29th GÖTTINGEN NEUROBIOLOGY CONFERENCE

The Neurosciences

From Basic Research to Therapy

The Francfort anatomist Johann Christian Gustav Lucae. Photography, ca. 1865

Neuroscience at Center Stage: From Basic Research to Therapy

We welcome you to Göttingen and the 5th Congress of the German Neuroscience Society that is also the 29th Göttingen Neurobiology Conference. We hope that this Conference will again be instrumental in stimulating the development of Neuroscience in Germany and in strengthening its position in the field of science politics. The Conference in Göttingen has become particularly attractive for young neuroscientists. It provides them, within pleasant surroundings, with the opportunity to experience the entire breadth of the field, from basic neuroscience to clinical applications. Thus we express a particular welcome to the younger neuroscientists. We have received almost 1000 posters and most of them will be presented by them. We also welcome our foreign guests and thank them for their efforts to contribute to the scientific success of the Conference.

By submitting proposals for symposia the membership of the German Neuroscience Society took a very active part in structuring the Conference. Of the 40 proposals received, the Program Committee had to chose 24 in a painstaking process. The Committee has made every effort to make the meeting attractive to as many neuroscientists as possible and to visualize the pace of the field. We are happy to house a symposium illustrating examples of the German-Israeli cooperation in neuroscience. Seven invited plenary lectures by internationally highly renowned scientists will highlight individual topics of interest for the entire neuroscience community. This will be flanked by lectures from two young scientists who have been awarded one of the two new prices of the German Neuroscience Society: the Novartis price being awarded for excellent and innovative work in the field of neuroscience and the T.I.L.L. Photonics Technology price for excellent achievements in developing novel techniques in neuroscience. Four satellite symposia provide insight into selected topics and further strengthen the attractivity of the Conference.

The scientific program presents new developments in basic and comparative neuroscience equally well as its applications in the field of pharmacology and clinical therapy. It illustrates both the importance of interdisciplinary work and the great impact of basic research for later application in industry or in the clinics. Today, the importance of scientific contributions are often solely weighed according to their immediate commercial or therapeutic value. Only an extensive promotion of basic science disciplines will provide the potential for entering into hitherto unknown fields of future application.

Novel developments in neuroscience have received considerable public attention and provoked intense political discussion regarding their ethical justification. This concerns in particular the import and application of embryonic stem cells for developing novel strategies in brain therapy. A compromise has been reached that allows the import of existing cell lines and thus the pursue of experiments to evaluate the potential of this therapeutic approach. The progress in cognitive neuroscience has not only opened new avenues for clinical application. It has a considerable impact on the understanding of the human brain and the control of its cognitive functions. It lead neuroscientists to discuss the consequences for our self understanding and the relationship between psyche and the material nervous system. This has become a challenge to many in the humanities and provoked passionate debate. The Conference in Göttingen will highlight recent developments in either field.

The format of the Conference publications has been altered. This program book and an abstract volume will be provided to the delegates. Full articles covering the field of the

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plenary lectures will no longer be published since – due to internet facilities – topical reviews published in review journals will be more easily accessible from there. It also needs to be mentioned that this year's Conference marks the tenth anniversary of the German Neuroscience Society. The Conferences of the Society are now held only every second year. In between, German neuroscientists are invited to attend the Forum of the European Neuroscience Societies. The next one will take place in Lisbon in 2004. We would also like to announce that the European Forum Meeting 2006 will be held in Vienna as a joined undertaking of the Austrian and German Neuroscience Societies.
We wish to thank all companies that supported the Conference and in particular the Deutsche Forschungsgemeinschaft whose generous support enabled us to invite a considerable number of foreign scientists. Our warmest thanks go to the many student volunteers in Göttingen who helped with the homepage, editing the printed program and abstract book and who also will be responsible for most of the work during the meeting, helping you to enjoy the Conference.
We wish you a rewarding and pleasant time in Göttingen.

Prof. Dr. Norbert Elsner

Prof. Dr. Herbert Zimmermann

29th Göttingen Neurobiology Conference
5th Congress of the German Neuroscience Society
Wednesday, June 11 – Sunday, June 15, 2003
Time Schedule

Wednesday	Time	Thursday	Friday	Saturday	Sunday
Satellite Symposia	08.00–09.00			Hanging of Posters II	
	09.00–10.00	**Symposia 1–6 Symposium C**	**Symposia 13–18 Symposium C**	**Brose**	**Sachse/Nieder**
	10.00–11.00			Poster II/A	Poster II/A
Symposium A 16.00–22.00	11.00–12.00			Poster II/B	Poster II/B
Symposium B 09.00–17.00	12.00–13.00	Hanging of Posters I		Meeting NWG	**Nottebohm**
	13.00–14.00	Poster I/A	Poster I/A	Poster II/A	
Symposium C 15.30–19.00	14.00–15.00	Poster I/B	Poster I/B	Poster II/B	
	15.00–16.00	14.30 **Sakmann**	**Hagner**	**Symposia 19–24 Symposium C**	
Symposium D 13.00–16.00	16.00–17.00	**Symposia 7–12 Symposium C**	Poster I/A		
	17.00–18.00		Poster I/B		
	18.00–19.00		Buffet	Buffet	
	19.00–20.00	Buffet	**Kuhl**	**Altenmüller**	
	20.00–21.00	**Frahm**			

Presentation time of posters Posters I (No. 7–634): Thursday 12 – Friday 18.00. Posters II (No. 635–1222): Saturday 8.00 – Sunday 12.00. A/B: Demonstration of even/odd numbered posters.

General Information

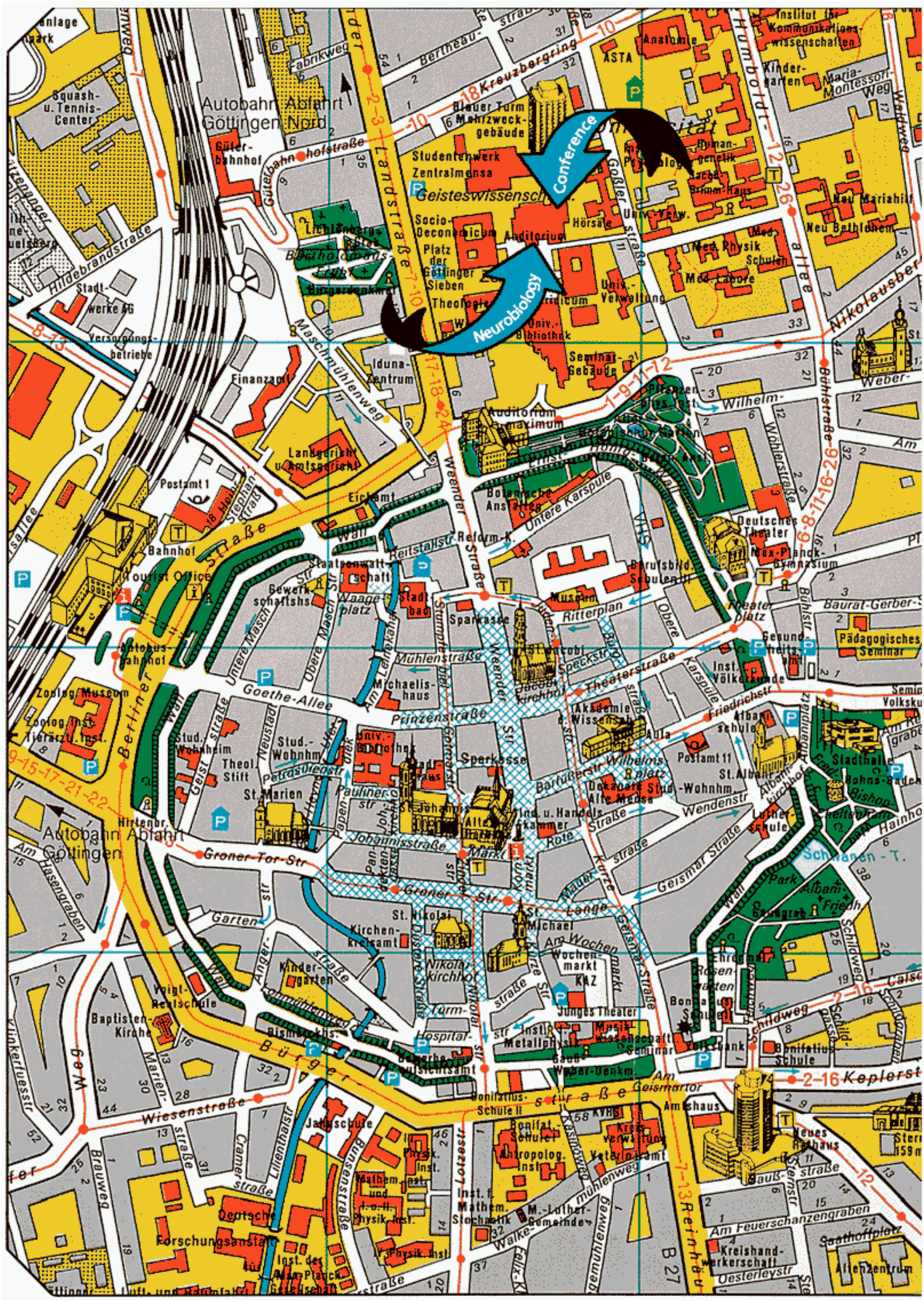
The conference will take place in the Central Lecture Hall Building (Zentrales Hörsaalgebäude – locally known as „ZHG“) of the University. You may use the adjacent seminar building as a point of orientation. Because of the colour of its sun protection windows, it is known as the “Blue Tower”. Göttingen is accessible from all directions within a few hours by way of the intercity railway network, therefore, we recommend travelling by train. There are only few parking spaces near the conference centre, and these are very expensive. A somewhat more economical alternative is the parking building at the corner of Goßlerstraße and Kreuzbergring (see city map). Regarding overnight accommodation it is recommended that reservations be made in good time, because the number of inexpensive rooms is limited. Please consult the Tourist Office, Altes Rathaus, D-37073 Göttingen (Phone 0551/49980–0; Fax 0551/49980–10; E-mail: tourismus@goettingen.de; Website: www.goettingen-tourismus.de; online booking both in German and in English).

