the dopamine transporter, substitute agonists, partial receptor agonists, and cocaine antibodies as medications. No transporter antagonists have been found, but antibodies have shown positive effects in animal models. Also, some substitute transporter agonists such as various cocaine analogue compounds synthesized by the Research Triangle Institute (RTI) are promising in preclinical tests and will probably be tested in humans. Thus, progress is being made and we eagerly await the results of the first clinical trials.

* The next Neurochemistry Winter Conference on Modeling Addiction (www.sambax.com) will take place again in Soelden, 5-10 April 2003, organized by Saria (alois.saria@uibk.ac.at) and Zernig (gerald.zernig@uibk.ac.at).

ISN small conference meeting report

2002 Summer Neuropeptide Conference

MARCO ISLAND, FLORIDA, USA, JUNE 29 – JULY 3, 2002

From the organizers:

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The 2002 Summer Neuropeptide Conference, which was held June 29-July 3, 2002 in Marco Island, Florida, USA, was attended by approximately 80 registrants from North and South America, Europe, the Middle East and Asia, including the countries of Brazil, Canada, France, Germany, India, Israel, Italy, Taiwan, the United Kingdom and the USA. This twelfth annual meeting presented the latest discoveries in basic and clinical neuropeptide research. The meeting is organized independently by neuropeptide researchers from universities, research institutes and pharmaceutical companies, and is a member of the nonprofit International Neuropeptide Society. This year's meeting in Florida provided an opportunity for neuropeptide researchers to present their findings in formal symposia, poster presentations and informal discussions. The symposium topics were neuropeptides and drug abuse, vasoactive intestinal peptide/pituitary adenylate cyclase activating polypeptide (VIP/PACAP), neuropeptides and immune responses, gastrointestinal (GI) peptides, nonpeptide antagonists, neuropeptide biotechnology, and neuropeptide polymorphisms. The keynote address was presented by Dr. John Hughes, who emphasized the sequencing of enkephalins. In addition, 17 presentations were made at the poster session. Eight predoctoral and/or postdoctoral fellows were provided travel awards to present their research at the meeting. Three of these awards were sponsored by the International Society for Neurochemistry (ISN) and were awarded to the following promising young scientists:

Juliana Carrazzone Borba (Brazil)

Littli Patnaik (India)

Andrew Grant (United Kingdom)

The symposium on neuropeptides and drug abuse was chaired by Dr. Z. Sarnyai

(Psychogenics Inc.). Dr. Sarnyai described the relationship of cocaine administration in rats and CRF receptors. Cocaine self-administration in rats is modulated by brain CRF receptors. Central CRF receptor blockade alleviates anxiety-like behavior associated with cocaine withdrawal. D. Adams (Yale Univ. Sch. Med.) demonstrated that chronic cocaine administration into rats increases melanocortin-4 receptors (MC4-R) in the striatum. Direct infusion of MC4-R antagonists into the rat nucleus accumbens blocks cocaine induced locomotor sensitization, conditioned place preference, conditioned reward and self-administration. S. Bhalla (Univ. Illinois) found that BQ123, an endothelin-A (ETA) receptor antagonist, potentiated morphine analgesia and prevented adverse effects of morphine. Dr. U. Shalev (Natl. Inst. Drug Abuse, NIH) found that food deprivation potently reinstates heroin and cocaine seeking behavior in rats and mice. Intraventricular administration of leptin, a hormone involved in energy balance and food intake, blocked this effect in heroin-trained rats. Dr. M. Picciotto (Yale Univ. Sch. Med.) found that galanin attenuates the rat place preference conditioned by peripheral administration of morphine. Acute rat treatment with morphine or naloxone decreased and increased, respectively, galanin receptor binding in the nucleus accumbens. Dr. F. Crespi (GlaxoSmithKline) demonstrated that cholecystikinin (CCK) receptor antagonists reduce spontaneous drug intake in ethanol drinking and cocaine drinking rats.

The symposium on VIP/PACAP was chaired by Dr. I. Gozes (Tel Aviv Univ.). Dr. M. Laburthe (INSERM) identified amino acids of VIP which were essential for high affinity binding to VPAC₁ and VPAC₂ receptors. By site-directed mutagenesis analysis of VPAC₁ receptors, amino acids 322 and 394 were found to be essential for coupling of the receptor to adenylyl cyclase. Dr. D. Brenneman (NIH) found that activity-dependent neurotrophic factor (ADNF) consists of a complex of 3 proteins that promote neuronal survival by stimulating protease activity. Dr. I. Gozes reported that VIP increases ADNP expression. ADNP is expressed early in development (E7.5) and homozygous ADNP-knockouts die in the uterus. Dr. N. Berman (Univ. Kansas Med. Ctr.) reported that CGRP is implicated in trigeminally mediated pain whereas estrogen modulates nociceptive or cerebrovascular responses, in part through NPY. Dr. J. Waschek (UCLA) reported that upregulation of PACAP in motor neurons after injury is due to the presence of inflammatory cells at the injury site and loss of target-derived factors. Dr. T. Moody (NIH) reported that PACAP but not VIP causes increased tyrosine phosphorylation of focal adhesion kinase and paxillin in human lung cancer cells. The signal transduction mechanisms of PACAP are more complex than those of VIP.

Dr. D. Ganea (Rutgers Univ.) chaired the session on neuropeptide immunomodulators. She found that VIP/PACAP alter the Th2/Th1 balance by acting on both macrophages and CD4 T cells. VIP and PACAP promote Th2 survival by inhibition of Fas ligand expression. Dr. E. Goetzl (Univ. California-San Francisco) characterized immune cells derived from VPAC₂ receptor transgenic animals. The survival advantage mediated by VPAC₂ receptors is greater for Th2 than Th1 cells due to increased proliferation and decreased apoptosis. Dr. J. Weinstock (Univ. Iowa) reported that interleukin (IL)12 and IL 18 but not IL10 act through a NFkB-

dependent pathway to induce expression of the NK-1 receptors on T cells. Dr. P. Rameshwar (Rutgers Univ.) found that high levels of substance P (SP) precursor peptides are present in meloproliferative diseases (MPD). Also, NK-1 receptors may be involved in the proliferation of breast cancer cells. These results suggest that VPAC₂ and NK-1 receptors are present on immune cells.

Dr. J. Pisegna (UCLA) chaired the session on gastrointestinal (GI) peptides. PAC₁ receptor activation causes phosphatidylinositol (PI) turnover and elevates cAMP in ECL cells. Deletion of 63 amino acids of the C-terminal of the PAC₁ receptors impairs PI turnover and elevation of intracellular cAMP. Dr. Y. Tache (UCLA) found that corticotropin releasing factor (CRF)₁ receptors regulate colonic defecatory and visceral pain in response to stress, whereas CRF₂ receptors regulate a gastroprotective effect and reduce visceral pain to colorectal distension. Dr. W. Chance (Univ. Cincinnati Med. Ctr.) found that glucagon-like peptide-2 (GLP-2) is growth-promoting in the proximal intestine if sufficient polyamines (putrescine and spermidine) are present. Dr. N. Bunnett (UCSF) reported that NK-1 receptors sequester β -arrestins in endosomes, depleting cytosolic pools and preventing (β -arrestin-dependent endocytosis and desensitization of the NK-3 receptor.

The keynote address was delivered by Dr. John Hughes (Univ. Cambridge). He discussed the sequencing of methionine- and leucine-enkephalin, conducted when he was at the University of Aberdeen. Also, he discussed the synthesis of nonpeptide cholecystokinin receptor antagonists when he was at the Parke-Davis Research Centre.

The session on nonpeptide receptor antagonists was chaired by Dr. J. Hughes. Dr. A. McKnight (Oxford Natural Products) described the development of NK-1 nonpeptide receptor antagonists. CP-96,345 may be useful as an anti-depressant and/or anti-emetic. Dr. S. Dax (Johnson & Johnson) described aminotetralin analogues as Y₅ receptor antagonists. Dr. C. Gerald (Synaptic Pharmaceuticals) discussed the development of ligands for orphan receptors. Also, the MCH-1 receptor antagonist SNAP 7941 was investigated as an anti-anxiety or anti-depressant agent. Dr. M. Webb (Pharmacopieia) characterized bradykinin (BK) antagonists. PS309799 binds with high affinity to B₁ receptors and antagonizes the effect of des-Arg-kallidin to cause hypotension and pro-inflammatory edema.

The symposium on peptide biotechnology was chaired by Dr. I. Gozes. Dr. R. Bohrer (Cal. Western Sch. Law) emphasized that requirements to patent neuropeptide receptor sequences are changing and require additional information about protein biological function. Also, genomics and proteomics are becoming an increasingly important research and patent area. Dr. S. McLean (Pfizer) discussed using microarray data for identifying new neuropeptide orphan receptors. Dr. A. Reitz (Johnson & Johnson) described the role of recombinant proteins such as Procrit and Remicade for therapeutic use. Dr. I. Antal (Bristol-Myers Squibb) discussed neuropeptides involved in obesity. NPY is well known for its ability to stimulate food intake in rats, whereas numerous neuropeptides such as bombesin and CCK inhibit

food intake. Recently, ghrelin was shown to be important in weight loss in humans.

The session on peptide polymorphisms was chaired by Dr. J. Quinn (Univ. Liverpool). Dr. C. Baerwald (Univ. Clinic Leipzig) identified two biallelic polymorphic sequences for CRF in rheumatoid arthritis (RA) patients. CRF promoter polymorphisms may represent new genetic markers for RA susceptibility. Dr. F. Grant (Harvard Med. Sch.) has identified over 30 polymorphisms in the vasopressin (VP) gene in familial diabetes insipidus (FDI) patients. Some of the mutations impair the ability of the kidney to concentrate urine. Dr. J. Quinn discussed the variable number of tandem repeats (VNTR) within intron 2 of the serotonin transporter gene, and the 3' untranslated region of the dopamine transporter gene. It remains to be determined if VNTR polymorphisms are associated with behavioral disorders. The application of all innovative ideas presented in this symposium to human therapy still needs to be elucidated.

Seventeen excellent poster presentations contributed to the breadth and depth of the scientific presentations. At the conference banquet, Dr. K. Seroogy (Univ. Kentucky; conference Financial Coordinator) presented certificates and monetary awards to the eight winners of Graduate Student and Postdoctoral Fellow Travel Awards. The beach location of the conference at the Marco Island Marriott combined with the recreational attractions of Marco Island enhanced the informal scientific interactions.

The venue for the Thirteenth Annual Conference was announced and will be a joint meeting of the Summer Neuropeptide Conference and the European Neuropeptide Club, to be held in Montauk, New York, USA, June 8-12, 2003 (email:IGOZES@POST.TAU.AC.IL).

SECOND WIERZBA CONFERENCE:

Glutamine, glutamate and GABA in the CNS: Transport and metabolism in health and disease

The conference was held in August 24-28, 2002 in Wierzba, a Convention Center of the Polish Academy of Sciences located in a picturesque, isolated, forest-shielded Mazurian Lakes region in Poland, 220 km north of Warsaw. Both the location and the title of the conference are likely to sound familiar to neuroscientists interested in neurotransmitter metabolism and function. A symposium under a similar title "Glutamate-Glutamine Homeostasis in the CNS: Physiological and Pathophysiological Aspects" took place in Wierzba a little more than 3 years ago, and the proceedings appeared in a special issue of *Neurochemistry International* (vol. 37, nos 1-2, 2000) co-edited by the organizers, Prof. Arne Schousboe of the Royal Danish School of Pharmacy in Copenhagen and the author of this report. The present symposium thus constitutes a step towards realizing the organizers' plans to make the Wierzba conferences evolve to a cyclic event. For a series of events to make sense, each must be not only be adequately linked to the past, but also keep pace with new developments. The past was clearly represented by the leitmotief of the program that is, the glutamate/ glutamine cycle. The new winds was manifested by a stronger than before representation of the inhibitory neurotransmitter GABA and more focus on cell membrane transport of all three amino acids, including an account of the explosion of new data on the role of glutamine transport in the Glu-GABA-Gln homeostasis. In our attempts to make the program of the symposium fit best to the state of the art we were very much helped by an Advisory Committee consisting of acknowledged experts in the field: Frode Fonnum (Kjeller), Leif Hertz (Gilmour), Jerzy Lazarewicz (Warsaw), Jon Storm-Mathisen (Oslo) and Simo Oja (Tampere). It was a particular pleasure to all of us to listen to the excellent opening lecture given by Professor Ole-Petter Ottersen, one of the pioneers in the field of immunocytochemistry of amino acids in the CNS. His presentation convincingly documented the power of the immunogold technique in visualizing mutual spatial relations between glutamate, GABA and glutamine at the ultrastructural level, but also in assessing the role of other factors involved in determining the homeostasis at the glutamate synapse, among these aquaporin 1 (AQP1), a water channel protein. selectively permeated by water driven by osmotic gradients.

The topics specifically dealt with during the symposium are best illustrated by the titles of the respective sessions, which were as follows:

- Glutamine transport at the BBB and at the cellular level
- Nitrogen shuttling between neurons and astroglia
- Glutamate transport and energy metabolism
- Glutamate transporters: Regulation of expression and pharmacological characterization
- GABA transporters: Expression patterns and pharmacological characterization
- Biosynthesis and metabolism of neurotransmitter glutamate and GABA. Aspects of compartmentation

- Amino acid transport and excitotoxicity
- Glutamine in brain pathology
- Disturbances in glutamatergic and GABAergic neurotransmission in HE
- Disturbances of glutamate and GABA transport in neurologic disorders and energy failure

It is beyond the scope of this report to provide an exhaustive coverage of the plethora of novel and intriguing data that have been communicated during the sessions. However, each presentation deserves at least a brief mention. Farrukh Chaudhry from Oslo and Stefan Broër from Canberra have described their most recent, successful attempts at cloning, and characterizing the functions of, the newly discovered cell membrane transporters that specifically mediate glutamine uptake and efflux in astrocytes and neurons. Their discoveries strongly support the notion that the glutamine-glutamate cycle is regulated by active glutamine transfer between the different compartments of the CNS. Specific, system N-mediated aspects of glutamine transport at the blood-brain and blood-CSF barriers were subject of a presentation by Richard Keep from Ann Arbor. The issue of glutamate transport has been dealt with by Baruch Kanner from Jerusalem, who dwelled on the molecular determinants for ion and substrate interactions with the different transporters, and again Farrukh Chaudhry, who provided a detailed description on the newly cloned vesicular glutamate transporters. Other speakers in the glutamate transport session included Georgi Gegelashvili from Copenhagen who covered various aspects of posttranscriptional regulation the transporters and Max Recasens from Montpellier, whose studies using an *in vitro* model emphasize the diverse functional roles of glutamate uptake in the hippocampus, including development, plasticity and stress response. With regard to GABA transport, Nathan Nelson from Tel Aviv took up the issue of the homology of the four different so far described GABA transporters (GAT1-4), and of their quite distinct mode of operation. Bente Frølund and Orla Larsson from Copenhagen reported their attempts at designing new, powerful and specific inhibitors of GABA transport. A number of reports have brought to light the power of magnetic resonance spectroscopy as a tool to examine compartmentation of glutamate and glutamine metabolism. Arne Schousboe from Copenhagen provided new insights into the role of alanine in nitrogen shuttling between neurons and glia, Rolf Gruetter from Minneapolis convincingly demonstrated the utility of NMR spectroscopy to unravel the details of cerebral glucose metabolism, and Ursula Sonnewald from Trondheim provided new data on the compartmentation of GABA and glutamate metabolism. The issue of GABA synthesis was taken up by Andreas Plaitakis from Crete, who presented evidence that minor amino acid substitution in the regulatory domain of human housekeeping (GLUD1) glutamate dehydrogenase alter the pattern of enzyme activity regulation by major cofactors, including ADP and L-leucine.