Posters will be presented in two shifts – the first half from Thursday noon until Friday evening, the second from Saturday morning until Sunday noon. The format is 1 x 1 m and both sides of the poster screens will be in use, which could mean that some posters belonging to one group are hung on the front and back of a screen, rather than side-by-side. If this should happen to you, please work out a swap with the group beside you. Despite the fact that the posters will be hung up on different days, there is still only a limited amount of space, and the ceilings are very low in some places. We, therefore, urge you to refrain from smoking in the whole poster area.

Regarding the facilities for projection during the symposia, it should be said that in all lecture rooms PowerPoint projectors as well as overhead and slide projectors are available, but there are not two of the latter. We therefore have to ask you to present your talk without double projections. Furthermore, we must point out that only one video projector is available, so that when seven symposia are going on in parallel, there might be problems. In any case, if you have special needs regarding projection, please, let us know by May 15, 2003 at the latest (contact Prof. Dr. Andreas Stumpner, E-mail: astumpn@gwdg.de). All such requests will be collected up to that date, after which you will be informed about what is possible and what is not.

Because the costs have increased disproportionally again, we had to increase the fees moderately. If you did not make use of the low rates by early registration we ask you to pay the following rates at the conference reception: 90 Euro for students, 130 Euro for non-student members of the German Neuroscience Society, and 170 Euro for all other participants. As in previous years, this is a flat rate covering the programme, the conference volume, the organisation, and all extra costs. As has been already announced, due to fiscal laws the costs for coffee, tea, pastry, cold drinks and three buffets (approximately 35 Euro) have to be receipted separately. The above mentioned rates in no way cover the costs of the conference and are only possible because of donations from companies and, above all, because of the honorary work done by numerous Göttingen students. We would like to ask you for a donation during the conference for them.

We look forward to your participation in the Göttingen Conference of the German Neuroscience Society and hope that you like the programme.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Obituary Werner Rathmayer (1937–2003)

Werner Rathmayer (1937–2003)

Colleagues, students, and his many friends were dismayed at the news of the sudden death of Prof. Werner Rathmayer, University of Konstanz, on the way to his lecture. A man of impressive stature, energy and activity, he had many plans for his final two years of research and teaching at the university as well as for his life as an emeritus.
Werner Rathmayer studied biology, chemistry and geology at the University of München and completed his teaching and doctoral examinations in 1962 with the thesis: „Das Paralysierungsproblem beim Bienenwolf *Philantus* (The problem of paralysis in the bee wolf *Philanthus*) under the supervision of Prof. Martin Lindauer. Supported by a grant from the Deutsche Forschungsgemeinschaft (German Research Foundation), he joined the laboratory of Prof. Ernst Florey at the University of Washington, Seattle, USA. In 1964 he returned to the group of Prof. Lindauer who had meanwhile moved to the University of Frankfurt. There he was an assistant professor until 1968 when he finished his habilitation with a thesis on the control of wolf spider legs and muscles. In the same year he received his professorship at the University of Konstanz, where he re-

mained, perhaps revealing a deeply rooted love for the beautiful lake on whose Bavarian shore he was born in 1937.
His scientific work centres on the functional importance of excitatory and inhibitory synaptic transmission, the role of neuromodulators, and the significance of different muscle fibres for the performance of muscles, in particular those of crustaceans. Starting from a behavioural question, he wanted to reveal the underlying cellular mechanism, and thus he used sophisticated electrophysiological as well as biochemical methods to achieve his goals. To mention only one example, he provided a beautiful explanation for the behavioural role of inhibitory neurones in blocking those subsets of muscle fibres which are not appropriate for a particular motor task. He is also noted for his work on animal toxins and their use by predators to block neuromuscular transmission. Werner Rathmayer was also a great zoologist, one who really loved animals and nature, and who never stopped enjoying going on excursions and hikes. He was a fantastic ornithologist. Going on a bird watching trip with him was pure joy as his enthusiasm infected just everyone.
He was a gifted lecturer and had all the features that make an excellent teacher: he motivated students and colleagues equally and was always enthusiastic about his subject. He could even explain difficult scientific matters, and in such a way that one understood the underlying problems. As a true Bavarian, he had firm beliefs and opinions, but yet he was very liberal and accepting of the ideas of others. It was great to be locked in a fierce discussion with him, that usually ended with enjoying a friendly glass of excellent wine together. He was an outstanding head for his laboratory group, providing space and support in every respect to those who successfully followed their own ideas, and providing guidance and advice to those who needed the help. Being exposed to his personality had a great impact on developing one's own character.
He played an important role for German, and in later years for European, neurobiology. Numerous people (some of whom may not even know it) owe him much for his long service as a principal reviewer for the Deutsche Forschungsgemeinschaft. His advice was often sought by many, and he always provided it with an eye to the greatest advantage for the cause of zoology. He was a great defender and advocate of fundamental science, and all who remember discussions with him admire him for his great passion. International collaboration and scientific exchange, in particular with Israel, were very important for him. He believed that international travel and collaboration educates people and enriches their science. His own rich life ended abruptly and unforeseeably, but he will always be remembered. Our condolences to his wife Martina, who has been his partner since his time as a postdoc.

Hans-Joachim Pflüger, Berlin

Georg-August-Universität Göttingen
29th Göttingen Neurobiology Conference
5th Meeting of the German Neuroscience Society

Chair: Herbert Zimmermann and Norbert Elsner

Wednesday, June 11th 2003
Satellite Symposia:

9.00–17.00 Satellite Symposium B in Lecture Hall 10
Chair: M. Bähr (Göttingen) and H. W. Müller (Düsseldorf)
Molecular Basis of Neural Repair Mechanisms

13.00–16.00 Satellite Symposium D in Lecture Hall 7
Chair: Klaus Benndorf (Jena) and Heinrich Terlau (Göttingen)
Novel properties of channels

15.30–19.00 Satellite Symposium C in Lecture Hall 8
Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt a. M.)
2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen

16.00–22.00 Satellite Symposium A in Lecture Hall 9
Chair: Günter Ehret (Ulm), Joachim Kirsch (Heidelberg) and Albert Ludolph (Ulm)
Inhibition: Molecules , Mechanisms, Functions

Thursday, June 12th 2003

8.30–17.00 Registration of participants in the foyer in front of Lecture Hall 3

8.30–12.30 Satellite Symposium C in Lecture Hall 8
Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt am Main)
2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen

9.00–12.00 Symposium 1 in Lecture Hall 7, Chair: N. Troje (Bochum) and M. Bach (Freiburg i. Brsg.)
Adaptation: the psychophysicist's microelectrode

9.00–12.00 Symposium 2 in Lecture Hall 9, Chair: U. Rose (Ulm) and S. Anton (Lund, Sweden)
Juvenile hormone as a mediator of behavioural plasticity in adult insects

9.00–12.00 Symposium 3 Lecture Hall 104, Chair: J. Mey (Aachen) and H. Siebert (Göttingen)
Cytokines as mediators of neuroglial interactions

9.00–12.00 Symposium 4 in Lecture Hall 10, Chair: J. Schulz (Tübingen) and Ch. Haass (München)
Transgenic animal models of neurodegenerative diseases

Thursday, June 12th 2003

9.00–12.00	Symposium 5 in Lecture Hall 11, Chair: Th. Berger (Bern) and M. Larkum (Heidelberg) **Signal integration in dendrites**
9.00–12.00	Symposium 6 in Lecture Hall 105, Chair: A. Reichenbach (Leipzig) and Ch. Steinhäuser (Bonn) **Neuronal death and neuroprotection: the role of glial cells**
12.00–12.30	Hanging of posters no. 7–634
12.30–13.30	Demonstration of posters no. 7–634 (even numbers)
13.30–14.30	Demonstration of posters no. 7–634 (odd numbers)
14.30–16.00	Opening of the Conference in Lecture Hall 11 by the President of the University; Roger-Eckert-Lecture, Chair: E. Neher (Göttingen) *Bert Sakmann (Heidelberg)* **Cortical microcircuits and their plasticity**
16.00–19.30	Satellite Symposium C in Lecture Hall 8 Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt am Main) **2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen**
16.00–19.00	Symposium 7 in Lecture Hall 7, Chair: U. Havemann-Reinecke (Göttingen) and V. Höllt (Magdeburg) **Drug addiction: mechanisms and therapy**
16.00–19.00	Symposium 8 in Lecture Hall 9, Chair: D. Heck (Freiburg i. Brsg.) and F. Sultan (Tübingen) **Precise timing in the brain: linking neuronal activity and behavior**
16.00–19.00	Symposium 9 in Lecture Hall 104, Chair: K. Krieglstein (Göttingen) **Ontogenetic cell death in the nervous system**
16.00–19.00	Symposium 10 in Lecture Hall 10, Chair: C. Duch and H.-J. Pflüger (Berlin) **Insect neural and motor systems: from development to function and mechanics**
16.00–19.00	Symposium 11 in Lecture Hall 11, Chair: G. Kempermann (Berlin) **Adult neurogenesis**
16.00–19.00	Symposium 12 in Lecture Hall 105, Chair: A. K. Engel (Hamburg) and Ch. E. Elger (Bonn) **Invasive recording from the human brain: linking clinical applications with neurobiological research**
19.00–20.00	Cold Buffet in the Lecture Hall Foyer
20.00–21.00	Plenary Lecture in Lecture Hall 11, Chair: H. Scheich (Magdeburg) *Jens Frahm (Göttingen)* Magnetic resonance neuroimaging: from anatomy to function