The involvement of glutamate, glutamine and GABA in brain pathology has been covered by a number of speakers in various sessions. Jerzy Lazarewicz from Warsaw and Michael Aschner from Winston-Salem discussed the mechanisms and significance of activation of different classes of glutamate receptors in homocysteine

and manganese neurotoxicity, respectively. John Phillis from Detroit emphasized the role of activation of phospholipase A2 in the ischemia-induced release of amino acid neurotransmitters, whereas Alan Hazell from Montreal demonstrated disturbances in the expression and functioning of astrocytic glutamate transporters accompanying impaired oxidative metabolism in rat brain. Frode Fonnum from Kjeller provided evidence for glutamate uptake being the target of a number of industrial toxins, including polychlorinated and polybrominated biphenyls.

Leading experts in the area of hepatic encephalopathy were invited to present their views on the roles of glutamate, GABA and glutamine in the pathomechanism of this disease. Roger Butterworth from Montreal reviewed evidence pointing to the involvement of alterations of glutamate receptors, E. Anthony Jones from Amsterdam presented arguments in favor of increased GABA-ergic transmission in liver failure, while Michael Norenberg from Miami discussed data gathered in his and other laboratories that are indicative of the induction of mitochondrial permeability transition (MPT) by ammonia. As pointed out by M. Norenberg and his collaborator K.V. Rama Rao, glutamine is largely responsible for MPT in hyperammonemic conditions. Vicente Felipo from Valencia presented evidence that ammonia-induced accumulation of glutamine is coupled to activation of nitric oxide synthase, an event downstream overactivation of NMDA receptors by ammonia. In a single presentation on the role of Gln in brain pathology unrelated to hyperammonemia, Jan Albrecht from Warsaw reported on the characteristics of glutamine transport in an astrocytoma cell line that are quite distinct from those found in cultured astrocytes or neurons.

The novelty of the 2002 conference was the junior sessions, a chance given to the Ph. D. students and just graduated scientists, to subject their achievements to an exchange of views between themselves, and with top experts in the field, in a generation gap-filling mode.

A total of 41 speakers presented their lectures during this symposium and of these, 13 were junior speakers. The speakers represented many nations: they came from Australia, Canada, Denmark, Finland, Greece, Israel, Norway, The Netherlands, Poland, Spain, Switzerland and USA. As was the case with the previous Wierzbica symposium, the proceedings of the present conference will appear in a special issue of *Neurochemistry International* in 2003.

The conference was a rewarding event not only in scientific, but also in social terms. It was held under the auspices of the Medical Research Centre of the Polish Academy of Sciences, whose newly elected Director, Professor Zbigniew Czernicki honoured the participants with an address printed in the Conference Program. Very special thanks are due to the members of the local organizing committee, all being staff members or students of the Medical Research Centre, for their endurance in coping with all the inevitable difficulties in the process of organizing the conference, and devotion to fulfill the professional and everyday needs of the participants. The key figures in this capacity were Hanna Klinowska and Wojciech Hilgier, helped by Inez

Fresko, Magdalena Zielinska, Marta Sidoryk and Marta Obara, all from the Department of Neurotoxicology, and Dorota Makarewicz from the Department of Neurochemistry. At last but not least our sincere thanks go to the Management of the Convention Center for providing competent services during the sessions and offering excellent food and accommodation.

A generous support of the International Society for Neurochemistry has made it possible to cover the costs of the participation in the symposium of all the junior speakers, and the travel expenses within Poland of all the participants.

*Jan Albrecht,
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Polish Academy of Sciences
Warsaw, Poland*

ESF Exploratory Workshop and ISN Small Conference on MYELIN STRUCTURE AND ITS ROLE IN AUTOIMMUNITY

Potenza, Italy, 5-8 June 2002

SCIENTIFIC REPORT

Overview

The ESF Exploratory Workshop and ISN Small Conference on “Myelin Structure and its Role in Autoimmunity” was held at the Giubileo Hotel, located at about 1200 m altitude and immediately adjacent to the forest of Rifreddo, near Potenza. The conference center was very pleasant, and all participants were comfortably accommodated on-site, thereby facilitating a good deal of interaction. In total, 75 registered participants attended the meeting: 55 were from Europe (40 from Italy, 7 from Germany, and 1 each from Finland, Sweden, England, Scotland, France, Spain, Switzerland, and Greece), 10 from Canada, 6 from the United States, 2 from Australia, and 1 each from Singapore and Japan. Numerous other colleagues wanted to but were unable to attend. Nonetheless, the overwhelming consensus of this research community was that it is important to decipher how myelin is made and structured in order to understand the mechanisms of assembly, function, and disruption in its physiopathology.

In accord with the stated objectives, the workshop brought together researchers whose interests were quite diverse ranging from biophysics, biochemistry, structural biology and bioinformatics, to neuroimmunology and therapy of multiple sclerosis. This diversity led to a number of discussions during the sessions, and to lively interactions during session breaks and “free time”, usually with wine or cappuccino. The sessions were highly focussed, with a good balance between basic and applied studies. This workshop was the first of its kind, and was made possible by generous financial contributions from the International Society for Neurochemistry (ISN), the Multiple Sclerosis Society of Canada, the National Multiple Sclerosis Society (U.S.A.), the Italian Society for Multiple Sclerosis, and the Standing Committee for Life and Environmental Sciences (LESC) of the European Science Foundation (ESF). In particular, the funds provided by the ISN were used to reduce the registration fees from \$500-560 to \$350-390 for 31 young scientists. This support provided incentive and opportunity for these individuals to attend and contribute to the meeting’s diversity and success. The funds provided by the ESF/LESC were instrumental, however, in providing registration and travel support for all other European participants.

Opening Lecture

– Post-Translational Modifications of MBP Suggest Novel Therapeutic Strategies.

Dr. Mario Moscarello (Hospital for Sick Children, Toronto, Canada) gave an overview of myelin basic protein (MBP), one of the most abundant proteins of the myelin sheath, and a candidate autoantigen in multiple sclerosis (MS). A Leitmotif of

MBP is its high degree of post-translational modification, leading to a set of “charge isomers” or components termed C1 to C8. A key modification of MBP associated with severity of multiple sclerosis is deimination of arginyl to citrullinyl residues by the enzyme peptidylarginine deiminase (PAD). Thus, PAD might be considered an appropriate therapeutic target. Moscarello’s group has found that the chemotherapeutic drug paclitaxel (Taxol) is a potent inhibitor of PAD activity *in vitro*. Using a transgenic, spontaneously demyelinating mouse line as a model for MS, and they have found that paclitaxel severely attenuated the disease, potentially by up-regulating the genes of several proteins involved in myelin development. Another post-translational modification of MBP, methylation of Arg107 (human sequence), is associated with healthy myelin. Thus, another therapeutic strategy for MS is to enhance the potential for methylation of MBP per se, as well as the methylation of the promoters of PAD genes to silence them. Vitamin B12 is the methyl donor in the biosynthesis of S-adenosylmethionine, and its use as an adjuvant to paclitaxel therapy led to an even greater degree of remyelination in the transgenic mice. Thus, therapies designed to modulate the post-translational modifications of MS have shown great promise in a mouse model for the disease. Clinical trials are presently underway to assess the combination therapy in humans.

Educational tutorials – Composition and structure of CNS and PNS myelin; multiple sclerosis; physical and computational techniques.

The first three sessions on the first day were educational tutorials, whose purpose was to bring participants of diverse backgrounds to a common starting level. The first session focused on the biology of myelin. Drs. GianLuigi Mancardi (Genova, Italy) and Maria Trojano (Bari, Italy) each spoke on MS and demyelinating diseases, reviewing the basic biology, epidemiology, and clinical aspects, and discussing recent advances in therapies. Unfortunately, the talk by Dr. Klaus-Armin Nave (Göttingen, Germany) on animal models of demyelination and dysmyelination had to be cancelled; Dr. Nave’s journey to Potenza was blocked by a sudden strike of airport workers, and he was unable to attend.

The second educational tutorial was a complete contrast to the first one, providing an overview of several biophysical approaches to studying myelin. Dr. Jordi Navarro (Amersham Biosciences, Spain) described mass spectrometric approaches to protein identification. In myelin, there is a low correlation between mRNA abundance and protein levels, and developing technologies such as MALDI-TOF mass spectrometry are proving invaluable for cataloguing developmental patterns of protein composition and modifications in myelin. Since mass spectrometry relies on a preliminary separation of proteins by two-dimensional gel electrophoresis, there was some discussion of this technique. In particular, an exciting development by Amersham Biosciences is isoelectric focusing strips with pH gradients of 3-12 and 9-12, which will be especially useful for separating the highly basic proteins of myelin. The product uses a new matrix that is significantly stabler than polyacrylamide; although it is not yet commercially available, it is hoped to be within the next year. Dr. Heinrich Haas (Campinas, Brazil) described the experimental reconstitution of myelin-like model systems by Langmuir and Langmuir-Blodgett techniques, and

fluorescence microscopical and scattering (X-ray and neutron) techniques to analyse the molecular packing of proteins and lipids within them. Dr. Jan Sedzik (Huddinge, Sweden) discussed the (black?) art of protein crystallization, for which he coined the term “crystallomics”. One of the major stumbling blocks of structural biology, and especially that of myelin structural biology, is our inability to get appropriate three-dimensional crystals of membrane-associated proteins. Developments in this field include the use of novel detergents to purify such proteins in lipid-bound form to retain their native structure, and novel lipid phases (e.g., cubic) to obtain crystals. Nonetheless, progress in this area remains slow and sporadic.

The third educational session had a more heterogeneous nature. Dr. Bernd Kieseier (Dusseldorf, Germany) described zymographic assays and ELISA for matrix-metalloproteinase (MMP) activity. MMPs are involved in the pathogenesis of demyelinating diseases, and reliable tests for MMPs are essential to understanding their function. Dr. Eugenia Polverini (Parma, Italy) gave an insightful and comprehensive overview of bioinformatics as it can be practiced by non-specialist scientists: sequence databases and websites containing programs for sequence alignment and structure prediction, and the theoretical backgrounds and reliabilities of commonly-used algorithms. Dr. Vladimir Brusic (Singapore) concluded with a description of data retrieval and warehousing. The tremendous amounts of sequence data being generated by molecular biologists worldwide present tremendous challenges to those who archive them, requiring new computational strategies and networking technologies.

Scientific sessions.

The remainder of the meeting was devoted to scientific sessions, organized into several themes. A summary of the topics of the presentations follows. On the one hand, a polarity can be perceived. On the other hand, there is a healthy diversity, and a synergy that must be created and facilitated by future meetings of this nature.

Cell biology, biophysics, and biochemistry – Myelin genes and proteins, and reconstituted systems.

Dr. Kazuhiro Ikenaka (Okazaki, Japan) – Development of the oligodendrocyte; Dr. Anthony Campagnoni (Los Angeles, U.S.A.) – Multiple functions of the myelin basic protein gene: roles in the immune and nervous systems; Dr. Daniel Kirschner (Boston College, U.S.A.) – Hierarchical studies on myelin P0: from gene to protein-protein and membrane-membrane interactions; Dr. Joan Boggs (Toronto, Canada) – Glycolipid-glycolipid and protein-protein interactions in myelin and involvement in transmembrane signaling; Dr. Maurizio Bifulco (Salerno, Italy) – The myelin-associated enzyme CNPase as a membrane anchor for tubulin; Dr. Robert Zand (Ann Arbor, U.S.A.) – Increased methylation and decreased phosphorylation in myelin basic protein from multiple sclerosis brain may reflect an important role in the etiology of MS; Dr. Paolo Riccio (Potenza, Italy) – MBP back home – investigations of lipid-bound MBP; Dr. Anthony Heape (Oulu, Finland) – Why is the human myelin-associated glycoprotein (MAG) so unstable, sometimes?; Dr. George Harauz (Guelph, Canada) – Structural investigations of the 18.5 kDa isoform of myelin basic

protein in reconstituted protein:protein and protein:lipid systems; Dr. Heinrich Haas (München, Germany) – Organized molecular assemblies from myelin basic protein and phospholipids at planar interfaces and in the bulk phase; Dr. Ranieri Rolandi (Genova, Italy) – Myelin basic protein-lipid complex: an atomic force microscopy study; Dr. Jan Sedzik (Huddinge, Sweden) – Crystallomics of myelin membrane proteins.

Oral investigations of young investigators.

An important outcome of the meeting was the participation of postgraduate students as well as young scientists, all of whom presented posters. Their exposure to good “frontier” science, as well as the possibility for them to meet more senior colleagues and discuss with them their research endeavours, was for many a unique opportunity and a most valuable experience. Four young investigators (all doctoral students) were selected to present their posters orally:

Giulia Carlone (Bari, Italy) – PNS protein PMP-22 (PASII) in lipid-bound form; Ian Bates (Guelph, Canada) – Structure and conformation of normal and quasi-citrullinated myelin basic protein using molecular dynamics and electron spin resonance; Corree Laule (Vancouver, Canada) – Myelin as seen from the world of MRI; and Maria Pagany (Martinsried, Germany) – Susceptibility to MOG-induced EAE is correlated with enhanced MOG expression.

Myelin breakdown – Factors influencing the formation, compaction and swelling of myelin; autoimmunity and myelin autoantigens.

Dr. Naren Banik (Charleston, U.S.A.) – Calpain upregulation coincides with the infiltration of inflammatory cells and onset of experimental allergic encephalomyelitis in Lewis rats; Dr. Grazia Liuzzi (Bari, Italy) – Interferon- γ treatment inhibits metalloproteinase activity in rat microglia cell cultures; Dr. Hans Berlet (Heidelberg, Germany) – The metal affinity of myelin basic protein: possible functional implications; Dr. Gianvito Martino (Milano, Italy) – MOG(35-55)-induced EAE in C57BL/6xSV129 FGF-II^{-/-} mice; Dr. Claude Bernard (Melbourne, Australia) – Molecular characterization of T and B cells’ responses in experimental autoimmune demyelination provoked by oligodendrocyte glycoprotein; Dr. Chris Linington (Martinsried bei München, Germany) – Milk and mimicry: modulation of self-tolerance to the myelin oligodendrocyte glycoprotein (MOG); Dr. Sandra Amor (London U.K.) – MOG is critical for progressive demyelinating disease in mice; Dr. Antonio Uccelli (Genova, Italy) – Immune responses to myelin antigens in marmoset EAE; Dr. Jean-Marie Matthieu (Lausanne, Switzerland) – Antibodies against MOG and protection by PPAR-agonists.

Autoimmunity and molecular mimicry.

Dr. Luca Massacesi (Firenze, Italy) – Immunomodulatory effects of Ifn γ and azathioprine in multiple sclerosis; Dr. Benedetta Mazzanti (Firenze, Italy) – Avidity repertoire of antigen-specific T cells; Dr. Massimo Degano (Milano, Italy) – X-ray crystallography of TCR/MHC complexes: how T cells recognize different antigens; Dr. John Matsoukas (Athens, Greece) – MBP(87-99) epitope conformation and

rational design of cyclic analogues; Dr. Graziano Pesole (Milano, Italy) – A novel algorithm to measure the probability of mimicry with self and nonself proteomes may predict immunodominant peptides; Dr. Marcella Attimonelli (Bari, Italy) – Analysis of MHC class II versus MHC class I human and microbial peptidomes; Dr. Eugenia Polverini (Parma, Italy) – Peptide propensity to ordered structure influences epitope selection by antigen presenting cells: consequences for self-nonself discrimination; Dr. Giovanni Ristori (Roma, Italy) – Self and nonself in the antigen presenting cells (PC) perspective; Dr. Mark Mamula (New Haven, U.S.A.) – Post-translational modifications of self proteins in autoimmunity; Dr. Vladimir Brusic (Singapore) – Prediction of peptide binding to MHC molecules; Dr. Marie-Paule Lefranc (Montpellier, France) – IMGT standardization and tools for the analysis of immunoglobulin and T cell receptor repertoires and 3D structures.

Assessment of Results – Outcome and future contribution to the field

The novel formula that the organizers had adopted for the meeting was highly successful in that it allowed strong and fruitful interactions amongst workers in various disciplines, namely, biophysics, neuroimmunology, bioinformatics and clinical neurology. Platform presentations were balanced to provide equal exposure to all disciplines, and the relationships and complementation amongst different fields were promoted. The scientific topics were well chosen in definite fields, and were organized in such way that structure was the first and the ultimate goal of a cycle of reports, starting from the myelin membrane and arriving to the peptides presented by the antigen presenting cells. There is no doubt that this conference will spread the seeds for future collaborations amongst individuals in different fields.

In planning a future meeting, effort will be made to facilitate the interactions between disciplines that are usually perceived to be far apart, such as biophysics and neuroimmunology. From this point of view, a second meeting to be held in 2004 or 2005 would be particularly appropriate. The International Meeting of the ISN is to be held in Austria in 2005. Thus, one option is to organize the second symposium on myelin structure as an ISN satellite meeting in 2005, which would have the added advantages of facilitating the participation of even more non-European scientists, and enlarging the spectrum of knowledge in this particular field.

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29 November 2002

Obituary

Stanislav Tucek (1932-2002)

Stanislav Tucek was born on 18 April 1932 in Pardubice and spent his boyhood with his parents at the nearby small town of Dasice. His medical studies, which began at the Charles University in Prague, continued at the Medical Institutes in Kazan and in Kharkov, and in 1957 he obtained the M.U.Dr. degree (equivalent to an M.D.). After one year as a physician at the Department of Internal Medicine of the regional hospital in Ústí nad Labem, he was appointed to a teaching post at the Department of Physiology, Medical Faculty of the Charles University in Pilsen. In 1964 he was awarded the C.Sc. degree (equivalent to a Ph.D.) and in 1970 he was offered a position in the Institute of Physiology of the Czechoslovak Academy of Sciences in Prague (now the Institute of Physiology of the Academy of Sciences of the Czech Republic) where he worked for the rest of his life.



Stan's interests in research, which began during his medical studies, encompassed the whole cholinergic field, from elucidation of the sources of the acetyl precursor groups required for acetylcholine synthesis to mechanisms of regulation of cholinergic neurotransmission. His studies on the sub-cellular distributions of all of the enzymes involved in the synthesis and degradation of acetylcholine, and the mechanisms of axonal transport of choline acetyltransferase, led to major contributions to our understanding of the mechanisms regulating the rates of acetylcholine synthesis and release in both nervous and non-nervous tissues. More recently he focussed on the effects of allosteric modulators on muscarinic receptor function, and on mechanisms for the regulation of muscarinic receptor expression.

His research career was stimulated by several extended stays in reputable laboratories abroad. In 1964 he spent 14 months with Catherine Hebb at the Agricultural Research Council Institute of Animal Physiology in Babraham, UK, where he met Ann Silver, Frode Fonnum and Victor Whittaker. A fruitful spin-off from this visit was that Catherine Hebb was able to arrange research support for Stan from the U.K. Wellcome Trust. Prevailing political conditions meant that it had to be administered from the U.K., first by Catherine, then by Ann Silver and finally by Herman Bachelard. Stan was deservedly fortunate to have this (albeit small) support during the difficult times in his country which continued until the east-west barriers disappeared. In 1969 Abel Lajtha invited him to participate with Dr. S.-C. Cheng in

investigations on acetylcholine synthesis at the New York State Institute for Neurochemistry and Drug Addiction. Further valuable collaborations were with Maurice Israël at the Salpêtrière Hospital, Paris, in 1974, with Brian Collier at the Department of Pharmacology, McGill University, in 1976, and more recently with Professor El-Fakahany, University of Minnesota. These visits created the basis for lasting international co-operation and life-long friendships, and also enabled his postgraduate students to enjoy training in the top laboratories in the field.

In 1978 he was awarded the Dr.Sc. degree (equivalent to D.Sc.) for his research, when his key textbook in the field “Acetylcholine Synthesis in Neurons” was published by Chapman and Hall (London). During his career he published more than 300 research papers, a substantial number of them in renowned international journals. His reputation in his chosen field was unparalleled: Maurice Israël observed that “Stan knew the cholinergic system in such a way that we may consider him as a Master”. His outstanding contributions to science have been recognised in the award of the Purkinje Medal of the Academy of Sciences of Czech Republic in 1997, and the dedication to him of a special issue of *Neurochemical Research*, edited by Annica Dahlström, to appear soon.

He was also heavily involved in teaching and administration in Prague. In 1973 he became Head of the Department of Neuromuscular Physiology and from 1977-1980 he acted as the Deputy Director of the Institute of Physiology. In 1980 he was appointed Head of the Department of Neurochemistry. He retired formally in 1997 but continued to act in this capacity until his death. In recognition of his educational activities he received an Honorary Professorship of Kazan Medical University in 1994 and the Quastel Visiting Professorship at the Hebrew University in Jerusalem in 1997. His contributions to the international neuroscience community involved membership of the Editorial Boards of several international journals including the *Journal of Neurochemistry*, and active participation in international scientific societies such as ISN, IBRO and FENS. Tony Turner commented that “Stan was one of the longest serving, and most dedicated, members of the editorial board of the *Journal of Neurochemistry* and was still acting in that capacity up to his untimely death”.

During the period when contacts between scientists in Eastern Europe and the rest of the World were severely restricted for political reasons, Stan worked very hard to create ways to facilitate contacts between Eastern and Western scientists, and to place many of his students in their laboratories. Ann Silver commented that despite the constraints on research in “iron-curtain” days, his laboratory in the Institute of Physiology in Prague was extremely productive. Shortly after the European Society for Neurochemistry (ESN) was established in 1976, with a key part of its remit to facilitate such contacts among neuroscientists, Stan became a very enthusiastic member of its Council and in 1984 was elected its President. In 1978, he organised an international symposium on “The Cholinergic Synapse” at the castle of Zínkovy (Western Bohemia), where many established cholinergists from the East and West were able to meet in person for the first time. In 1986 he hosted a most successful

meeting of the ESN in Prague at a time of the utmost political difficulty, especially in that the Republics of the Eastern Bloc were refusing passports to Israeli citizens. It was typical of Stan's dedication and ability to work quietly behind the scenes that the problem was overcome without fuss or public acknowledgement. Indeed it is these qualities, combined with his warm kindness, humanity and tolerance, that his friends and colleagues appreciated most. Victor Whittaker also expressed such sentiments by saying that he came to "admire his careful work, his precise ways of thinking and of expressing himself and his calm and diplomatic manner in handling tricky situations".

Abel Lajtha commented that "we think of someone (like Stan) who is soft-spoken and polite, who clearly appreciates the ideas and the background of others, who is happy to listen to and happy to help others, as a weak person, not as a leader. We often think that leaders have to be assertive, perhaps single-minded, and not appreciative of the ideas of others. Stan was of the first type, but to my mind, he was among the very best leaders in neuroscience".

During the latter period of his life, Stan's humanity was illustrated by his active participation in the work of the Foundation for Old Age and Illness, founded by his wife Dana, a geriatrician, in Prague at the beginning of the 1990s. He also helped Dana in her activities for the Sue Ryder Home in Prague.

Stan Tucek died suddenly of pneumonia on September 27. However, he had been suffering from leukaemia for several years and pneumonia was the final complication of this primary insidious disease. He successfully hid his health condition from his friends and co-workers. He did not want to be treated as an indisposed person and wished to be active in the laboratory and in civic life as long as possible. His friends and co-workers all over the world will miss him greatly, and join with us in extending our deepest sympathy to his widow Dana, his son Martin, and his daughter Lenka, in the loss of a caring and courageous man.

Vladimír Dolexal, Annica Dahlström and Herman Bachelard

20th Biennial ISN Meeting



ISN

August 21-26, 2005, Innsbruck, Austria

Destination

The 20th Biennial Meeting of the ISN will be held in conjunction with the ESN in Innsbruck, Austria. Innsbruck is a charming city, located at the very heart of Europe. At the time of this meeting, it offers a wide variety of cultural, sports and recreational activities. Pre- and post-conference-tours may include visits to renowned cultural sights in Austria, Germany and Italy, such as Vienna, Venice or Neuschwanstein Castle.

Local Organising Committee

As an old university town Innsbruck has a long-standing tradition in hosting international conferences. The Local Organising Committee represents the large neuroscience community of the Universities of Innsbruck and Vienna:

C. Bandtlow	Innsbruck
R. Fischer-Colbrie	Innsbruck
W.W. Fleischhacker	Innsbruck
B. Grubeck-Lobenstein	Innsbruck
L. Klimaschewski	Innsbruck
A. Laslop	Innsbruck
W. Poewe	Innsbruck
A. Saria	Chairman, Innsbruck
W. Sieghart	Vienna
E. Singer	Vienna
N. Singewald	Innsbruck
G. Sperk	Innsbruck
H. Winkler	Innsbruck
G. Zernig	Innsbruck

Venue

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A-6020 Innsbruck, Austria
www.congress-innsbruck.at

Congress Innsbruck, „Best congress centre 2001” – AIPC Apex Award Winner, is located next to the Old Town with its numerous restaurants, cafés and bars.

Registration Fees

To enable as many scientists as possible to attend the meeting, the LOC will keep the registration fees at a low level, i.e. at approximately € 200.- for members. Furthermore, there will be substantial reductions for students.

Accommodation

Innsbruck offers a range of accommodation which is well balanced both in terms of price and quality, from 5-star hotels to cosy guest houses. Prices are between € 44.- to € 140.- for the single room including breakfast, service and all taxes. Most hotels are within easy walking distance to the conference centre, situated right at the centre of Innsbruck. In August, Innsbruck also offers rooms in student dormitories at bargain prices.

Enquiries:

PCO Tyrol Congress

Mrs Ina Kaehler

Rennweg 3

A-6020 Innsbruck

Tel.: +43/512/575600

Fax: +43/512/575607

e-mail: isn2005@congress-innsbruck.at

The ISN Committee for Aid and Education in Neurochemistry

The Committee for Aid for Neurochemistry (CAEN) of the International Society of Neurochemistry (ISN) has been formed with the aim of supporting neurochemical research in economically deprived countries. CAEN provides small travel and research grants as well as grants to support educational workshops. Moreover, applications for obtaining free subscriptions to the Journal of Neurochemistry will be considered.

Inquiries and requests for application should be directed to:

Steven E. Pfeiffer, USA (Chairperson)

Dept. Microbiology, L2032

University Connecticut Medical School

263 Farmington Avenue

Farmington CT 06030-3205

USA

Phone: +1-860-679-3395

Fax: +1-860-679-1239

E-mail: pfeiffer@neuron.uchc.edu

International Society for Neurochemistry Program for Conferences

As in previous years, the International Society for Neurochemistry (ISN) supports and announces programs intended to promote conferences in the science of neurochemistry throughout the world in accordance with the Society's Articles of Association.

ISN is prepared to support conference organizers up to US\$ 15,000, provided that conferences are related to neurochemistry subjects. The financial support is meant to be used for Young Neurochemists Conference Fellowships and for the Organizers. Grants will be given on the competitive basis, favouring program with international speakers and audiences. Additionally, the ISN Council decided at its Meeting in Buenos Aires 2001 that it may also be possible to sponsor activities related to the scientific program of meetings of other societies, again provided that the topics are related to neurochemistry. Applications will be examined twice a year (30 April and 31 October)

Please note that applications must be submitted at least 4 months prior to the meeting to be sponsored and that the meeting, if sponsored, should be held no later than 1 year after the application has been handled.

Inquiries and requests for application forms should be directed to:

Prof. Agustina Garcia,
Chairman of ISN Conferences
University Autònoma de Barcelona
Institut de Biologia Fundamental
V. Villar Palasi
Bellaterra
08193 Barcelona
SPAIN
Phone +34 3 581 2802
Fax +34 3 581 2011
E-mail ibftina@blues.uab.es

APPLICATION DEADLINES

APRIL 30 and OCTOBER 31,

for 2003 and the following years, unless otherwise stated.