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Friday, June 13th 2003

9.00–11.00	Satellite Symposium C in Lecture Hall 8 Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt am Main) **2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen**
9.00–12.00	Symposium 13 in Lecture Hall 7, Chair: W. Magerl (Mainz) and R.-D.-Treede (Mainz) **Long-term potentiation and long-term depression of nociceptive CNS processing**
9.00–12.00	Symposium 14 in Lecture Hall 9, Chair: R. Heinrich (Göttingen) and E. A. Kravitz (Boston, USA) **Towards a molecular understanding of behavior**
9.00–12.00	Symposium 15 in Lecture Hall 104, Chair: P. Skiebe (Berlin) and S. Kreissl (Konstanz) **Peptide co-transmitters in identified neurons**
9.00–12.00	Symposium 16 in Lecture Hall 10, Chair: I. Neumann (Regensburg) and K. Braun (Magdeburg) **Early environmental programming: molecular, neuroanatomical, neuroendocrine and behavioural effects**
9.00–12.00	Symposium 17 in Lecture Hall 11, Chair: A. Konnerth and J. Hartmann (München) **New forms of cerebellar signaling**
9.00–12.00	Symposium 18 in Lecture Hall 105, Chair: B. Gaese (Frankfurt) and H. Luksch (Aachen) **Complex sensory processing in the vertebrate midbrain**
12.00–13.00	Lunch break
13.00–14.00	Demonstration of posters no. 7–634 (even numbers)
14.00–15.00	Demonstration of posters no. 7–634 (odd numbers)
15.00–16.00	Plenary Lecture in Lecture Hall 11, Chair: N. Rupke (Göttingen) *Michael Hagner (Berlin)* **Enchanted looms: on brains and scientists in the 19th and 20th centuries**
16.00–18.30	Satellite Symposium C in Lecture Hall 8 Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt am Main) **2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen**
16.00–17.00	Demonstration of posters no. 7–634 (even numbers)
17.00–18.00	Demonstration of posters no. 7–634 (odd numbers)
18.00–19.00	Cold Buffet in the Lecture Hall Foyer
19.00–20.00	Plenary Lecture in Lecture Hall 11, Chair: R. Menzel (Berlin) *Dietmar Kuhl (Berlin)* **Learning about activity-dependent genes**

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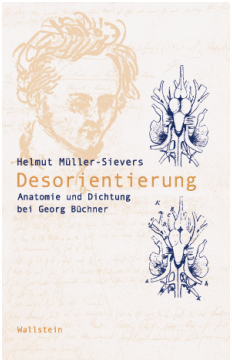

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Saturday, June 14th 2003

8.00–9.00	Hanging of posters no. 635-1222
9.00–10.00	Plenary Lecture in Lecture Hall 11, Chair: R. Jahn (Göttingen) *Nils Brose (Göttingen)* **Presynaptic plasticity: dynamic regulation of neurotransmitter release at active zones**
10.00–12.00	Satellite Symposium C in Lecture Hall 8 Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt am Main) **2. International TMS und tDCS Symposium Göttingen**
10.00–11.00	Demonstration of posters no. 635-1222 (even numbers)
11.00–12.00	Demonstration of posters no. 635-1222 (odd numbers)
12.00–13.00	Meeting of the German Neuroscience Society in Lecture Hall 9
13.00–14.00	Demonstration of posters no. 635-1222 (even numbers)
14.00–15.00	Demonstration of posters no. 635-1222 (odd numbers)
15.00–18.30	Satellite Symposium C in Lecture Hall 8 Chair: W. Paulus, F. Tergau, M. Nitsche (Göttingen) and U. Ziemann (Frankfurt am Main) **2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen**
15.00–18.00	Symposium 19 in Lecture Hall 7, Chair: D. M. Yilmazer-Hanke and O. Stork (Magdeburg) **Function and dysfunction of the amygdala: fear and epilepsy**
15.00–18.00	Symposium 20 in Lecture Hall 9, Chair: V. Leßmann (Mainz) and K. Gottmann (Bochum) **Transsynaptic signalling at central glutamatergic synapses**
15.00–18.00	Symposium 21 in Lecture Hall 104, Chair: H. Neumann (Göttingen) and M. Bähr (Göttingen) **Molecular basis of axonal damage in inflammatory and degenerative CNS diseases**
15.00–18.00	Symposium 22 in Lecture Hall 10, Chair: H. Ehrenreich and E. Rüther (Göttingen) **Neurotrauma: a trigger for schizophrenia?**
15.00–18.00	Symposium 23 in Lecture Hall 11, Chair: B. Sakmann (Heidelberg) **German-Israeli cooperation in neuroscience**
15.00–18.00	Symposium 24 in Lecture Hall 105, Chair: S. Treue (Göttingen) **Attentional modulation of sensory information processing in man and monkey**
18.00–19.00	Cold Buffet in the Lecture Hall Foyer

Saturday, June 14th 2003

19.00–20.00 Otto-Creutzfeldt-Lecture in Lecture Hall 11, Chair: N. Elsner (Göttingen)

Eckart O. Altenmüller (Hannover)
From Laetoli to Carnegie: musician's brains and neuroplasticity

Sunday, June 15th 2003

9.00–10.00 NeuroFutureLectures in Lecture Hall 11, Chair: T. Bonhoeffer (Martinsried) and K. Braun (Magdeburg)

Silke Sachse, New York (TILL Photonics Award)
Odor processing in the honeybee antennal lobe

Andreas Nieder, Cambridge, Mass. (Novartis Award)
Of neurons and numbers: How the primate cortex encodes numerical information

10.00–11.00 Demonstration of posters no. 635-1222 (even numbers)

11.00–12.00 Demonstration of posters no. 635-1222 (odd numbers)

12.00–13.00 Ernst-Florey-Lecture in Lecture Hall 11, Chair: H. Zimmermann (Frankfurt am Main)

Fernando Nottebohm (Millbrook, USA)
Neuronal replacement in adult brain

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The Neurowissenschaftliche Gesellschat e.V. gratefully acknowledge the financial contribution of TILL Photonics GmbH and Novartis Pharma GmbH. The two prizes will be awarded during the Annual General Assembly of the Neurowissenschaftliche Gesellschaft on Saturday, June 14, 2003 (12.00 h).

Both prize winners will give a lecture on Sunday, June 15, 2003 at 9.30 h.

Contact:

TILL Photonics GmbH
Lochhamer Schlag 19
D-82166 Grafelfing
Phone: +49 (0)89 895662-0
Fax: +49 (0)89 895662-101
info@till-photonics.com
www.till-photonics.com

Novartis Pharma GmbH
Roonstraße 25
90429 Nürnberg
Phone: +49 (0)911 273 - 0
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novartis.online@pharma.novartis.com
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Aktivitäten

Neuroforum
Die Zeitschrift Neuroforum erscheint vierteljährlich. Die Miglieder erhalten sie kostenlos. Neuroforum informiert über Themen, Trends, Fortschritte, neue Methoden, Forschungsschwerpunkte, Fördermöglichkeiten, Stellenangebote und Ausschreibungen.

Methodenkurse
Mehrmals jährlich werden insbesondere für Studenten, Doktoranden und junge Wissenschaftler Methodenkurse angeboten.

Info und Stellenmarkt
In regelmäßigen Abständen werden an alle Mitglieder mit eMail-Zugang Rund-eMails mit Informationen zu Drittmitteln, Stipendien u. Stellenanzeigen u.a. verschickt.

Homepage
Die Homepage informiert über Kongresse, bietet Links zu Institutionen, Fördereinrichtungen, neurowissenschaftlichen Zeitschriften, informiert über Bezugsquellen und Produkte und die Aktivitäten der Gesellschaft (http://nwg.glia.mdc-berlin.de).

Kongresse
Mit der Veranstaltung und Förderung von neurowissenschaftlichen Kongressen und Tagungen verfolgt die Gesellschaft ihr interdisziplinäres Konzept weiter. Neurowissenschaftler aller Fachrichtungen aus Forschung und Industrie sind zu einem lebendigen und fruchtbaren Meinungsaustausch aufgefordert.

Stipendien
Die NWG stellt Stipendien für Studenten, Doktoranden und junge Wissenschaftler für die Teilnahme an eigenen wie auch auswärtigen Kongressen zur Verfügung.

Förderpreise
Die NWG vergibt jährlich den Novartis Preis sowie den T.I.L.L. Photonics Technologie-Preis an Nachwuchswissenschaftler. Jeder Preise ist mit 2.500,-- Euro dotiert.

Freier Zugang zu EJN online
Die Mitglieder haben kostenlosen Zugang zur online-Version des European Journal of Neuroscience.

Lehrerfortbildung
Bundesweit werden Fortbildungsveranstaltungen für Lehrer der gymnasialen Oberstufe angeboten.

Slots für das SfN Meeting
Die NWG vergibt jedes Jahr für das Meeting der amerikanischen Society for Neuroscience sog. „society sponsored abstract slots“. Mitglieder der NWG zahlen die selbe reduzierte Tagungsgebühr beim SfN Meeting wie die SfN Mitglieder.