New Membership Directory

Members will receive later in the year the new Membership Directory (issue 2002). It is intended to produce a joint membership list for members for the ISN, ASN and ESN. Members are kindly asked to immediately check the current directory (2000) and inform the Treasurer's Office on any errors in or omissions from postal and telecommunication addresses that they find. In addition, all changes in addresses that have occurred in the meantime should be communicated to the Treasurer's office. It would be appreciated if such information could be provided on the accompanying sheet 'Change of addresses' that can be cut out from this issue of the ISN News. In particular, it is important to inform the Treasurer of the e-mail address as in the future all correspondence of the membership will be carried out using e-mail.

Change of address

Please fax, mail or send to:

Dr. Roger Butterworth

Treasurer of ISN

Neuroscience Research Unit

CRC AV/Hopital Saint-Luc

1058 rue St. Denis

University of Montreal

Montreal PQ H2X 3J4

Canada

Fax: +1-514-412-7314

E-mail: Roger.Butterworth@UMontreal.CA

Name: _____

Mailing address: _____

Phone number: _____

Fax number: _____

E-mail address: _____

Web-addresses

Journal of Neurochemistry: www.blacksci.co.uk/jnc

ISN Portal: www.neurochem.org
(this page gives access to ASN, APSN and ESN)



European Society for Neurochemistry

ESN elected Officers:

- President:** Prof. Ferdinand Hucho
Dept. Biochemistry
Freie Universität Berlin
Thielallee 63
D-14195 Berlin
GERMANY
Phone: +49 30 88385545
Fax: +49 30 88385753
E-mail: hucho@chemie.fu-berlin.de
- Treasurer:** Prof. Gianfrancesco Goracci
Dept. Biochemistry and Medical Chemistry
University of Perugia
Via del Giochetto
06122 Perugia
ITALY
Phone: +39 075 5853420
Fax: +39 075 5853420 or 5853428
E-mail: goracci@unipg.it
- Secretary:** Prof. Vera Adam-Vizi, Secretary of ESN
Dept. of Biochemistry
Semmelweis University of Medicine
Puskin St. 9
P.O.Box 262
H-1444 Budapest
Hungary
Phone: +36 1 266 2773
Fax: +36 1 267 0031
E-mail: av@puskin.sote.hu

The 14th ESN Meeting

1-4 June 2003, Warsaw, Poland

A Conference on “Advances in Molecular Mechanisms of Neurological Disorders”

The conference will be focused on Parkinson’s disease, neurodegeneration and pain.

The meeting will start at the afternoon June 1st, with an opening lecture by Prof. **Peter Jenner** (London) “*Molecular Advances in the Molecular Pathophysiology of Parkinson’s Disease – Therapeutic Implications*”.

On three consecutive days the morning sessions will be devoted to the following symposia:

1. *Intercellular communication in brain in health and disease*
Organised by Prof. Joan Abbott (London) and Prof. K.A. Nalecz (Warsaw)
2. *Oxidative Stress*
Organised by Prof. Vera Adam-Vizi (Budapest)
3. *Molecular Aspects of Pain*
Organised by Prof. Ferdinand Hucho (Berlin) and Prof. Barbara Przewlocka (Cracow)

The following workshops will be organized in the afternoons:

- New techniques in Parkinson’s research;
- Apoptosis;
- Stem cells – potential therapeutic use;
- Diabetic neuropathy;
- Lipid mediators in brain function/dysfunction;
- Defects in protein degradation in familial Parkinson’s disease;
- Neurone/astrocyte interaction;
- Mitochondrial disease;
- Drug delivery and blood-brain barrier;
- Neurochemical basis of chronic pain therapy.

More up-to date information at:

<http://www.nencki.gov.pl/esn>

IMPORTANT:

Early registration and abstract submission deadline – January 15th, 2003

Programme Committee:

John B. CLARK (London, UK) *Chairman*, Ferdinand HUCHO (Berlin, Germany) – *ESN President*, Vera ADAM-VIZI (Budapest, Hungary) – *ESN Secretary*, Gianfrancesco GORACCI (Perugia, Italy) – *ESN Treasurer*, Katarzyna A. NALECZ (Warsaw, Poland) - *local organizer*, Carlos B. DUARTE (Coimbra, Portugal), Bernd HAMPRECHT (Tübingen, Germany), Jakob KORF (Groningen, The Netherlands), Olle LINDVAL (Lund, Sweden), Pierluigi NICOTERA (Leicester, UK), Andreas PLAITAKIS (Crete, Greece), Barbara Przewlocka (Cracow, Poland)

Local Organizer: Katarzyna A. Nalecz, Nencki Institute of Experimental Biology, 3 Pasteur Street, 02-093 Warszawa, Poland

Scientific Secretariat: esn@nencki.gov.pl



Application for (check one)

- Ordinary Membership
 Corresponding Membership (outside Europe)
 Affiliate Membership (young researchers ?)

Name _____

Last First Middle _____

Current Position _____ Degree(s) _____

Institution _____

Mailing Address _____

Country _____

Telephone _____ Fax _____

E-mail: _____

All applicants must enclose a current Curriculum Vitae and a list of recent publications.

I share the aims of the European Society for Neurochemistry.

Signed _____ Date _____

This application must be signed by one Ordinary Member of the Society.
(I do not remember what we decided)

Supporting Sponsor Signature _____

Name (typed or printed) _____

American Society for Neurochemistry

The logo consists of the letters 'ASN' in a bold, stylized, sans-serif font. The 'A' is formed by two thick strokes meeting at a point. The 'S' is a single, thick, continuous stroke. The 'N' is formed by two thick strokes meeting at a point. The letters are dark gray and centered on the page.

Newsletter



Winter 2002

www.ASNeurochem.org

Message from the President

A message from Robert Yu, President

I would like to take this opportunity update you on the recent events of ASN. During the past year, we witnessed many changes and new challenges that directly affected us. One of the most significant changes was the departure of Linda Garcia, who resigned from being the Meeting Organizer for 2003. Linda has been associated with the Society for a number of years, and her responsibilities included providing administrative management of the ASN Office and organizing the annual meetings. She has decided to devote her effort to the management aspect of the Society, and will continue to work with the Society to ensure the smooth functioning of the Office. To replace Linda's role as Meeting Organizer, we were able to quickly appoint Ms. Sheilah Jewart, Amazing Occasions, Inc., to this indispensable position. Sheilah has had many years of experience in organizing scientific meetings at the national level. She is pleasant, highly organized, and is able to quickly restore stability to our Society. She has worked diligently with Jean deVellis, Chair of the Local Host Committee, Greg Sutcliffe, Chair of the Program Committee, and Linda Garcia to ensure the smooth planning of the 2003 Newport Beach Meeting. In this regard, I wish to thank Jean and Greg for their effort in planning the scientific and social programs, which promises to make the 2003 meeting one of the most successful gatherings in recent years.

We I took over the Presidency, I resolved to respond to the following challenges that were facing our Society:

- To remain as a vital and forward-looking Society, we must actively recruit young, talented and vibrant neurochemists and to expand our membership base. A special membership drive, "ASN Hat", is underway under the chairmanship of Alex Chiu. I am requesting that each of you nominate at least one new member a year.
- To better improve our scientific programs, a membership opinion survey is underway with George DeVries taking the lead. I have also established an ad hoc Publication Committee, chaired by David Shine, to examine the possibility of having an ASN affiliated scientific journal that will substantially increase the impact and financial stability of our Society. The ad hoc Committee on the Future of ASN continues to provide invaluable advices that will improve the well-being of the Society and to ensure that the Society stay at the cutting-edge of neuroscience. We are grateful to Jean deVellis for his remarkable leadership in this Committee.
- To improve our competitiveness in the neuroscience arena and to more accurately reflect the nature of our scientific inquiries, I have established a new ad hoc Committee chaired by Wendy Macklin to examine the possibility of a society name change. This plan has the endorsement of the Council.

- To improve fundraising, I have appointed an ad hoc Fundraising Committee chaired by Jean Merrill to coordinate our fundraising efforts.
- We have increased substantially the number of travel awards to young and Latin investigators to enable them to attend the meeting. We also sought special funds to support members who would otherwise not be able to attend the Palm Beach meeting due to adverse economic situation. A conference application that requests substantial financial support has been submitted for assisting these initiatives. We will continue these efforts for future meetings.

I wish to announce the appointment of Dr. Robert Ledeen as Chair of the 2004 ASN Meeting. Dr. Ledeen is an outstanding neurochemist and has served in the same capacity before. He has already assembled a team that consists of many prominent scientists to assist him in this important function. We look forward to a wonderful scientific program for the 2004 Annual Meeting.

Finally, I would like to acknowledge several colleagues, notably David Shine, Lynn Hudson, and Jean deVellis, to name a few, whose constant, capable assistance has made my job to serve you a pleasant one.

ASN's *Coordinates* are.....

American Society for Neurochemistry

P.O. Box 143060

Gainesville, FL 32614-3060

(Express mail only: 8032 SW 45th Lane, Gainesville, FL 32608)

phone: 352/271-3383

fax: 352/271-3060

E-mail: LindaHou@aol.com

Secretary's Corner

Amendments ByLaws Passed

All amendments to the Bylaws passed. Of the 503 dues paying members a majority, 268, voted. Thanks to Linda Garcia for collecting and counting the ballots. The new bylaws are posted on the ASN website. The final vote tabulation was:

Amendment	Yes	No	Abstain
1	261	6	1
2	254	12	2
3	251	15	2
4	260	3	5
5	251	11	6

A major overhaul of the bylaws to remove phrases that are no longer germane, to improve the grammar, and removed parenthetical phrases is planned.

Nominations Open

You should soon receive nomination forms for President-Elect, Secretary, Treasurer and five Councilors for the term 2003-2005. Please participate in this vital function of our society. The announcement is reprinted in this newsletter.

Standing Rules Updated

Cara-Lynne Schengrund, ASN's Parliamentarian and the Standing Rules Committee have updated the Standing Rules as passed by Council. They are posted on ASN's web site.

Dues Time

You should have received your dues notice for 2003. Please pay promptly. This year we are strictly following the bylaws and striking members from our roles that are in arrears. If you did not get a dues notice please contact Linda Garcia in the ASN business office.

Please Contribute to Award Funds

When you pay your dues please consider contributing funds for the Marian Kies and Jordi Folch-Pi Award Funds. Karen Chandross wrote this for the Kies fund that is relevant to both awards:

Please consider making a contribution to the American Society for Neurochemistry Marian W. Kies Memorial Fund. Dr. Kies was a successful and well respected neurobiologist who set high scientific standards and was a mentor to young scientists. It is therefore appropriate that this award recognizes the accomplishments of junior scientists who demonstrate outstanding predoctoral

thesis research in the field of neuroscience. The award includes a cash prize of \$1500.00 and the opportunity to organize and chair a symposium in 2004, with financial support from the ASN. The continued vitality of this award depends on your support.

Encourage strong candidates from your laboratories to apply for the 2002 Folch-Pi and Kies Awards now. The winners will be announced at the 34rd annual meeting in Newport Beach, CA.

Round Up New Members!

The Membership Committee has streamlined the membership process and one can submit an application online. Please ask your colleagues to join the Society. Note that the membership categories have been changed to Ordinary and Corresponding. Students and postdocs are not considered Ordinary members. An application is on the last page of this newsletter.

Membership Survey

President-Elect George DeVries is writing a survey to gather information from the ASN membership. Look for it in the mail and please fill it out and return it. This will help the officers and council chart the course of ASN for the future.

Bernard Haber Award Lecture

President Robert Yu has appointed Dr. Nicolas Bazan the first chairman of the Bernard Haber Award Lecture Committee. This award recognizes the contribution of Dr. Bernard Haber to the ASN as Secretary for 6 years and as Intersociety Liaison Officer for more than 2 years. It will honor an ASN member who has contributed greatly to intersociety interactions between the ASN and the ISN or other societies.

Basic Neurochemistry on PubMed

Basic Neurochemistry, 6th edition, has been added to the on-line reference book shelf of PubMed. It will shortly be available for searching and link out in the PubMed database.

Missing Members

We have lost contact with these members. If you know their whereabouts please ask them to contact the ASN business office or me.

Tyson Tildon – Univ. of Maryland
R. Lingaman – Cornell
John Fitzgerald – Hines VA
Francis LeBaron – Mashpee, MA
Diwakar Deshmukh – Staton Island, NY
Karine Galoian – Allegheny Univ. Health Sci.
John Hines – Texas Woman's Hospital
Patricia Johnston – Champaign, IL
Duska Separovic – Cleveland, OH

NOMINATIONS FOR OFFICERS AND COUNCILORS AMERICAN SOCIETY FOR NEUROCHEMISTRY

Nominations are requested for President-Elect, Secretary, Treasurer and five Councilors for the American Society for Neurochemistry for the term 2003-2005.

1. Fill out the nomination form that you received in the mail.
2. Return the form either by fax or regular mail before January 9, 2003
- *E-MAIL RESPONSES CANNOT BE ACCEPTED*

FAX: (352)271-3060 (include member name on cover sheet)

REGULAR MAIL – Place the form in the envelope that you received and sign your name on the outside. Add postage.

American Society for Neurochemistry
P.O. Box 143060
Gainesville FL 32614-3060

Our current President-Elect is George DeVries; he will assume office in May, 2003. The nomination for President-Elect is for the individual who will succeed George DeVries as President in 2005. The current Secretary and Treasurer are eligible to run for election again. Nominations for Councilors are needed to replace 5 Councilors with terms ending in 2003.

View the current list of officers and twelve Council members at the “Structure” section of ASN web site, www.ASNeurochem.org. According to ASN by-laws and standing rules, each member may nominate no more than one candidate for each vacancy. Since there are five Council vacancies, each member may nominate five individuals for Council. All nominations are forwarded to the Nominating Committee, which will construct a list of candidates (individuals who received nominations from at least six members, plus individuals nominated by the committee). Ballots will be mailed out in early February.

Please contact the chair of the nominating committee by phone or e-mail if you have any questions.

Joyce Benjamins
Chair, ASN Nominating Committee

Phone (313)577-1275

E-mail joyce.benjamins@wayne.edu

Kunihiko Suzuki Awarded the Japan Academy Prize

by Pierre Morell



Your eyes are not fooling you – this is a photograph of our very own Kunihiko Suzuki. This is indeed the same casual and informal “Kuni”, whom we have never before seen wearing anything more elegant than a turtleneck. Still, after 40 years of arguing his way into formal receptions and prestigious restaurants in casual attire, Kuni has apparently met his match. Apparently, the protocol office of the Government of Japan could not be bluffed. In order to be at a reception hosted by the Emperor and Empress of Japan, and to receive a medal from the hands of the Emperor, one wears a tuxedo. This was the case on June 10, 2002 when Kuni was the recipient of the Japan Academy Prize.