Ziele

Die Neurowissenschaftliche Gesellschaft e.V. hat sich zum Ziel gesetzt, die Neurowissenschaften in Forschung und Lehre zu fördern und in allen ihren Teilbereichen im In- und Ausland zu repräsentieren. Sie versucht, forschungspolitische Schwerpunkte mit neurowissenschaftlicher Thematik zu setzen und neue Konzepte anzuregen.

Sie steht in Kontakt mit innerdeutschen Fördereinrichtungen und privaten Stiftungen. Sie unterstützt die neurowissenschaftliche Ausrichtung der Förderprogramme der Europäischen Gemeinschaft. Sie fördert die Kontakte zur Industrie.

Sie tritt für die Etablierung eines interdisziplinären neurowissenschaftlichen Ausbildungskonzepts ein.

Sie verfolgt ausschließlich gemeinnützige Zwecke.

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Die Neurowissenschaftliche Gesellschaft e.V.

vertritt deutsche Neurowissenschaftler in der IBRO.

ist Gründungsmitglied der Federation of European Neuroscience Societies (FENS) und vertritt die nationalen Interessen in der FENS.

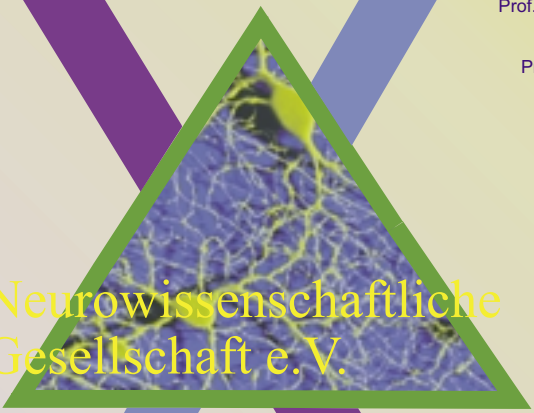
ist kooperatives Mitglied des Verbandes Deutscher Biologen (vdbiol).

Die Deutsche Gesellschaft für Neurologie ist förderndes Mitglied der Neurowissenschaftlichen Gesellschaft.

Mitgliedschaft

Mitglied der Gesellschaft kann werden, wer auf einem Gebiet der Neurowissenschaften oder in verwandten Fächern tätig ist. Das Aufnahmegesuch ist mit der Befürwortung von zwei Mitgliedern der Gesellschaft an die Geschäftsstelle zu richten, über die Aufnahme entscheidet der Vorstand.

Der Mitgliedsbeitrag für Studenten beträgt 25 Euro, für Vollmitglieder 50 Euro pro Jahr.



Neurowissenschaftliche Gesellschaft e.V.

Vorstand der Amtsperiode 2003 - 2005

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Fax: 030 9406 3819
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Introductory Remarks to the Satellite Symposium A:

Inhibition: molecules, mechanisms, functions

Symposium of the Neurocenter, University of Ulm, Germany

Günter Ehret, Joachim Kirsch and Albert Ludolph*

Functions of the nervous systems of animals and humans emerge from the mutual antagonism of excitation and inhibition including modulatory influences of neurons with excitatory and inhibitory net effects. Inhibition may attenuate, filter, and shape excitatory states and excitatory outputs of neurons both in magnitude and timing.

Inhibitory actions can be studied at many levels of neural systems. Starting with the cellular level, inhibitory effects are mediated by receptors of neurotransmitters and neuromodulators and carried out directly by ion-channel activities and indirectly by intracellular signaling cascades. Many genes code, in mostly unknown ways, for subunits of receptors and ion channels, so that it is important to know the differential pattern of differential gene expression in neurons in order to predict functions and malfunctions of inhibition at the cellular level. At the level of neural networks, inhibition is involved, for example, a) in setting thresholds, general levels of activity and the exact timing of excitatory actions of neurons and the whole network, b) in differentially shifting activity to certain neural subpopulations of the neural network and gating the network output pattern via certain pathways, c) in generating oscillations, rhythms and spatial maps of graded activity. At the level of system functions such as sensory processing and perception, the coordination of movements and control of emotions, inhibition becomes most evident whenever it is impaired so that the normal balance between excitation and inhibition is disturbed and perceptional, movement, cognitive and emotional control is out of order, giving rise to abnormal and pathological states.

One goal of this symposium is to sensitize all those who are working on neurons and brains, to consider inhibition in their research as a pervasive strategy of nervous systems evolved to ensure an optimum of function and functional adaptability. The expert speakers of the symposium will present examples of inhibitory regulation and regulation by inhibition from all the levels mentioned above including invertebrate, and vertebrate species (humans inclusive).

*Now, at the University of Heidelberg, Germany

SATELLITE SYMPOSIUM A

Wednesday, June 11th 2003, 16.00–20.05, Lecture Hall 9

Inhibition: molecules, mechanisms, functions

Chair: Albert Ludolph, Ulm (Germany)

16.00 *Günter Ehret, Ulm (Germany)*
Welcome

16.05 *Joachim Kirsch, Heidelberg (Germany)*
Molecular determinants of inhibitory synapses

16.25 *Hannah Monyer, Heidelberg (Germany)*
Chemical and electrical synapses at GABAergic interneurons and significance thereof for synchronous network activity.

17.05 *Hanns Möhler, Zürich (Switzerland)*
$GABA_A$-receptor subtypes as targets for antiepileptic drugs

17.45 *Holger Lerche, Ulm (Germany)*
Impaired inhibition as a pathophysiological mechanism in idiopathic epilepsies

18.05 Coffee Break

Chair: Günter Ehret, Ulm (Germany)

18.25 *Harald Wolf, Ulm (Germany)*
Why inhibition of muscles makes them move faster: arthropod common inhibitors

18.45 *Bendedikt Grothe, München (Germany)*
Glycinergic inhibition in audition: specific functions in temporal processing

19.25 *Heiko Neumann, Ulm (Germany)*
Extra-classical receptive field responses – balanced inhibition and excitation in visual Gestalt organization

19.45 *Manfred Spitzer, Ulm (Germany)*
Inhibition and the prefrontal cortex: a central mechanism for cognitive and emotional control

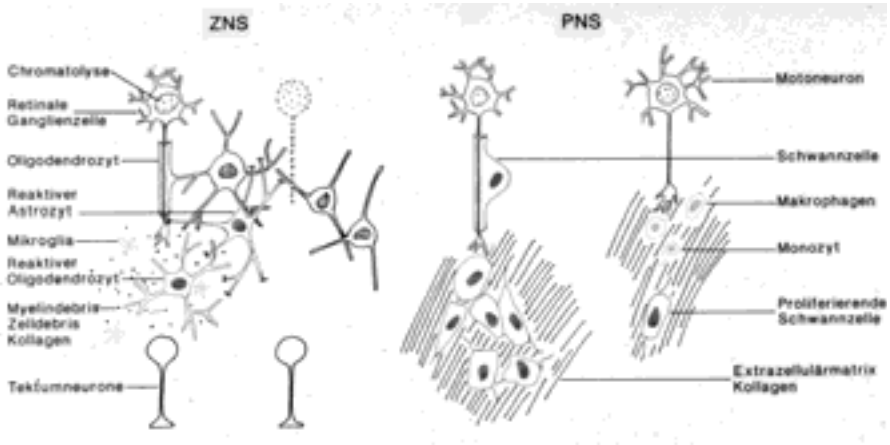
20.05 **Reception and Poster Session**

Introductory Remarks to Satellite Symposium B:

Molecular basis of neural repair mechanisms

Mathias Bähr and Hans-Werner Müller

The adult central nervous system (CNS) of mammals possesses only little ability for self-repair after an injury, that is, most parts of the CNS cannot generate new neurons and do not regenerate axons. Therefore, repair of damaged functional circuits is severely limited. This is in contrast to the peripheral nervous system (PNS) or the immature mammalian CNS, where a successful regeneration is possible. At present no therapies are available that can be applied to human patients. However, from a basic science perspective many recent advances in this field have been made, which provide a solid foundation for progress towards the development of effective treatments. The apparent lack of the adult mammalian CNS to regenerate occurs albeit the inherent ability of CNS axons to re-initiate axon growth at least to some extent. Work of the last decades has led to the characterization of factors associated with this inability such as lack of growth-encouraging factors, the inability to express the full set of molecules required for outgrowth and guidance, scar formation at the site of injury, the presence of growth inhibitory molecules, and also the degeneration of axotomized neurons which will be discussed further in this symposium. At present, various approaches are being investigated aimed at overcoming these obstacles, including the use of neutralizing monoclonal antibodies against growth-inhibiting activities, interference with signalling pathways activated by inhibitory molecules, prevention/removal of the scar tissue, blocking of apoptosis, implantation of growth-promoting or stem cells or the expression of growth promoting proteins via several routes including vector based strategies.
With an increased understanding of the factors contributing to the inhibition of regeneration and therapeutically targeting, the possibility arises that finally regeneration of axons and topographically correct re-innervation of their target tissue may be achieved.



SATELLITE SYMPOSIUM B

Wednesday, June 11, 2003, 9.00–17.00 Lecture Hall 10

Molecular basis of neural repair mechanisms

Chair: Mathias Bähr, Göttingen

Neuroprotection

9.00 *Jörg Schulz, Tübingen*
Neuroprotection by the inhibition of apoptosis

9.20 *Pierluigi Nicotera, London (UK)*
Molecular switches in neuronal cell death

9.40 *Ulrich Dirnagl, Berli*
Neuroprotection by ischemic preconditioning

10.00 *Dan Lindholm, Upsala (Sweden)*
Role of inhibitory apoptosis proteins (IAPs) in neurodegenerative disease

10.20 **Coffee – Tea**

Regenerative Axon Growth and Axon Guidance

10.50 *Alain Chédotal, Paris (France)*
Slits and semaphorins, not just axon guidance molecules

11.10 *Claudia Stürmer, Konstanz*
Reggie and Nogo functions in neurite growth

11.30 *Joost Verhaagen, Amsterdam (Netherland)*
Chemorepulsive semaphorins in neuroregeneration

11.50 **Lunch Break**

Chair: H.W. Müller, Düsseldorf

Neuron-Glia Interfaces

13.00 *James Fawcett, Cambridge (UK)*
The role of Proteoglycans in regeneration and plasticity

13.20 *Andreas Faissner, Bochum*
Tenascin-C and related ligands in CNS wound reaction and repair

13.40 *Almudena, Ramon-Cueto, Valencia (Spain)*
Olfactory ensheathing glia autotransplantation: a therapy to repair injured spinal cords in primates

14.00 *Larry Benowitz, Boston (USA)*
Axon regeneration through the mature optic nerve

Cell-Based Therapies

14.20 *Anders Björklund, Lund (Sweden)*
Toward a stem cell therapy for Parkinson's disease

14.40 *Oliver Brüstle, Bonn*
ES cell-based neural transplantation

15.00 *Patrik Brundin, Lund (Sweden)*
Brain repair in experimental and clinical Parkinson's disease

15.20 **Coffee – Tea**

Gene-Therapy

15.50 *Jacques Mallet, Paris* (France)
Optimization of viral vectors for gene transfer in the nervous system

16.10 *Patrick Aebischer, Lausanne (Switzerland)*
The potential of lentiviral vectors for neurodenegerative diseases

16.30 *Steve Dunnett, Cambridge (UK)*
The role of training and experience in graft-derived recovery of function

Applications are invited for a tutorial course on

Computational Neuroscience

24. - 28. 9. 2003

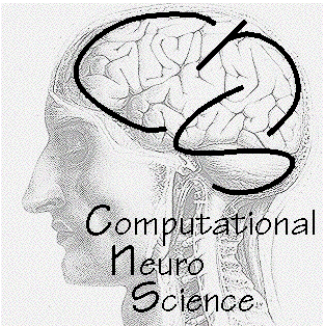
at the MPI for Fluid Dynamics, Göttingen

presented by the

German Neuroscience Society

and organized by

M. Herrmann, M. Diesmann, and T. Geisel



The course is intended to provide graduate students and young researchers from all parts of neuroscience with working knowledge of theoretical and computational methods in neuroscience and to acquaint them with recent developments in this field. The course includes topics such as

- Mechanisms and models of visual attention
- Models of synaptic background activity
- Theory of neural coding
- Structure and function of large-scale cortical networks
- Theory of sensor-motor learning
- Dynamics in local neural networks.

Tutorials and lectures will be given by: Prof. Dr. Stefan Treue (Göttingen), Dr. Nicolas Brunel (Paris), Dr. Michael Rudolph (Paris), Prof. Dr. Klaus Pawelzik (Bremen), PD Dr. Markus Lappe (Münster), PD Dr. Rolf Kötter (Düsseldorf), and by the organizers.

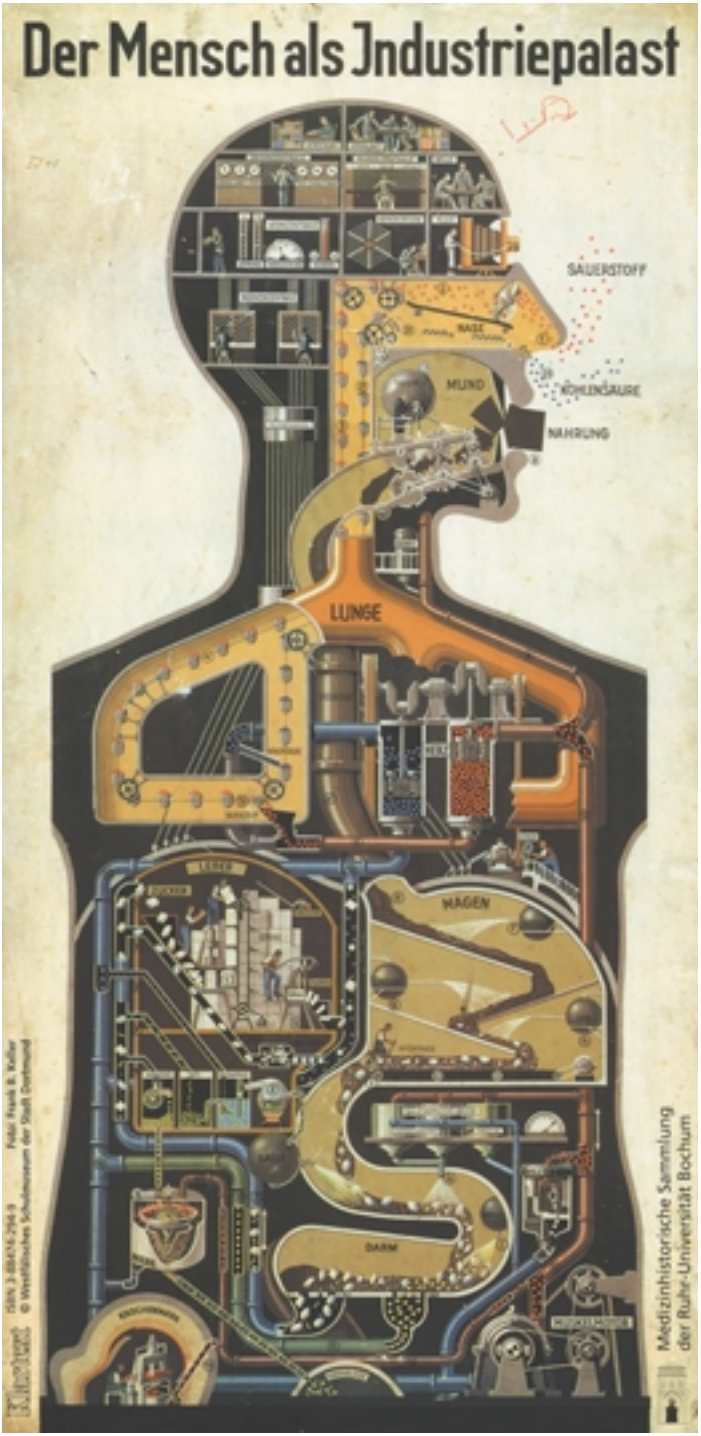
The course takes place at the Department of Nonlinear Dynamics of the Max Planck Institute for Fluid Dynamics, Bunsenstr. 10, D-37073 Göttingen. The course is free for members of the German Neuroscience Society, while non-members are charged a fee of 100 EUR. All tutorials are given in English. The number of participants is limited to 20.

To apply please fill out the form at: www.chaos.gwdg.de/~nwg-course by

July 1, 2003

For further information please contact: nwg-course@chaos.gwdg.de.

Der Mensch als Industriepalast



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Das Gehirn und sein Geist

Hg. von Norbert Elsner und Gerd Lüer

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Wolf Singer: Vom Gehirn zum Bewußtsein
Gerd Lüer: Simulationsmodelle für den menschlichen Geist: Kann man die psychischen Tätigkeiten nachahmen?
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Hg. von Norbert Elsner
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Karl Kardinal Lehmann: Kreatürlichkeit als Grundpfeiler des christlichen Menschenbildes

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Introductory Remarks to the Satellite Symposium C:

2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen

W. Paulus, F. Tergau, M. Nitsche and U. Ziemann

The interaction of human brain function with artificially induced intrinsic brain electricity is the central topic of this symposium. Short electric currents in the brain can be induced pain free by pulsed transcranial magnetic stimulation (TMS). With TMS applied in a repetitive mode (rTMS) succeeding pulses interact and may induce excitability alterations outlasting the stimulus train. Finally, transcranial direct current stimulation (tDCS) can directly modulate membrane polarisation and firing rates of cortical neurones. This symposium updates the knowledge of brain function gained by TMS and tDCS since the introduction of TMS in 1985. It was designed as a follow-up meeting of a first symposium held in Göttingen in 1998 and expands to recently developed areas of neuroimaging, neuropsychology and neural plasticity research using these techniques. TMS now has a definite place in neurological diagnostics in order to quantify alterations of conduction velocity or axonal loss of the pyramidal tract. More selective stimulation techniques in terms of coil design and pulse shape are currently being developed. tDCS has regained interest in recent years after it was shown that it definitely modulates cortical excitability. rTMS and tDCS after-effects can be shaped with concurrent drug applications. Several paired stimulation techniques allow detection of after-effects lasting 24 hours and longer.

In addition, electric stimulation of the brain may be used as a therapeutic tool in neuropsychiatric diseases. An already established therapeutic application of electric stimulation is deep brain stimulation in Parkinson's disease or dystonia. Non-invasive stimulation techniques would avoid invasive surgery and are approached in future as experimental therapeutic research. So far progress has been made in using rTMS in the treatment of depression, whereas the use of rTMS in other diseases such as epilepsy or movement disorders is still experimental. Technical innovations are a prerequisite for the biological progress of this field. Interactive discussions about techniques, their applications and objectives are expected in order to move this research forward.