A little background concerning this prize. It is awarded by The Japan Academy – a group somewhat equivalent to the U.S. National Academy of Science but representing also non-science based disciplines such as literature and history. This body annually selects several recipients of the Japan Academy Prize for outstanding accomplishments in the respective fields. It is considered the most prestigious honor that can be bestowed upon Japanese academics. For 2002, there were ten recipients in 9 subject areas (the prize for one subject was given to two collaborators). Kunihiko Suzuki, Director emeritus of the Neuroscience Center, Professor emeritus of Neurology and Psychiatry at University of North Carolina at Chapel Hill, and President emeritus of both the ASN and the ISN, was among this year’s awardees. He was honored for his life-long work on “Pathogenetic mechanism of neurogenetic disorders, particularly sphingolipidoses”.

The formal award ceremony took place in the morning on June 10, 2002 at the Japan Academy in Tokyo. It was presided by the Emperor and the Empress of Japan, and the Prime Minister and the Minister of Education and Science also attended the session. Prior to the ceremony, individual recipients were asked to explain their work to the Emperor and Empress with prepared poster exhibits. A few “guests” were allowed at the ceremony for each of the prize recipients, and Dr. Suzuki invited as his guests the first four then-young Japanese

postdoctorals who came to his laboratory in the 1960's and the earliest part of the 1970's. They were Shigehiko Kamoshita, Yoshiyuki Suzuki, Yoshikatsu Eto and Tadashi Miyatake. The award ceremony was followed by an afternoon "tea" session at the Imperial Palace where, in addition to the Emperor and Empress, the Crown Prince and his younger brother and their respective wives were also in attendance allowing mingling and chatting with the prize recipients during the two-hour session of light meal. The day was concluded by dinner at a hotel in downtown Tokyo hosted by the Minister of Education and Science.

For those not acquainted with the scope of Kuni's career in neurochemistry there follows a summary prepared as a new release by the University of North Carolina news bureau.

Dr. Kunihiko Suzuki received his undergraduate degree in History and Philosophy of Science from Tokyo University and then graduated from the Faculty of Medicine of Tokyo University. Immediately after a year of rotating internship, he was first trained as a clinical neurologist in the United States before committing his life to research in genetic neurological disease. His laboratory work reflects these different phases of his background training.

Despite the rapid progress in recent decades, genetic neurological disorders, particularly those that affect infants and children, still pose a formidable challenge to our understanding of their pathogenetic mechanisms and eventual effective treatment. Methodological approaches to these problems have been evolving rapidly in the past 40 years. In the 60's, it was primarily analytical and structural. Metabolic/enzymological approaches were predominant in the 1970's. Then, the field shifted to the present heavily molecular biological emphasis in the past 20 years. This evolution was intimately associated with technological as well as conceptual developments in all fields of basic biology. Throughout his research career spanning the past 40 years, the focus of Dr. Suzuki's research has always been on the pathogenetic mechanism of these diseases with a firm belief that effective treatment would not be possible without first understanding their biology. He has always been at the forefront of the field by adopting new methodological developments. It would be difficult to describe progress in research in neurogenetic diseases, particularly sphingolipidoses, during the second half of the 20th century without extensive references to Dr. Suzuki's numerous contributions.

Among his extensive contributions, the series of work on Krabbe disease (globoid cell leukodystrophy) best represents the nature of Dr. Suzuki's research accomplishments. In 1970, Dr. Suzuki, together with Dr. Yoshiyuki Suzuki who was in his laboratory at that time discovered the genetic basis of this classical genetic disease affecting myelin as a galactosylceramidase deficiency. This discovery opened the way for the first time for non-invasive ante-mortem and prenatal diagnosis of this disease. Almost immediately, the team also discovered that the dog model previously known on the basis of

neuropathology was genetically equivalent to the human disease. A few years later in 1972, Dr. Suzuki and Dr. Tadashi Miyatake, then in his laboratory, proposed a hypothesis that has since been known as the “psychosine hypothesis” in order to explain some of the unusual features of Krabbe disease. This hypothesis was the first serious attempt at clarifying the mechanistic basis of how these genetic diseases affect brain function. Although greeted by skepticism initially, the hypothesis has not only been gaining wide acceptance during the past 30 years but is now recognized as being also relevant to other brain diseases. The discovery of a mouse model of the disease, “the twitcher mouse”, in 1982 by Dr. Suzuki together with the late Dr. Takuro Kobayashi provided one of the most extensively utilized naturally occurring animal models of genetic neurological diseases. Dr. Suzuki himself utilized it for nerve transplantation, bone marrow transplantation, transgenic treatment, and more recently gene therapy experiments of this disease.

During the past decade, Dr. Suzuki applied the nucleic acid technology, particularly that of mouse genome manipulation (“gene knockout”) to clarify the pathogenesis of various sphingolipidoses and associated disorders. Notable among the mutant mice Dr. Suzuki’s laboratory has generated are the mouse with total sphingolipid activator protein deficiency, a β -galactosidase deficient mouse (GM1-gangliosidosis mouse), and a galactosylceramide synthase knockout mouse. Extensive cross breeding experiments among the mutant mice gave insight into the pathogenesis of individual diseases that no other approaches could have provided. Most recently, Dr. Suzuki established, together with Dr. Junko Matsuda, that one of the sphingolipid activator proteins, “saposin A” is the second gene, deficiency of which can cause Krabbe disease and that pregnancy dramatically improves the phenotype of saposin A-deficient female mice. This may well mark the beginning of a new important series of studies with significant implications to these currently incurable genetic neurological diseases.

In recognition of his scientific contributions, Dr. Suzuki has been President of the International Society for Neurochemistry, and of the American Society for Neurochemistry, Chief Editor of the Journal of Neurochemistry, and Director of the Neuroscience Center at University of North Carolina. He is a member of the Institute of Medicine of the National Academy of Sciences of the United States. Another of his significant contributions to Japanese Science is that he has continually accepted and trained young Japanese researchers in his laboratory in the past 40 years and their number has reached 30. Many of his younger colleagues who passed his laboratory have played critically important roles in advancing the field after they returned to Japan.

REMEMBER.....

Please see that your address information updated.

Send your information to the ASN business office:

American Society for Neurochemistry

P.O. Box 143060

Gainesville, FL 32614-3060

Voice: 352/271-3383 Fax: 352/271-3060

E-mail: LindaHou@aol.com

You can check your information at <http://www.ASNeurochem.org>



**Announcing the
34th Annual Meeting
May 3-7, 2003
Newport Beach, CA**



Dear Colleagues:

We invite you to join us in an excellent scientific program that will be presented at the 34th Annual Meeting of the American Society for Neurochemistry in Newport Beach, California, May 3-7, 2003. Our colleagues in the Western Hemisphere and around the world interested in cellular, neurochemical and molecular studies of the nervous system in health and disease should plan to attend the ASN 2003 Annual Meeting.

The opening reception will be held Saturday, May 3rd at the Hyatt Newporter. The scientific program will begin Sunday with the Basic Neurochemistry Lecture presented by Joshua Sanes of Washington University, St. Louis, MO. Plenary speakers including Carla Schatz, Fred Gage and approximately 140 speakers will participate in 13 Symposia, 16 Colloquia and 5 Workshops designed to encompass the broad range of interests of Society members.

Short oral presentations will be featured in the meeting along with daily Poster Sessions. Abstracts will be published in a Journal of Neurochemistry supplement. Substantial time is allotted throughout the meeting for scientific discussion both formally and informally.

Special efforts have been taken to encourage young investigator participation. ASN travel awards are available and funding has been requested through NINDS for additional travel funds. A student/post doctoral Dinner will be given to the first 50 students/post docs to register for the ASN meeting. Over dinner, attendees will have an opportunity to discuss career and research issues with more senior scientists from academia, industry and funding agencies.

ASN has selected a worldclass destination for the 34th Annual Meeting in Southern California with Balboa Island, Catalina Island, Disney theme parks and the Pacific Ocean, all within a 20 mile radius. The ASN meeting headquarters located in the Hyatt Newporter is situated on 26 beautifully landscaped acres overlooking the Back Bay of Newport Beach with three swimming pools and golf course.

The ASN California Host Committee, Officers and Council look forward to sharing with you in a most productive scientific conference and a time of comradery.

Robert K. Yu
President, ASN

J. Gregor Sutcliffe
Program Chair

Jean de Vellis
Host Committee Chair

Important Deadlines:

Nov 4, 2002	On-line Abstract submission available at website
Dec 16, 2002	Deadline for on-line Abstract submission Meeting registration fees must accompany submissions
March 15	Deadline for registering as Exhibitor
March 15	Applications due: -Marian Kies Memorial Award -Jordi Folch Pi Award -Young Investigators Educational Enhancement Awards -Young Latin American Scholars Awards
April 1	Deadline for request of Committee meeting space (Limited space – based on availability)
April 11	Best-rate meeting registration deadline
April 25	Final date for ASN negotiated rates at Hyatt Newporter Final date for on-line or faxed registrations
May 2	After 5:00 pm registration on-site at Hyatt Newporter
May 3-7	3:00 pm – 7:00 pm Registration Desk open at meeting. 6:30 am – 6:00 pm Registration desk open.

ASN Meeting Coordinator

Sheilah Jewart, CMP
Amazing Occasions, Inc.
9037 Ron Den Lane, Windermere, FL 34786
Tel: 407-909-9064
Fax: 407-876-0750
Cell: 407-234-3274
e.mail: amazing@iag.net

Questions / Information / Registration

Annual Meeting Preliminary Agenda

Friday, May 2

3:00 pm – 7:00 pm ASN Registration Desk Open – Hyatt Newporter

Saturday, May 3

6:30 am – 6:00 pm ASN Registration Desk Open – Hyatt Newporter

8:00 am – 5:00 pm Pre-Meeting Workshop:

“Stem Cell Biology & Technology: Implications for Neural Repair”

3:00 pm – 6:00 pm ASN Council Meeting I*

6:30 pm – 8:30 pm ASN Annual Meeting Opening Reception

Sunday, May 4

6:30 am – 6:00 pm ASN Registration Desk Open – Hyatt Newporter

8:00 am – 5:45 pm ASN Annual Meeting Sessions

11:30 am – 1:00 pm Journal of Neuroscience Research Editorial Board*

6:00 pm – 8:00 pm Public Outreach Forum – Parkinson’s Disease

7:30 pm- 10:00 pm Intersociety Dinner*

Monday, May 5

6:30 am – 6:00 pm ASN Registration Desk Open – Hyatt Newporter

6:45 am – 8:00 am ASN Council Meeting II*

8:00 am – 5:45 pm ASN Annual Meeting Sessions

11:30 am – 1:00 pm Journal of Neurochemistry Editorial Board*

11:45 am – 1:15 pm ASN Task Force Committee*

5:45 pm – 7:00 pm Women In Neurochemistry (WIN) Reception*

Tuesday, May 6

6:30 am – 6:00 pm ASN Registration Desk Open – Hyatt Newporter

8:00 am – 6:30 pm ASN Annual Meeting Sessions

12:30 pm – 2:00 pm Developmental Neurosciences Editorial Board*

6:30 pm – 8:00 pm ASN Business Meeting

8:00 pm – 10:00 pm Student / Post Doc Dinner

Wednesday, May 7

6:30 am – 6:00 pm ASN Registration Desk Open – Hyatt Newporter

8:00 am – 5:45 pm ASN Annual Meeting Sessions

12:30 pm – 2:00 pm ASN Council Meeting III*

11:30 am – 1:00 pm Neurochemical Research Editorial Board*

6:30 pm – 10:00 pm ASN Farewell Reception / Dinner

Thursday, May 8

7:00 am – 10:00 am ASN 2004 Conference Committee

9:00 am – 5:00 am Clark / Blackwell Meeting*

* Indicates private function

Scientific Program Committee

Gregor Sutcliffe, Chair
Robert Yu, (ex officio)
Oscar Bizzozero
Scott Brady
Jorge Busciglio
Tony Campagnoni
Monica Carson
David Colman
Julie Ellison
Minnetta Gardinier
Lynn Hudson
Jean Merrill

Randy McKinnon
Bob Moore
Richard Olsen
Paul Patterson
Steve Pfeiffer
Jeff Redwine
Ian Simpson
Joan Schwartz
David Sweatt
Elizabeth Thomas
Scott Whittemore
Terri Wood

California Local Host Committee

Jean deVellis, Chair
Claude Baxter
Steve Bondy
Carl Cotman
Tony Campagnoni
Julie Ellison
Larry Eng

Caleb Finch
Carol Readhead
Eric Shooter
Paul Salvaterra
Gregor Sutcliffe
James Waschek

CALL FOR ABSTRACTS

Abstracts must be submitted on-line by December 16, 2002

The Scientific Program Committee invites registered participants and invited Speakers to submit Abstracts. All presenting authors must pay meeting registration fees by December 16 to receive abstract acceptance. Please read the instructions carefully. Abstracts will be published in the Journal of Neurochemistry. The publisher of the Journal of Neurochemistry requires that abstracts be submitted electronically by the internet. Go to www.ASNeurochem.org to submit an abstract online. If on-line abstract submission is not possible, please contact ASN Meeting Coordinator by Nov 30th.

ORAL PRESENTATION

Please indicate on the abstract computer form whether the Abstract should be considered for oral presentation of short communications. Selected oral presentations will be also assigned poster space. Those presenting oral presentations will receive notification of the date and time allocation of their presentation.

POSTER PRESENTATION

A banner should be prepared indicating the abstract title, author(s) and affiliation(s). Lettering should be at least one inch high (2.5cm). The poster boards will be 6' (1.8m) wide by 4' (1.2m) high. Pushpins will be provided in the poster area. Presenting authors must be present at the poster time period specified. Posters must be setup between 7:00 am – 9:00 am on assigned day.

Group A	Group B	Group C	Repeat Group C
<i>Sunday 5/4</i>	<i>Monday 5/5</i>	<i>Tuesday 5/6</i>	<i>Wednesday 5/7</i>
9:15 – 9:45	11:45 – 1:15	10:00 – 10:30	10:00 – 10:30
3:15 – 3:45	12:30 – 2:00	12:30 – 2:00	12:30 – 2:00
5:45 – 7:30	5:45 – 7:15	4:00 – 4:30	3:15 – 3:45

NOTIFICATION OF ACCEPTANCE

Notification of Abstract receipt AND acceptance will be provided by e.mail before March 2003.

MEETING REGISTRATION AND HOTEL RESERVATION INFORMATION

MEETING REGISTRATION

Deadline for best Registration rates is April 11, 2003

The Meeting Registration Form can be downloaded below. Registration and credit card payment is also available on-line through April 25th. After April 25th, registration will need to be on-site at the Hyatt Newporter.

The ASN Registration Desk will be open:

Friday, May 2 3:00 pm – 7:00 pm

Saturday thru Wednesday 6:30 am – 6:00 pm

HOW TO REGISTER:

Use the on-line application form at www.ASNeurochem.org

Download registration form from www.ASNeurochem.org and fax to 407-876-0750 or mail to:

ASN Conference Coordinator,
Amazing Occasions, Inc.,
9037 Ron Den Lane,
Windermere, FL 34786

Fax or mail the registration form found in the meeting brochure to the above fax number or address.

Only Mastercard and Visa credit cards are accepted.

Make check payable to: American Society of Neurochemistry (Check must be payable in US funds)

STUDENT / FELLOW / POST DOC REGISTRATION:

Students working toward a degree in neurochemistry or an allied field qualify for the Student rate. Fellows and Post Doc Students (received degree after July 1, 1999) also qualify for the Student registration rate. The first 40 to register for the meeting will be invited to a complimentary dinner Tuesday, May 6, for an opportunity to discuss career plans and other scientific issues with more senior scientists. Documented proof of student or postdoc status must be faxed to the ASN Meeting Coordinator's office at 407-876-0750. Registrations without documented proof of status will not be able to receive the Student rate.

CANCELLATION POLICY:

Registration fees, minus \$50 handling fee, are refundable if notified by April 11. After April 11, no refunds can be made.

LOCATION:

The city of Newport Beach lies along the Pacific Ocean on the coast of California in Orange County; 50 miles (80km) south of Los Angeles, 85 miles (136 km) north of San Diego, 14 miles (22 km) west of Disneyland/Anaheim, 14 miles (22 km) south of Long Beach. Elevation ranges from sea level to 691.3 feet. There are several different villages that make up Newport Beach such as Balboa Island, the Balboa Peninsula, Cannery Village, Corona Del Mar, and numerous attractions within 25 miles radius such as Disneyland, Sea World, Universal Studios, Knott's Berry Farm, Laguna Art Museum and Catalina Island.

TRANSPORTATION:

The John Wayne County Airport (SNA) serves as the hub of Newport Beach with convenient access to the hotels and attractions. The Hyatt Newporter provides a complimentary airport shuttle for guests from the John Wayne Airport.

Distance from Airports to Hyatt Newporter:

John Wayne Airport (SNA) – 11 miles – (Hyatt Shuttle 949-729-6107)

Los Angeles International Airport (LAX) – 47 miles (Super Shuttle 310-782-6600)

Long Beach Airport (LGB) – 27 miles – (Taxis available)

Ontario International Airport (ONT) – 48 miles – (Super Shuttle 909-467-9600)

CLIMATE:

Orange County benefits from ocean breezes to keep its weather milder than the inland regions. The average daytime temperature in Spring is 71-49F (22-9C), so bring a light jacket or sweater for the evening functions.

HOTEL RESERVATIONS

The official ASN Annual Meeting Headquarters hotel is:

Hyatt Newporter Resort

1107 Jamboree Road, Newport Beach, CA 92660

Tel: 949-729-1234 – Fax: 949-644-1552

Website: www.hyattnewporter.com

ASN has negotiated discounted group rates for the meeting at the Hyatt Newporter, the meeting headquarters hotel. Situated on 26 beautifully landscaped acres overlooking the Back Bay of Newport Beach, this resort-style hotel reflects the warmth and charm of Southern California.

To Make Reservations – Be sure to mention “ASN Group” for discount rate:

- Either call Hyatt Reservations at 1-800-233-1234
- or go to <http://newporter.hyatt.com/groupbooking/neur>
- or call Hyatt Newporter's Reservation Department at 949-729-1234

DEADLINE FOR GROUP RATE FRIDAY, APRIL 11, 2003

Scientific Program

Saturday, May 3, 2003

6:30 – 6:00 – Registration

8:00 – 5:00 – Pre-meeting Workshop:

Stem Cell Biology & Technology: Implications for Neural Repair

Organizers: Jean deVellis and Hydeyuki Okano

6:30 – 8:30 – Opening Reception

Sunday, May 4, 2003

8:00 – 8:15 – Opening Remarks

Robert Yu, President, American Society for Neurochemistry

Jean de Vellis, Chairman, Host Committee

8:15 – 9:15 – Basic Neurochemistry Lecture

Introduction by Robert Yu

Joshua Sanes title TBA

9:15 – 9:45 – Coffee Break

9:45 – 11:45

Symposium 1: Neurochemistry of Cognition

Organizer: J. David Sweatt

J. David Sweatt

Daniel Storm

Alcino Silva

Kim Huber

9:45 – 11:45

Colloquium 1: Multiple Functions of Myelin Proteins in the Immune and Nervous Systems

Organizer: A. T. Campagnoni

Tony Campagnoni

Rhonda Voskuhl

George Harauz

Vijay Kuchroo

9:45 – 11:45

Workshop 1: Regenerative Responses of Neural Stem Cells to Injury and Disease

Organizer: Steve Levison

Aliya U. Zaidi

Francis Szele

Steve Levison

John R. Fike

11:45 – 1:15 – Lunch on your own

11:45 – 1:15 – Posters

1:15 – 3:15

Symposium 2: The Role of Neuronal Plasticity in Neurodegenerative Diseases

Organizer: J Busciglio

Mark Mattson	Eliezer Masliah
Carl Cotman	Jorge Busciglio

1:15 – 3:15

Colloquium 2: Microglial Responses to Injury & Strategies for Managing Them

Organizer: Joan P. Schwartz

Monica Carson	Serge Przedborski
Ian Duncan	Robert Friedlander

1:15 – 3:15

Workshop 2: Volume-Dependent Anion Channels & Channel-Mediated Amino Acid Release – New Players in Brain Communication and Brain Pathology

Organizer: Harold K. Kimelberg

Herminia Pasantes-Morales	Nicolas Hussy
Alexander A. Mongin	John W. Phillis

3:15 – 3:45 – Coffee Break

3:45 – 5:45

Symposium 3: Transcriptional Controls Directing Neural Differentiation

Organizers: Lynn Hudson and Robin Miskimins

Joerg Leheste	Regina Armstrong
Paul Gardner	Alex Gow

3:45 – 5:45

Colloquium 3: Astrocyte-neural cell interactions

Organizer: Ian Simpson

David Spray	Ursula Sonnewald
Phillip Haydon	Jeff Rosenstein

3:45 – 5:45

Colloquium 4: Estrogen and Neurodegenerative Diseases: A Protective Role?

Organizer: Darrell W. Brann

Phyllis M. Wise	Mary Sano
Darrell W. Brann	Catherine S. Woolley

5:45 – 7:15 – Posters

6:00 – 8:00 – Public Outreach Forum

Parkinson's Disease

Organizer: *Minnetta Gardinier*

Monday, May 5, 2003

8:00 – 9:00 – Plenary Lecture

Carla Schatz title TBA

9:00 – 9:15

Presentation of the Marian W. Kies Memorial Award
by Karen Chandross

9:15 – 9:45 – Coffee Break

9:45 – 11:45

Symposium 4 Jordi Folch-Pi Memorial Symposium:
Dynamic Phospholipid Signaling In Life and Death

Organizer: *Sandra Hewett, 2002 Jordi Folch-Pi Memorial Award Recipient*

Nicolas G. Bazan Bradley Alger
Sandra Hewett David Greenberg

9:45 – 11:45

Colloquium 5: Myelin-Axolemmal Bidirectional Signaling

Organizer: *Steve Pfeiffer and Matt Rasband*

Alyson Fournier Carmen Melendez-Vasquez
Cecilia Marta Haesun Kim

9:45 – 11:45

Colloquium 6: Gene Transfer to the Nervous System: from Basic
Mechanisms to Novel Therapeutics

Organizer: *Diana Jerusalisky*

David Shine Fernando Pitossi
Pedro Lowenstein Osvaldo Uchitel

11:45 – 1:15 – Lunch on your own

11:45 – 1:15 – Posters

1:15 – 3:15

Symposium 5: Astrocyte signaling in brain inflammation

Organizer: *Etty Benveniste*

Celia Brosnan Etty Benveniste
Joel Pachter Iain Campbell

1:15 – 3:15

Colloquium 7: Genomic Approaches to CNS Injury

Organizer: Julie Ellison

Julie Ellison Frank R Sharp
Carolee Barlow Tracy K. McIntosh

1:15 – 3:15 – Oral presentations

Chair: Cara Lynne Schengrund

3:15 – 3:45 – Coffee Break

3:45 – 5:45 – Colloquium 8: Significance of Mitochondrial Heterogeneity in Neuronal-Glial Interactions

Organizers: Arne Schousboe and Roger Butterworth

Carmen Manella Helle Waagepetersen
Mary McKenna Gary Fiskum

3:45 – 5:45

Colloquium 9: To Gli or Not to Gli: What Makes an Oligodendrocyte?

Organizers: Karen Chandross and John Kessler

John Kessler Steve Goldman
Jim Goldman Tim Vartanian

3:45 – 5:45

Workshop 3: Recent Advances in Non-Invasive Imaging Techniques for Rodent Research

Organizer: Jeffrey M. Redwine

Jeffrey M. Redwine Simon R. Cherry
Russell E. Jacobs James P. Basilion

5:45 – 7:15 – Poster Session

7:15 – 8:30 – Annual Business Meeting

Tuesday, May 6, 2003

8:00 – 10:00

Symposium 6: FGFs in the Developing Nervous System

Organizers: Terri Wood and Rashmi Bansal

Manny DiCicco-Bloom David Ornitz
Susan McConnell Elizabeth Grove

8:00 – 10:00

Colloquium 10: Opioid Peptides: Normal & Pathologic Signaling Mechanisms In Neurons & Glia

Organizers: Pamela E. Knapp & Kurt F. Hauser

Carmine Coscia Kurt Hauser
Georgy Bakalkin Pamela Knapp

8:00 – 10:00

Workshop 4: Dietary management of neurological and neurodegenerative diseases

Organizer: Thomas N. Seyfried

Thomas N. Seyfried Richard L. Veech
Marc Yudkoff Mark Mattson Purna Mukherjee

10:00 – 10:30 – Coffee Break

10:30 – 12:30

Symposium 9: Marian W. Kies Memorial Symposium

Zinc in Neurodegeneration

Organizer: Kirk E. Dineley, 2002 Marian Kies Memorial Award Recipient

Elias Aizenman Jae-Young Koh
Kirk Dineley John Wiess

10:30 – 12:30

Colloquium 11: CNS Inflammation and Microglial Activation: Can They be Beneficial for the CNS?

Organizer: Monica J. Carson

G. Miller Jonakait Phillip G. Popovich
Kathryn J. Jones Wolfgang J. Streit

10:30 – 12:30

Colloquium 12: Hedgehog, BMP and Notch Signaling in Oligodendrocyte Specification

Organizer: James A. Waschek and Vincent Lelievre

Robert H Miller Monique Dubois-Dalcq
David Rowitch Gord Fishell

12:30 – 2:00 – Lunch on your own

12:30 – 2:00 – Posters

2:00 – 4:00

Symposium 8: Signaling Complexes in the Nervous System

Organizer: Heather S. Duffy

Richard Weinberg David Spray
Alex Gow Mario Delmar

2:00 – 4:00

Colloquium 13: Complement in the Brain:
New & Exciting Insights in an Old Arena of Immunology
Organizer: Jessy Alexander & Tony Wyss-Coray

Tony Wyss-Coray Scott Barnum
Andrea Tenner Giulio Maria Pasinetti

2:00 – 4:00 – Oral presentations

Chair: Minnetta Gardinier

4:00 – 4:30 – Coffee Break

4:30 – 6:30

Symposium 9: MMPs in the Nervous System: Functions in Physiology,
Pathology and Recovery

Organizer: V. Wee Yong

Zena Werb Eng H. Lo
Sarah McFarlane V. Wee Yong

4:30 – 6:30

Colloquium 14: Integrin Signaling in the Nervous System: Involvement with
Tetraspan & Related Proteins

Organizer: Wendy Macklin

Jeff Bronstein Wendy Macklin
Senitiroh Hakomori David Weinstein

4:30 – 6:30

Symposium 10: Neural Differentiation

Organizers: Randall D. McKinnon

Chris Q. Doe Sam Pfaff
Mark Henkemeyer John B. Thomas

6:30 – 8:00 – Posters

8:00 – 10:00 – Student/Postdoc Dinner

Wednesday, May 7, 2003

8:00 – 10:00

Symposium 11: Synapse Formation and Resculpturing

Organizer: Richard Olsen

Heinrich Betz Kimberley McAllister
Istvan Mody Roger Nicoll

8:00 – 10:00

Colloquium 15: Integrins as Targets for Therapeutic Intervention in Multiple Sclerosis

Organizer: Jean Merrill

Stefan Brocke Ted Yednock
Stephen Miller David Miller

8:00 – 10:00

Workshop 5: Proteolipid Protein (PLP): Mutations in the Gene Lead to Complex Phenotypes Affecting Myelin and Axons

Organizer: *Ian D. Duncan*

Ian D. Duncan Julia M. Edgar
Odile Boespflug-Tanguy Robert P. Skoff

10:00 – 10:30 – Coffee Break

10:30 – 12:30

Symposium 12: Cell Death Signaling Mechanisms in Chronic and Acute Trauma

Organizer: *Regino Perez-Polo*

Stephen Rossner John Bethea
Olivera Nesic Ian Hendry

10:30 – 12:30

Colloquium 16: Gene Profiling in Disease

Organizer: *Elizabeth Thomas*

Jean Loring Elizabeth Thomas
Frank Middleton John Kelsoe

10:30 – 12:30 – Oral presentations

Chair: *Oscar Bizzozero*

12:30 – 2:00 – Lunch on your own

2:00 – 3:00 – Plenary Lecture

Fred Gage title TBA

3:00 – 3:15

Presentation of Jordi Folch-Pi Memorial Award by Regina Armstrong

3:15 – 3:45 – Coffee Break

3:45 – 5:45

Symposium 13: Stem cell biology and their potential for CNS repair

Organizer: *Scott Whittemore*

Mahendra Rao Scott Whittemore
Harley Kornblum

7:00 -10:00 – Final Banquet

ASN YOUNG INVESTIGATOR EDUCATIONAL ENHANCEMENT AWARDS

The American Society for Neurochemistry is pleased to announce the availability of awards for young investigators to attend the ASN 34th Annual Meeting, Newport Beach, California.