This symposium has been generously supported by

Deutsche Forschungsgemeinschaft
Deutsche Gesellschaft für Klinische Neurophysiologie
Land Niedersachsen

SATELLITE SYMPOSIUM C

Wednesday, June 11th – Saturday, June 14th, 2003, Lecture Hall 8

2. International transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) Symposium Göttingen

Wednesday, June 11th 2003

Chair: Reiner Benecke (Rostock) and Mark Hallett (Bethesda, USA)

TMS Basics and Methods

15.30 *Anthony Barker, Sheffield (UK)*
Eighteen years of TMS – Principles and Practice

15.45 *Stephan Brandt, Berlin*
Contributions to the field by Bernd-Ulrich Meyer and Simone Röricht

16.05 *Jarmo Ruohonen, Helsinki (Finland)*
Modelling of the stimulating field generation in TMS

16.20 *Thomas Weyh, München*
Comparing coil characteristics

16.35 *Michel R. Magistris, Geneva (Switzerland)*
The triple stimulation technique

16.50 *Risto Illmoniemi, Helsinki, (Finland)*
EEG reactions to transcranial magnetic stimulation

17.05 **Coffee Break**

17.30 *Martin Sommer, Göttingen*
Bipolar versus monopolar transcranial magnetic stimulation

17.45 *Friedemann Awiszus, Magdeburg*
TMS and threshold hunting

18.00 *Tatsuya Mima, Kyoto (Japan)*
Effect of rTMS over the premotor cortex on the cortico-muscular coherence

18.15 *Vincenzo Di Lazzaro, Rome (Italy)*
Generation of I-waves in the human: spinal recordings

18.30 *Mark Hallett, Bethesda (USA)*
Surround inhibition

Thursday, June 12th 2003

Chair: Roger Lemon, London (UK) and Otto Witte (Jena)

Animal studies

8.30 *Klaus Funke (Bochum)*
TMS and single unit recordings in the visual cortex of the cat

8.45 *Sarah Lisanby, NewYork (USA)*
Neurophysiological effects of magnetically induced seizures in monkeys and humans

9.00 *Yoshikazu Ugawa, Tokyo (Japan)*
Long term effects of rTMS over the motor cortex studied in humans and monkeys

9.15 *Otto Witte, Magdeburg*
Functional inhibition in the surround of experimental focal cortical dysplasias

9.30 *Vahe Amassian, New York (USA)*
TMS and I-waves: their phylogeny and origin

Chair: Günther Deuschl (Kiel) and Ulf Ziemann (Frankfurt)

Motor cortex physiology

10.15 *John Rothwell, London (UK)*
Functional connectivity of human premotor and motor cortex explored with TMS

10.30 *Robert Chen, Toronto (Canada)*
Interactions between different inhibitory systems in the motor cortex

10.45 *Tihomir Ilic (Frankfurt)*
Paired pulse TMS: The dimension of stimulus intensity

11.00 *R. Hanajima, Tokyo (Japan)*
Paired pulse TMS: different mechanisms for intracortical inhibition induced by paired pulse TMS at different intervals

11.15 *Shaheen Hamdy, Salford (UK)*
The organisation and reorganisation of human swallowing in the motor cortex

11.30 *Christian Gerloff, Tübingen*
Inhibitory control of acquired motor programmes in the human brain

11.45 *A Muenchau, Hamburg*
Functional connectivity of human motorcortical areas

12.00 *Kerry Mills, Oxford (UK)*
Mapping motor cortex projections to single motor units in humans with transcranial magnetic stimulation

12.15 *Ulf Ziemann, Frankfurt*
Pharmacology of TMS

Chair: Mark George (Charleston, USA) and Thomas Paus (Montreal, Canada)

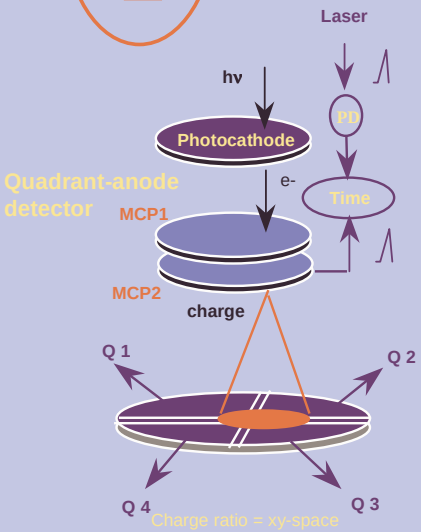
TMS and imaging

16.00 *Tomas Paus, Montreal (Canada)*
Studies of neural connectivity in healthy and disordered human brain

Novel SPC Imaging Detectors for Study of Picosecond FLIM & FRET in Living Cells and Single Molecules at Ultra-Low Excitation Levels

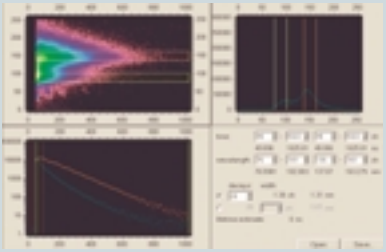
EuroPhoton GmbH

Klaus Kemnitz

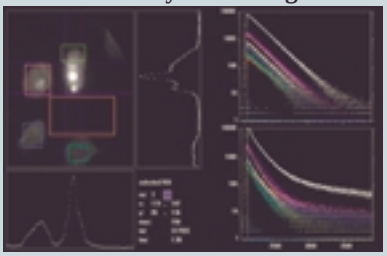


FRET analysis of xFP-proteins

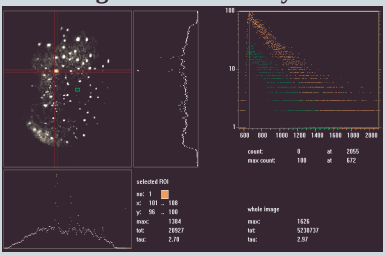
Spectroscopic analysis of FRET



Lifetime-analysis in living cells



Single molecule analysis



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D-12247 Berlin
Tel (49-30) 771-90145

Fax: (49-30) 771-4450
kkemnitz@t-online.de

Laboratory:
Rudower Chausse 29
D-12489 Berlin
Tel: (49-30) 6392-6301

16.15 *Daryl Bohning, (Charleston, USA)*
Interleaving fMRI and rTMS

16.30 *Jürgen Baudewig, Göttingen*
Methodological considerations for simultaneous TMS and fMRI studies

16.45 *Sven Bestmann, Göttingen*
BOLD MRI interleaved with high frequency TMS of the motor cortex

17.00 *Hartwig Siebner (Kiel)*
Applications for combined TMS-PET studies in clinical and basic research

Chair: Joseph Classen (Würzburg) and Frithjof Tergau (Göttingen)

rTMS in Neurology and Psychiatry

17.45 *Frithjof Tergau, Göttingen*
Epilepsy

18.00 *Jens Rollnik, Hannover*
rTMS for the treatment of pain

18.15 *R. H. Belmaker, Beersheva (Israel)*
TMS animal models of psychiatric diseases

18.30 *Martin Keck, München*
The neurobiological basis of therapeutic use of rTMS in psychiatric disorders

18.45 *Mark George, Charleston (USA)*
rTMS in Psychiatry

19.00 *Leon Grunhaus, Sheba (Israel)*
ECT vs TMS, cortical excitability and more

19.15 *Frank Padberg, München*
TMS and depression

Friday, June 13th 2003

Chairs: Vincent Walsh (London, UK) and Paolo Rossini, Rome (Italy)

Visual system, cognition and memory

9.00 *Alan Cowey, Oxford (UK)*
Transcranial magnetic stimulation and cognitive Neuroscience

9.15 *Vincent Walsh, London (UK)*
Complementary localization and lateralization of orienting and motor attention

9.30 *Thomas Kammer, Tübingen*
Phosphenes and visual suppression by occipital TMS

9.45 *Rudolf Töpper, Hamburg*
Motor cortex and speech

10.00 *Babak Boroojerdi, Aachen*
Rapid experience-dependent plasticity in the visual system

10.15 *Peter Schwenkreis, Bochum*
Fluctuations of motor cortex excitability in pain syndromes

10.30 *Andrea Antal, Göttingen*
Visual perception influenced by TMS and tDCS

10.45 *Hugo Theoret, Boston (USA)*
Controlled paradoxical functional facilitation with TMS

Chairs: Leonardo Cohen (Bethesda, USA) and Charles Epstein (Atlanta, USA)

Plasticity and learning

16.00 *Leonardo Cohen (Bethesda, USA)*
Behavioral and physiological correlates of cortical plasticity: studies with TMS

16.15 *Joseph Classen, Würzburg*
Paired stimulation techniques in conjunction with TMS

16.30 *Charles Epstein, Atlanta (USA)*
rTMS and learning

16.45 *Martin Tegenthoff, Bochum*
Cortical and psychophysical effects of rTMS in Hebbian learning

17.00 *Paolo Rossini , Rome (Italy)*
Prefontal cortex in long-term memory: an „interference" approach using magnetic stimulation

17.15 *K Irlbacher, Berlin*
Motor cortex plasticity after hand amputation

17.30 *Volker Hömberg, Düsseldorf*
TMS in neurorehabilitation

17.45 *C Bütefisch, Düsseldorf*
Modulation of use-dependent plasticity by amphetamine

18.00 *Konrad J. Werhahn, Mainz*
Bihemispheric plasticity after acute hand deafferentation

Saturday, June 14th

Chair: Grzegorz Hess (Krakow, Poland) and John Rothwell (London, UK)

Transcranial direct current stimulation

10.00 *Roger Lemon, London (UK)*
Primate motor cortex physiology

10.30 *Grzegorz Hess, Krakow (Poland)*
LTP and DC stimulation in rat motor cortex slices