Eligibility requirements:

1. Graduate students in their final year of study or scientists who received a Ph.D. or M.D. within the past 6 years.
2. Applicants must reside in the Western Hemisphere but need not be members of the ASN.
3. Applicants must be first (presenting) author of a submitted abstract.
4. Priority will be given those who have not previously received this award from the ASN.
5. The number of applicants from any one laboratory is limited to two.

Application materials to be submitted:

(Note: Eight copies of each must be provided)

1. A cover letter containing:
 - a. A brief statement of career goals.
 - b. Name of dissertation supervisor or research director
 - c. A statement as to whether you have or have not received an ASN education (travel) award previously.
 - d. Current source of financial support. Note that only partial support for attendance at the meeting will be provided. The amount will be dependent on the resources available to the Society and will take into consideration the applicant's travel expenses to reach Newport Beach, CA.
2. A brief curriculum vitae or biographical sketch including, at a minimum, origin and date of award of undergraduate and graduate degrees, and a list of publications and/or title of dissertation.
3. Text of submitted abstract to ASN Newport Beach Meeting

Deadline is March 15, 2003.

Application material should be sent to:

Thomas N. Seyfried
Department of Biology
Boston College
Chestnut Hill, MA 02167 USA
Phone: 617/552-3563 Fax: 617/552-2011 thomas.seyfried@bc.edu

Applications must be received by March 15, 2003 and will be reviewed by members of the Award Committee.

JORDI FOLCH-PI MEMORIAL AWARD

The Jordi Folch-Pi Award, to be used for travel, is given to an outstanding young investigator who has demonstrated a high level of research competence and originality, who has significantly advanced our knowledge of neurochemistry and who shows a high degree of potential for future accomplishments. The award is a cash prize of \$1,500 and the recipient has an opportunity to organize a symposium at the ASN 35th Annual Meeting.

Past awardees include: Drs. Gary Gibson, Robert J. DeLorenzo, Bruce Trapp, James Gusella, Oscar Bizzozero, Charles Nemeroff, Scott Brady, Wendy Macklin, Eric Nestler, Errol De Souza, Cheryl Craft, Brian Popko, Randy McKinnon, Daniel Kaufman, Mark Mattson, Peter Baas, Regina Murphy, Eric Murphy, Mark Smith, Kelsey Martin, and Sandra Hewett.

ELIGIBILITY REQUIREMENTS:

1. Nominee need not be an ASN member.
2. Nominee must reside in the Western Hemisphere.
3. No more than ten years past awarding of their doctorate at the time of nomination.

NOMINATION MATERIALS TO BE SUBMITTED:

Eight (8) copies of all materials MUST be provided.

1. Complete curriculum vitae
2. A one page statement from the nominee of accomplishments and goals
3. Three letters of recommendation.
4. Reprints of three peer reviewed publications.

EIGHT (8) COMPLETE COPIES OF NOMINATION MATERIALS SHOULD BE SENT TO:

Regina Armstrong – Chair
Dept. of Anatomy, Physiology and Genetics
Uniformed Services University of the Health Sciences
4301 Jones Bridge Road
Bethesda, MD 20814-4799
Voice: (301) 295-3205 Fax: (301) 295-1715 email: rarmstrong@usuhs.mil

The deadline for receipt of letters of nomination and all required items is March 15, 2003.

MARIAN KIES MEMORIAL AWARD

This is a call for nominations for the Marian Kies Memorial award to honor a junior scientist for outstanding research conducted during graduate training. The award is named in memory of Marian Kies who devoted much of her energy and enthusiasm to fostering the development of young scientists and consists of a \$1,500 cash prize and the opportunity to organize a colloquium for the ASN 35th Meeting.

Eligibility requirements:

1. Ph.D. or thesis awarded between May 2001 and December 2003
2. Ph.D. dissertation research of exceptional quality in the field of neuro-chemistry
3. Nominee must submit an abstract to the ASN 34th Annual Meeting
4. Nominee must have performed dissertation work in the Western Hemisphere

Nomination materials to be submitted:

(Note: Six copies of ALL materials must be provided)

1. Letter of recommendation from nominee's Ph.D. advisor and one other appropriate senior scientist familiar with the nominee's research.
2. A summary of the nominee's pre-doctoral research (maximum length of 1 single-spaced page), which highlights the impact and contribution to her/his field of study.
3. Curriculum vitae of the nominee that includes list of all publications, honors, awards as they relate to thesis research, involvement and activities in scientific societies, and extracurricular/service activities.
4. Two peer-reviewed manuscripts, manuscripts in press, or manuscripts submitted for publication.
5. Text of the abstract submitted to the ASN 34th Annual Meeting in Newport

Nomination material should be sent to:

Karen Chandross, Ph.D.
Aventis Pharmaceuticals
CNS Division
Blg J Room 3346, Route 202-206
Bridgewater, NJ 08807-0800
Voice: 908-231-3991 Fax: 908-231-2413
email: karen.chandross@aventis.com

Nominations must be received by March 15, 2003 and will be reviewed by members of the Award Committee. Please submit six copies of the nomination materials.

YOUNG LATIN AMERICAN SCHOLARS AWARD

The American Society for Neurochemistry announces the availability of scholarships for young neuroscientists from Latin American countries. These awards will provide \$2,000 in travel expenses to attend the annual meeting of the American Society of Neurochemistry, along with the visit, for a period not shorter than one additional week, to an established neuroscience laboratory in North America. The ASN hopes that these awards will foster mentoring relationships, to teach young scientists new techniques, and to share and encourage new ideas and approaches to neurochemical problems.

Eligibility requirements:

Awards are restricted to graduate students, postdoctoral fellows, and faculty no more than 35 years of age at the time of application.

Application materials to be submitted:

(Note: Six copies of each must be provided)

1. Cover letter stating the applicant's career goals and the purpose of his/her visit to a particular neuroscience laboratory in North America.
2. Complete curriculum vitae of the applicant.
3. Letter of recommendation from a senior scientist who is familiar with the applicant's research.
4. Letter from the host laboratory accepting the visit of the applicant.
5. Text of submitted abstract to the ASN Newport Beach meeting.

Deadline is March 15, 2003.

Application material should be sent to:

Dr. Oscar Bizzozero
Dept. Cell Biology and Physiology
University of New Mexico Health Science Center
Basic Med Sci Bldg. Rm 149
Albuquerque, NM 87131-5218
Tel: 505-272-5520 Fax: 505-272-9105
email: obizzozero@salud.unm.edu

Applications must be received by March 15, 2003 and will be reviewed by members of the Award Committee.

ASN welcomes the following new members for 2002:

Ordinary Members

Marta Antonelli
Universidad de Buenos Aires
Buenos Aires, Argentina

Darrell W. Brann
Medical College of Georgia
Augusta, GA

Susan E. Browne
Weill Medical College of Cornell Univ
New York, NY

Colin Combs
Univ. of North Dakota, School of
Medicine
Grand Forks, ND

C. Edward Dixon
Univ. of Pittsburgh School of Medicine
Pittsburgh, PA

Yogesh Dwivedi
Univ. of Illinois at Chicago
Chicago, IL

James A. Hewett
Univ. of Connecticut Health Center
Farmington, CT

Soo-Youl Kim
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Cornell Univ.
White Plains, NY

Mark S. Kindy
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Lexington, KY

Lawrence Litt
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San Francisco, CA

Ann Marini
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Health Sciences
Bethesda, MD

Paul M. Mathews
NYU Medical School
Orangeburg, NY

Susan McGuire
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Center
Chicago, IL

Olivera Nestic
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Francisco Nualarta
Univ. de Concepcion
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Columbia, MO

Iyer K. Ramaswamy
UCLA School of Medicine
Los Angeles, CA

Keiji Suetake
Sapporo Medical Univ.
Sapporo, Hokaido Japan

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The Scripps Research Institute
La Jolla, CA

Margaret J. Velardo
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Tian Xia
Medical College of Georgia
Augusta, GA

Lijun Zhou
Baylor College of Medicine
Houston, TX

Judy Zhu
Hunter College – CUNY
New York, NY

Corresponding member:

John Melville Land
Institute of Neurology
London, England

In Memoriam



Nancy Dahl

Emeritus member, Nancy Ann Dahl, 70, Lawrence, died Aug. 8, 2002 in Lawrence. She joined KU as an assistant professor in 1964 and became a full professor of physiology and cell biology in 1992. Her work with KU included nearly 40 scientific papers and presentations about the eye and energy flow to nerve tissue. She was inducted into the KU Women's Hall of Fame in 1993 and received the Dean's Scholars Program Mentor Award in May 1994 and the Chancellor's Club Career Teaching Award in 1995. Survivors include her husband Dennis, son Julian and daughter Kathleen R. Nuckolls, all of Lawrence

Willa Folch-Pi

Dr. Willa Folch-Pi the widow of Jordi Folch-Pi died on November 16, 2002. An obituary will be posted in the next newsletter.

ASN Officers (2001-2003)

President: Robert K. Yu

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David Colman (1999-2003)
Ian D. Duncan (2001-2005)
Charissa A. Dyer (2001-2005)
Minnetta Gardinier (2001-2005)
Steven W. Levison (2001-2005)
Wendy Macklin (1999-2003)
Mary C. McKenna (2001-2005)
Jean Merrill (1999-2003)
J. Regino Perez-Polo (2001-2005)

Alternates to Council:

Alexander Gow (2001-2005)
Richard C Wiggins (2001-2005)

ASN Standing Committees

Basic Neurochemistry – Editorial Board

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Edward Hines Jr. Hospital
Dept. Veteran Affairs
Hines, IL 60141
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Members:

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1. President's comments – Dr. Katsuhiko Mikoshiba

November 2002

The Japanese Society for Neurochemistry was established 44 years ago aiming to understand brain function and patho-physiology of neuronal diseases at the molecular and biochemical level. Our community is one of the largest neurochemical societies in the world and is composed of 1800 active members, most of which are Japanese in various universities and firms in Japan. However, I realized that our society should be open for people in the world including the young researchers in an Asian Pacific Area. In addition, we decided to newly add this role on our ordinary function of JSN, and to support activities of APSN as a member of APSN. As the first step, we started to remodel our annual meeting's style from the 46th annual meeting of this year (2002) at Sapporo, to facilitate communication among not only domestic members but also Asian and Oceanian neurochemists. To be comfortable for such overseas presenters and visitors, all abstracts were now English-written. Name-plates, directories, and program in the abstract book had both style of language.

Furthermore, we held the first ISN symposium during the Sapporo JSN meeting. This was extremely successful and was great occasion for us to improve JSN toward the internationally-opened society. The topic we discussed in the ISN Symposium was "Molecular Basis of Neuronal Signaling". Briefly, Dr. Robert Mailnow (Cold Spring Harbor Laboratory, New York) presented AMPA receptor trafficking during plasticity; Dr. Masayoshi Mishina (Tokyo University), Molecular genetics of the synapse formation and function; Dr. Nancy Y. Ip (Hong Kong University of Science and Technology, China), cdk5 – a new player at the neuromuscular synapse; Dr. Brian A. Hemmings (Friedrich Miescher Institute, Switzerland), Structure, regulation and function of Akt/PKB – a pleiotropic protein kinase involved in multiple signaling pathways; and Dr. Susan G. Amara (Vollum Institute, Oregon Health & Science University), Excitatory amino acid transporters: New insights into structure, function and regulation.

I hope these kinds of activity of JSN stimulate neurochemists in countries belonging to APSN as well as world-wide neurochemists and help more the advancement of neurochemical understanding of brain function.

(Dr. Katsuhiko Mikoshiba is Prof. of University of Tokyo: mikosiba@ims.u-tokyo.ac.jp)

2. Dr. Marshall Nirenberg was elected as the first honorary foreign member of JSN

At the general assembly during the Sapporo meeting, Dr. Marshall Nirenberg, Chief of the Laboratory of Biochemical Genetics, National Heart, Lung, and Blood Institute, National Institutes of Health, Maryland, USA, was elected as the first foreign honorary member of JSN. He is the 1968 Nobel prize winner for translation of the genetic code and its function in protein synthesis. And his well-known discoveries of cloning of neuroblastoma cells, A2B5 monoclonal antibody used in glial cell lineage, and cDNA cloning of the *Drosophila's* NK2 homeobox gene in neuronal development are landmark contribution to neurochemical research. In November 13, 1980, he, together with Drs. E Caraway and A. Guidotti, delivered his special lecture at the 23th annual meeting of JSN at Matsuyama, organized by Prof. Yasuo Kakimoto, the past president of JSN. He invited many Japanese young researchers as (post-doctoral) Visiting Fellow or Visiting Scientist in NIH, most of them now have gotten high positions in many universities and play an important role in JSN.

3. Annual meeting 2003: Joint meeting with the Japanese Biophysical Society

Niigata 2003. The 46th annual meeting of the Japanese Society for Neurochemistry will be held at Niigata, the middle part of the Honshu Island facing the Japan Sea, on September 24-26, 2003 (<http://brain.bri.niigata-u.ac.jp/~jsn46>). Professor Shoji Tsuji, formerly at Department of Neurology and Clinical Neurosciences, Brain Research Institute, Niigata University, and currently at Department of Neurology, Graduate School of Medicine, University of Tokyo, will serve as the Chairman of the meeting. Our 46th meeting is a joint one with the 41st annual meeting of the Biophysical Society of Japan. The president of the Biophysical Society is Professor Toshio Yanagida (Osaka University) and the chairman of the Biophysical Society's Meeting is Professor Shigeki Mitaku, Tokyo Agriculture and Technology University. The meeting will overlap by 2 days (24th and 25th). The joint conference in Niigata 2003 will be held at the Toki Messe, the Niigata Convention Center (www.niigata-babdaijima.com), at the water front of the bank of River Sinano in Niigata City.

The special topic of the joint meeting focuses on 'Application of one molecule imaging in neuronal cells: real time imaging and functional analysis from one molecule to cellular physiology'. The following symposium topics are listed: Transplantation medicine on degenerative neuronal diseases; Ubiquitin/ proteasome and neuronal diseases; Model animals in neuronal and psychological diseases; Polyglutamine diseases and translational defects; Molecular mechanism of synaptic transmission; Total understanding of brain and mind; Prion diseases and its

pathophysiology; Biochemical analysis of Alzheimer's diseases; Mechanism of axonal growth; Plasticity and molecular chemistry of pain; Development and differentiation of nervous system; and Neuronal diseases based on DNA repair. Special lectures are planned. All these planning are smoothly progressing.

At Niigata 2003, our society meeting is open more for not only domestic members, but also for neurochemists in Korea, China, Hong Kong, Australia, and other Asian-Pacific countries. We decided to present most of symposia and all posters in English. The papers are welcome to accept from members in APSN. Applications are also invited for the APSN Young Investigator Awards (e mail: jsn46@bri.niigata-u.ac.jp). In particularly, we hope that young investigators will visit Niigata and enjoy scientific and social communication.

4. The 2nd ISN-sponsored symposium 2003 in Japan

At the Niigata 2003 meeting, the second ISN-sponsored symposium in Japan is planed on the topic, "Research Frontiers of Neurodegenerative Disease". The program committee has nearly finalized the program for the ISN symposium. The final program is available on the meeting website (<http://brain.bri.niigata-u.ac.jp/~jsn46>). The following topics and speakers are suggested: Dr. E. E. Walker, Max-Planck-Institute, on Polyglutamine disease; Dr. V. M.-Y. Lee, University of Pennsylvania, on Alpha-synucleinopathies; and Dr. R. R. Kopito, Stanford University, on Aggresome.

5. Abstracts of 2002 ISN symposium in Japan

The talk of Dr. Robert Mailnow (Cold Spring Harbor Laboratory, New York) was 'AMPA receptor trafficking during plasticity'. The molecular mechanisms underlying synaptic plasticity are becoming much clearer. Activity-induced synaptic enhancement in hippocampus is accomplished at different developmental ages by delivery of AMPA receptors containing the different but homologous subunits GluR4 or GluR1. Here we show that activity-driven phosphorylation of GluR4 by PKA is necessary and sufficient for delivery of receptors to synapses during early development. Such phosphorylation relieves a retention interaction that, in the absence of synaptic activity, maintains GluR4-containing receptors away from the synapse. In contrast, phosphorylation of GluR1 by PKA is necessary but not sufficient for delivery of receptors to synapses in older animals. For GluR1, CaMKII is also necessary. Thus, a mechanism that mediates plasticity early in development becomes a gate for plasticity later in development. Increasing requirements over development may be one way that plasticity becomes more specific and also recalcitrant with age.

'Molecular genetics of the synapse formation and function' was presented by Dr. Masayoshi Mishina (Tokyo University). Cumulative evidence suggests that the dynamic structure-function relationship of the brain largely depends on the

experience-dependent development and functioning of synapses. Studies on the glutamate receptor (GluR) channel shed light on the molecular basis of neural input-dependent synapse dynamics. The N-methyl-D-aspartate (NMDA) subtype of GluR plays key roles in synaptic plasticity as a molecular coincidence detector. We revealed that the multiple GluR/NR2 subunits carrying the glutamate binding site are major determinants of the NMDA receptor molecular diversity. We examined the physiological roles of respective GluR subunits by gene targeting. Mutant mice lacking GluR1 showed increased thresholds for both hippocampal long-term potentiation and contextual learning. Heterozygous mutant mice with reduced GluR2 exhibited exaggerated startle responses to acoustic stimuli, indicating forebrain-specific GluR2 regulates the reflex because the primary circuit of the startle response is located in the lower brainstem. GluR2 mutant mice died shortly after birth and failed to form the whisker-related neural pattern (barrelettes) in the brainstem trigeminal complex. These results suggest that the molecular diversity of GluR underlies the diverse physiological roles of NMDA receptor channels. GluR is a novel member of the GluR channel family we found by molecular cloning. GluR2 is selectively localized in cerebellar Purkinje cells. Analyses of GluR2 mutant mice revealed that the GluR2 subunit plays essential roles in motor learning and cerebellar LTD and in refinement and maintenance of Purkinje cell synapses. GluR2 mutant mice failed to learn in the delay paradigm of eyeblink conditioning, but not in the trace paradigm. On the other hand, GluR1 mutant mice exhibited an impairment of learning in the trace paradigm, suggesting that neural substrates underlying eyeblink conditioning are different depending on the temporal overlap of the conditioned and unconditioned stimuli. Our molecular genetic studies on GluR channels imply that learning and memory may share a common molecular mechanism with synapse formation during brain development. Thus, elucidation of how the neural network is formed and modulated is essential to understand how the brain functions. To address this issue, we developed a novel strategy that enables visualization and manipulation of developing neurons *in vivo*. Olfactory and retinotectal projection systems in transparent zebrafish embryos are suitable to analyze axonal pathfinding and synptogenesis *in vivo*. Microinjection of an olfactory marker protein gene promoter-driven double-cassette vector directed the expression of both the dominant form of PKA and green fluorescent protein fused with the microtubule-associated protein tau in the same olfactory neurons. The dominant-negative form of PKA enhanced the turning of olfactory neuron axons in the olfactory placode, whereas the disturbance effect of the constitutively active form on the axonal pathfinding was prominent in the olfactory bulb. These results suggest that the switching of PKA signaling in developing olfactory sensory neurons is important for axonal pathfinding through the boundary between the olfactory placode and the olfactory bulb *in vivo*. We thus propose that the regulation of PKA signaling plays a key role in the long-distance axonal pathfinding through intermediate guideposts. Microinjection of the nicotinic acetylcholine receptor 3 (nAChR3) gene promoter-driven double-cassette vectors directed the expression of dominant-negative glycogen synthase kinase-3 (dnGSK-3) and EGFP in the same neurons. We found that expression of the dominant negative form of zebrafish GSK-3 suppressed the arborization field of RGC axon terminals in the tectum as

estimated by the reduction of arbor branch length and arbor areas. Furthermore, suppression of GSK-3 activity increased the size of vesicle-associated membrane protein 2-EGFP puncta in RGC axon terminals at the early stage of innervation to the tectum. These results suggest that GSK-3 regulates the arborization field and maturation of RGC axon terminals *in vivo*.

Dr. Nancy Y. Ip (Hong Kong University of Science and Technology, China) talked 'cdk5 - a new player at the neuromuscular synapse'. The development of the neuromuscular junction (NMJ) depends on reciprocal signaling between motor neurons and their target muscle fibers. A number of ligand-receptor interactions have been implicated to mediate these signaling cascades at the NMJ. For example, MuSK receptor is part of the receptor complex that mediates agrin-induced aggregation of AChRs while activation of ErbB receptors by neuregulin (NRG) regulates the synaptic transcription of acetylcholine receptors (AChR). My laboratory is interested in delineating the roles of receptor tyrosine kinases in the development and maintenance of the NMJ. During the course of our studies on the regulation of AChR expression, we have identified a new player, cyclin-dependent kinase 5 (Cdk5), which is involved in signaling at the NMJ. Cdk5, a member of the Cdk family, is involved in cellular functions that are not related to the regulation of cell cycle progression. While other members of the family associate with cyclins, the serine/threonine kinase activity of Cdk5 depends on its regulatory partners, p35 or p39, which are prominently expressed in the CNS. To date, the best-characterized role for Cdk5 is in regulating neuronal migration and axonal guidance as well as cytoskeletal dynamics. There is also increasing evidence that links Cdk5 activity to the regulation of membrane transport and dopamine signaling. In addition to its known functions in neuronal development, recent evidence suggests a novel role for Cdk5 in the modulation of post-synaptic functions. For example, Cdk5 can regulate the expression of AChR in muscle and NMDA receptors in cultured hippocampal neurons. In adult muscle, Cdk5 and its activators are localized to the NMJ and mediate the actions of NRG by associating with the ErbB receptors. Treatment of cultured myotubes with NRG increases the p35-associated Cdk5 kinase activity. While inhibition of Cdk5 activity attenuates NRG-induced ErbB activation and AChR transcription in cultured myotubes, overexpression of p35 results in an increase in AChR promoter activity in muscle. To further understand the regulation of Cdk5 and p35 in rat skeletal muscle during the formation of NMJ, we have examined whether the expression of p35 and Cdk5 can be regulated after nerve injury. While the transcripts of cdk5 and p35 increase in muscle after denervation, they exhibit different temporal profiles suggesting that these changes in gene transcription might be regulated by different mechanisms. Furthermore, the dependence of p35 mRNA expression on both trophic factors and electrical activity is similar to that observed for AChR. Recent analysis of the Cdk5 null mice in our laboratory reveals an interesting abnormal phenotype at the developing NMJ. Moreover, our studies on the identification of p35-interacting proteins would delineate the signaling molecules that act in concert with the Cdk5 complex to affect the process of synapse formation. Dr. Brian A. Hemmings did not submit his abstract.

'Excitatory amino acid transporters: New insights into structure, function and regulation' was presented by Dr. Susan G. Amara (Vollum Institute, Oregon Health & Science University). Neurotransmitter transporters present on the plasma membrane contribute to the clearance and recycling of neurotransmitters and have a profound impact on the extent of receptor activation that occurs during neuronal signaling. Work in our laboratory has focused on the structure, regulation and cellular physiology of sodium-dependent transporters for the biogenic amines and another family of sodium-dependent carriers for excitatory amino acid neurotransmitters. Five different human subtypes of excitatory amino acid transporters (EAATs1-5) and their homologues in several species have been identified. The glial carriers (EAAT1 and EAAT2) are abundantly expressed and are the major regulators of extracellular glutamate concentrations in the CNS. The neuronal isoforms of glutamate transporters (EAAT3 and EAAT4) are located predominantly outside the synapse, in the perisynaptic membrane and/or in spine regions in contact with glial cells. Thus, rather than being poised to influence synaptic glutamate concentrations, the neuronal carriers are positioned to regulate the amount of glutamate that escapes the synapse. This unique distribution implies that glutamate carriers may indirectly control the amount of glutamate available to activate metabotropic glutamate receptors and thus links them to the feedback control of excitability and neurotransmitter release. Although the EAATs are thought to regulate CNS concentrations of glutamate, several subtypes also possess a ligand-gated chloride channel activity that may regulate neuronal excitability and signaling in a different way. Structure-function studies support the notion that the binding sites for substrates, inhibitors, and co-transported ions, as well as, the transport pathway are formed from multiple domains that may or may not be adjacent to one another in the primary sequence. Efforts to link specific domains with various functional properties have demonstrated the importance of residues within highly conserved domains in the C-terminal half of the carrier for substrate binding and transport. Recently a cluster of residues in an extracellular helix has been identified where mutations or modifications disrupt transport, but preserve the substrate-activated anion current. These observations suggest that the substrate-gated chloride conductance is linked to a different pathway or conformation than that required for substrate transport. This lecture will consider some of the novel aspects of neurotransmitter transporter function and will present the results of molecular genetic, electrophysiological and cell biological approaches aimed at defining the relationships between neurotransmitter transporter structure, substrate transport, inhibitor binding and ion permeation.

6. Report on Annual Meeting 2002 at Sapporo

Sapporo 2002: The 45th meeting of the Society was held at the Karude 27 and Hotel Sapporo Garden Palace in Sapporo from 17th-19th July, 2002, organized by Prof. Yasuyuki Nomura, Department of Pharmacology, Hokkaido University Graduate School of Pharmaceutical Sciences. It was its own society's meeting and thereby the meeting had a strong and substantial program that sharply focused on specific

interest to all JSN members. It included four Plenary lectures: Dr. Nobutaka Hirokawa (Tokyo University, Graduate School of Medicine) on Neuronal function and intracellular transport, gene, structure, dynamics, function and diseases of kinesin superfamily motor proteins KIFs; Dr. Yoshihide Tsujimoto (Osaka University Graduate School of Medicine) on Molecular basis of apoptosis, Bcl-2 family proteins as life-or-death switch; Dr. David S. Bredt (University of California San Francisco, USA) on Synaptic plasticity regulated by stargazin and PSD-95; and Dr. Peter Dunkley (University of Newcastle, Australia) on Tyrosine hydroxylase phosphorylation. Four invited lectures given are by Dr. Ken-ichi Honnma (Hokkaido University) on Molecular mechanism and hierarchical structure of circadian clock; Dr. Yutaka Kirino (Tokyo University) on Molecular and neuronal mechanism of eyeblink conditioning studied with mutant mice; Dr. Takeharu Nishimoto (Kyushu University Graduate School of Medicine) on Regulation on nucleocytoplasmic transport and cell proliferation by Ran and GTPase; and Dr. Yoshinori Ohsumi (National Institute for Basic Biology) on Bulk protein degradation, molecular dissection of autophagy..

The society's symposium opened for citizen was on 'Molecular probing of human mind and behavior'. This symposium was organized by Prof. Takeda, Osaka University and Prof. Nishikawa, Tokyo Medical Dental University. Topics were the following: Understanding human behavior through gene targeting animals by Dr. Wada; Modulation of brain function elicited by intracellular signal transduction, by Dr. Saito; How behavior changes along human development, by Dr. Watanabe; To understand emotion, cognition, and behavior, by Dr. Eniwa; New aspects in the research field for schizophrenia, by Dr. Nishikawa; and Mood disturbance, by Dr. Koyama.

We invited 4 members (3 Chinese and 1 Korean) of APSN and supported their traveling expenses for attending the 45th meeting. They presented excellent posters and could discuss with members of JSN, which was nice. From this first experience JSN expects for many APSN members to attend and promote communication in future.

7. Young Investigator Award 2002

We recognized research achievements of promising young neurochemists, who were younger than 38 years of age at the time of the annual meeting. Four researchers were chosen out of many candidates by the award committee and the four persons presented a 30-min lecture in an open session. The Gold medal winner was Dr. Taiichi Katayama, Osaka University Graduate School of Medicine for his study of Disturbed activation of endoplasmic reticulum stress transducers by familial Alzheimer's disease-linked presenilin 1 mutations. The silver medals were awarded to Dr. Masashi Katsura, Kawasaki Medical University, on Functional relationship between diazepam binding inhibitor (DBI) expression and L-type voltage-dependent calcium channels in drug dependence, and Dr. Eiji Watanabe, National Institute for Basic Biology, Na⁺ channel (*Na_v*) involved in CNS sodium-level sensing; and Dr. Nobuyuki Fukushima

(Hokkaido University Graduate School of Medicine) on Lysophosphatidic acid signaling in the regulation of neuronal cell dynamics. The award was handed by the award committee chair Kazuhiro Ikenaka.

(Written by the Chairman of International Affair committee, Haruhiro Higashida, Kanazawa University. Haruhiro@med.kanazawa-u.ac.jp)