10.45 *Nils Birbaumer, Tübingen*
Early human studies on direct current stimulation

11.00 *David Liebetanz, Göttingen*
Safety aspects of tDCS in the animal model and the human

11.15 *Michael Nitsche (Göttingen)*
Inducing LTP and LTD like effects in the human motor cortex

11.30 *Nicolas Lang, Göttingen/London*
Combining rTMS and DC stimulation of the motor cortex

11.45 *Walter Paulus (Göttingen)*
Pharmacology of tDCS

Chairs: Christian Hess (Bern, Switzerland) and Reinhard Dengler (Hannover)

Neurological diseases

15.00 *Christian Hess, Bern (Switzerland)*
TMS in clinical neurophysiology

15.15 *Reiner Benecke, Rostock*
TMS: relation to deep brain stimulation

15.30 *Alberto Priori, Milano (Italy)*
Motor cortex excitability in chorea and myoclonus

15.45 *Guenter Deuschl, Kiel*
TMS and tremor

16.00 *Karl Wessel, Braunschweig*
TMS and cerebellum

16.15 *Reinhard Dengler, Hannover*
TMS in ALS

16.30 **Coffee Break**

17.00 *Hiroshi Shibasaki, Kyoto (Japan)*
The effect of rTMS on sensorimotor function and focal dystonia

17.15 *Kai Rösler, Bern (Switzerland)*
Triple stimulation technique: clinical applications

17.30 *Ludwig Niehaus, Berlin*
Interhemispheric inhibition in stroke

17.45 *Peter Urban, Mainz*
Magnetic stimulation and brainstem

18.00 *Joachim Liepert, Hamburg*
TMS in stroke patients

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Introductory Remarks to the Satellite Symposium D:

Novel properties of channels

Special Interest Group „Ionenkanäle" der Deutschen Physiologischen Gesellschaft

Klaus Benndorf, Heinrich Terlau and Frank Lehmann-Horn

Ion channels embedded in the plasma membrane of cells fulfil multiple physiological functions, including signal processing, secretion, or regulation of the cell volume. In the symposium recent data on novel channel proteins, channel activation mechanisms, and so far unknown channel functions will be presented.

Transient receptor potential proteins (TRP) form a family of Ca^{2+} permeable channels that are activated by a variety of signals as decreased intracellular Ca^{2+}, noxious thermal and chemical stimuli, and increased cell volume. Functional properties of TRPV4 channels, originally identified as osmotically activated channels, will be presented by C. Harteneck.

Cutaneous cold receptors are activated by the cooling of the skin and also by the application of menthol. Recently, one of the channels mediating the cold and menthol response has been identified and named CMR-1. This channel also belongs to the TRP-family (TRPM8). G. Reid will focus an the ionic channels involved in cold sensing.

Sensation of color by cone photoreceptors in the retina is mediated by cyclic nucleotide-gated (CNG) channels. Mutations in the A and B subunits of the CNG channels were identified to cause various forms of complete and incomplete color blindness (achromatopsia). Molecular mechanisms underlying these channelopathies will be presented by R. Seifert.

R. Blum will focus on the activation of TTX-insensitive $Na_v1.9$ sodium channels by neurotrophins, a surprising activation mechanism of sodium channels because these channels usually open in response to a voltage change across the membrane.

Pacemaker channels (hyperpolarization-activated pacemaker channels, HCN channels) have been cloned several years ago and it has become clear now that these channels are used by nature in different organs to induce rhythmical electrical activity. Properties and function of these channels in both the heart and the thalamus will be presented by M. Biel.

SATELLITE SYMPOSIUM D

Wednesday, June 11th 2003, 13.00–16.00

Novel properties of channels

Chair: Klaus Benndorf and Heinrich Terlau

13.00 *Christian Harteneck, Berlin*
Characterisation of TRPV4 and potential functions

13.35 *Gordon Reid, Bukarest*
Ion channels involved in cold sensing

14.10 *Robert Seifert, Jülich*
Preliminary CNG channels and sour taste

14.45 *Robert Blum, München*
Nav1.9, a sodium channel involved in neurotrophin-evoked depolarization

15.20 *Martin Biel, München*
Pacemaker channels of heart and thalamus

Introductory Remarks to Symposium 1

Adaptation: the psychophysicist's microelectrode

Nikolaus Troje and Michael Bach

Adaptation is a very general and basic phenomenon in biological information processing, covering a broad range from gain control to „fatigue". Adaptation provides an active mechanism for efficient data compression by removal of redundancy: encoding changes of properties rather than the properties themselves allows the visual system to acquire, transmit, process and store information in a highly economical manner while minimising losses. However, besides its functional significance, adaptation has also proven to be a valuable scientific instrument to non-invasively investigative, characterize and isolate sensory information processing pathways.

In the visual domain, adaptation has traditionally been used mainly to study early visual processing. During the last few years, however, it has become evident that adaptation and corresponding after-effects also play a major role in high-level cognitive processing and that it can be employed to study phenomena such as face recognition or biological motion perception. In this symposium, we want to trace this development spanning the whole range between low-level vision and high-level cognitive processes, on the one hand, while emphasizing the dualistic nature of adaptation as a neural mechanism and as an investigative tool, on the other hand.

J. Zanker will open the series of presentations by providing a general introduction into the concepts of spatio-temporal visual signal coding that lead to the phenomena of after-effects in time as well as to simultaneous contrast enhancement in space. Using the example of motion boundaries in time and space he will illustrate this point in more detail by comparing results from a computational motion detection model to psychophysical observations.

The next two talks will provide illustrative examples of the use of adaptation for probing the properties of low level visual filters. In the contribution of M. Fahle selective adaptation is used as a tool to study the effects of perceptual learning on the characteristics of orientation selective visual filters. M. Bach uses a complex double adaptation paradigm to isolate direction specific motion responses in VEPs from direction unspecific flicker responses.

M. Greenlee's contribution is particularly interesting because he shows that contrast gain control, a mechanism that implements adaptation to varying light intensities, itself can be highly adaptive, therefore demonstrating „second order adaptation" in the visual system.

In the last two contributions it is shown that adaptation is not only a low-level visual phenomenon. D. Leopold presents data on aftereffects in face recognition and N. Troje finds similar effects for biological motion perception.

SYMPOSIUM 1

Thursday, June 12th 2003, 9.00–12.00, Lecture Hall 7

Chair: Nikolaus Troje (Bochum) and Michael Bach (Freiburg)

Adaptation: the psychophysicist's microelectrode

9.00 *Johannes M. Zanker*
Adaptation and contrast enhancement as universal coding strategies in the human visual system

9.30 *Manfred Fahle*
Orientation bandwidth of perceptual learning

9.50 *Michael Bach and J. P. Maurer*
Uncovering veridical human motion detectors in the EEG using „double adaptation“

10.15 **Coffee Break**

10.45 *Mark W. Greenlee*
Contrast gain control in visual cortex: evidence from psychophysics and fMRI

11.10 *David A. Leopold, I. V. Bondar, A. J. O'Toole and N. K. Logothesis*
Aftereffects with faces: evidence for prototype referenced encoding of identity

11.35 *Nikolaus Troje and Henning Geyer*
High-level aftereffects in biological motion perception

Introductory Remarks to Symposium 2

Juvenile hormone as a mediator of behavioural plasticity in adult insects

Uwe Rose and Sylvia Anton

Since its discovery by Wigglesworth in 1934, Juvenile hormone (JH) has been known as an important regulator of insect developmental processes. In recent years, JH has also been pointed out as one of the major hormones regulating reproductive development in adult insects. In addition to its effect on the maturation of reproductive organs, it also influences the morphology and function of the nervous and muscular system, thereby regulating sexual and other age-related behaviour. This symposium will highlight JH-regulated behaviour and possible mechanisms of JH action in different insect species.

In general, an increase of JH biosynthesis during early adult life has been shown in all insect species investigated and the link of the observed behaviours with JH levels has been made through manipulation of these levels either by allatectomy or by injection of JH or JH analogs. Effects of JH have been shown to be reversible, demonstrating the plasticity of hormone-mediated behaviour.

At the beginning of this symposium P.E.A. Teal and Y. Gomez-Simuta will discuss the pivotal role of JH for the development and coordination of sexual signalling in Tephritid fly species. The ability to perceive signals from possible mating partners is important for a successful mate finding and the talks by C. Gadenne and J. Stout deal with JH-dependent changes in the sensitivity of two different sensory systems. In male noctuid moths, C. Gadenne showed that behavioural sensitivity and sensitivity of central olfactory neurons to female-emitted sex pheromone increase with age and JH level. J. Stout will show that the attraction of female crickets by calling songs produced by conspecific males changes with JH level. Plasticity and development of the female phonotactic behaviour can be understood by changes in the response properties of prothoracic auditory neurons.

In some insect species egg-laying behaviour is triggered by elevated JH levels. U. Rose will talk about the locust motor system that undergoes JH-dependent morphological and functional remodelling which are a pre-requisite for a successful egglaying behaviour. In the mushroom bodies of crickets, JH has been shown to stimulate neurogenesis and M. Cayre will discuss the question whether these newly generated neurons play a role in the maturation of egg-laying behaviour.

In honey bees age-related division of labour depends on JH hemolymph titers. Division of labour is also associated with plasticity in circadian rhythms and G. Bloch will present data on possible interactions of JH with the circadian clock.

Although only at its beginnings, the diversity of hormonal effects in adult insects is evident and strikingly resembles comparable effects in vertebrates. Future research will have to show how JH acts at the cellular and biochemical level.

SYMPOSIUM 2

Thursday, June 12th 2003, 9.00–12.00, Lecture Hall 9

Chair: Uwe Rose (Ulm) and Sylvia Anton (Lund, Sweden)

Juvenile hormone as a mediator of behavioural plasticity in adult insects

9.00 **Introductory remarks**

9.05 *Peter Teal, Florida (USA) and Y. Gomez-Simuta Chiapas, (Mexico)*
Juvenile hormone regulation of reproductive maturity and sexual signaling in tephritid fruit flies

9.30 *Christophe Gadenne, Bordeaux (France)*
Effect of juvenile hormone on olfactory guided behaviour and on central nervous processing of odours in a moth

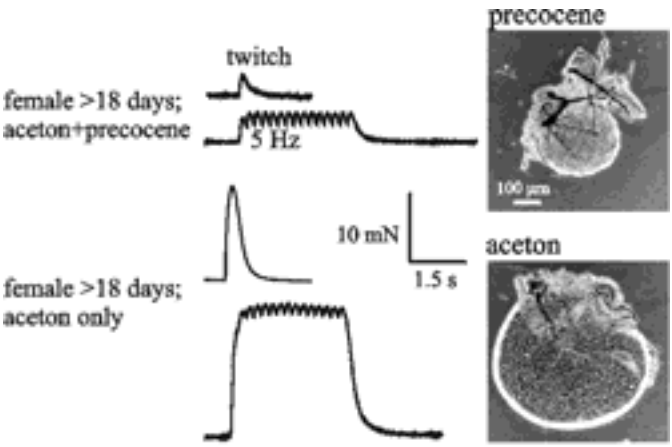
9.55 *John F. Stout, Michigan (USA)*
Juvenile hormone III influences phonotactic behavior by female crickets through regulation of the response properties of identified auditory interneurons

10.20 **Coffee Break**

10.45 *Uwe Rose, Ulm*
Morphological and functional maturation in the adult locust neuromuscular system regulated by juvenile hormone

11.10 *Myriam Cayre, Marseille (France)*
Juvenile hormone, neurogenesis and behaviour in the adult cricket

11.35 *Guy Bloch, Jerusalem (Israel)*
Juvenile hormone and task-related plasticity in circadian rhythms in the honey bee



Inhibition of corpora allata function (right) by precocene alters contraction properties of locust muscle fibers. (Adapted after Rose et al. 2001)

Introductory Remarks to Symposium 3

Cytokines as mediators of neuroglial interactions

Jörg Mey and Heike Siebert

After traumatic nerve injury, in ischemic brain damage and in neurodegenerative diseases, it is of foremost clinical concern to prevent nerve cell death and to develop strategies for the support of axonal regeneration. This requires an understanding of traumatic processes in the nervous system and of their regulation by intercellular signals. Originally deriving from immunological research the cytokine concept has gained increasing relevance in this context. In the CNS, cytokines mediate interactions between astrocytes, microglia cells, neurons and, under pathological conditions, infiltrating leukocytes from the circulation. Peripheral nerve lesions also activate paracrine signals between macrophages, Schwann cells and neurons. Cytokines are polypeptides that bind with high affinity to specific cell surface receptors and activate intracellular second messenger cascades. Unlike hormones, cytokines are not stored in glands as preformed molecules but are rapidly synthesized and secreted by a variety of cell types after stimulation. In development and under pathological conditions they act on many different targets and frequently affect the action of other cytokines in a synergistic or antagonistic manner. Their physiological functions in the nervous system will be discussed in this symposium.

Stefan Wiese's contribution focuses on the neuropoietic cytokines (including IL-6, CNTF, LIF). They share the gp130-family of receptors that activate janus kinases and the STAT transcription factors. This pathway is activated as part of the immediate inflammatory reaction. In addition, neurotrophic properties of CNTF and LIF have been reported for various neuronal populations. Discussed by Hans Werner Müller, the chemokines comprise a large family of small proteins, who mediate their biological effects through G-protein-coupled receptors. They are primarily characterized as chemoattractants of hematogenous cells. Chemokines and matrix metalloproteinases are the subject of Heike Siebert's talk. Various metalloproteinases appear in the nervous system, in particular after blood brain barrier leakage, and contribute to the removal of extracellular matrix. Gennadij Raivich has investigated a number of cytokines including TGFβ and TNF. Binding of TGFβ to cell surface receptors requires its local release from a latency associated peptide. In consequence, Smad-proteins are phosphorylated in the target cell and translocate to the nucleus, where they form heteromeric complexes to regulate gene transcription. Astrocytes, oligodendrocytes, microglia and neurons have been shown to be targets of TGFβs which tend to cause cell cycle arrest and differentiation. TGFβ activates ECM deposition by astrocytes and fibroblasts and also modulates the activity of a large number of other cytokines that are involved in immune reactions. TNFα effects are also mediated by plasmamembrane receptors. Via recruitment of intracellular adaptors proteins it can trigger apoptosis or activate the transcription factors NFκB and JUN. Its functions in peripheral nerve de- and regeneration will be covered by Claudia Sommer. In contrast to the cytokines proper, the lipophilic retinoic acid penetrates cellular membranes. Its receptors are localized in the cell nucleus where they act as ligand-activated transcription factors. Jörg Mey will discuss the role of retinoic acid as a regulator of cytokines after peripheral nerve injury.

SYMPOSIUM 3

Thursday, June 12th 2003, 09.00–12.00, Lecture Hall 104

Chair: Jörg Mey (Aachen) and Heike Siebert (Göttingen)

Cytokines as mediators of neuroglial interactions

9.00 **Introduction**

9.05 *Stefan Wiese and Michael Sendtner, Würzburg*
The role of STAT-3 for developing and regenerating neurons

9.30 *Hans Werner Müller, Düsseldorf*
SDF-1 chemokines in the mammalian nervous system: expression, regulation and function

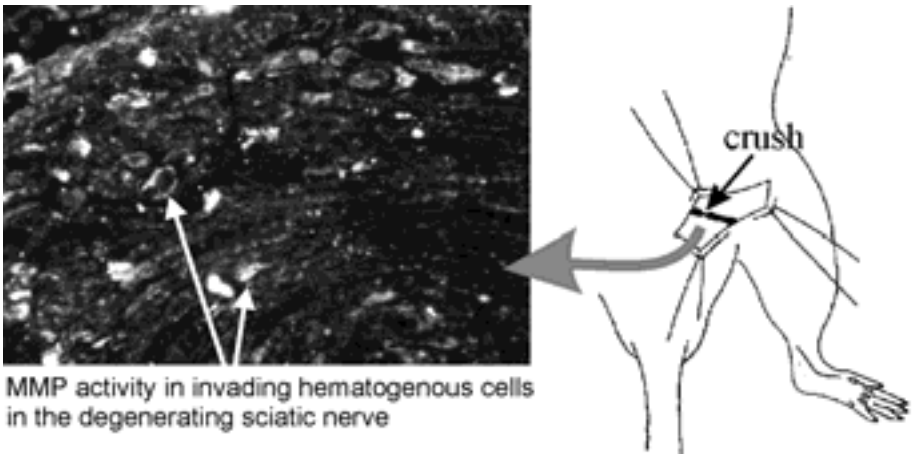
9.55 *Heike Siebert, Göttingen*
Cytokines and proteases that influence sciatic nerve degeneration

10.20 **Coffee Break**

10.45 *Gennadij Raivich, London*
TGFβ1, TNFα and their function in neuronal degeneration

11.10 *Claudia Sommer, Würzburg*
Expression and transport of TNFα in peripheral nerve injury

11.35 *Jörg Mey, Aachen*
Retinoic acid as a regulator of cytokine signaling in peripheral nerve regeneration



MMP activity in invading hematogenous cells in the degenerating sciatic nerve

Introductory Remarks to Symposium 4

Transgenic animal models of neurodegenerative diseases

Jörg B. Schulz and Christian Haass

Identification of genetic causes underlying either typical hereditary neurodegenerative diseases (e.g. Friedreich's ataxia) or rare hereditary forms of typically idiopathic neurodegenerative diseases (e.g. Alzheimer's and Parkinson's disease) has raised new opportunities to study the pathogenesis of these neurodegenerative disorders. Although molecular and biochemical consequences of mutations may be studied in cell lines and primary cell cultures, only animal models allow to study functional consequences in a complete organism, in their biological context and the consequences in behavior. Model systems like *C. elegans* and *D. melanogaster* allow to study organisms from birth to death in a short time period. Furthermore, hypotheses can be tested rapidly by simple and quick genetic manipulations. In the year 2002 the nobel prize committee honored researchers who identified *C. elegans* as a model system for disease and who helped to identify its complete genome.

Philipp Kahle will discuss transgenic mouse models of synucleinopathies based on ectopic expression of disease-related α-synuclein. Somatodendritic accumulation of α-synuclein was observed in dopaminergic neurites. Ultimately, formation of Parkinson's disease pathology causes severe locomotor dysfunction in transgenic mice. Oligodendroglial expression of α-synuclein induces pathological and biochemical changes resembling multiple system atrophy.

Hélène Puccio will review her work on frataxin-deficient mice. Whereas knockout mice are intrauterine lethal, mice with a neuronal or muscular deficiency of frataxin develop behavioral symptoms, pathological and biochemical changes soon after birth and have a life expectancy of only 5 and 9 weeks, respectively. They serve as valuable animal models for Friedreich's ataxia.

Bart de Strooper will focus on the physiological function of presenilins and pathological changes occurring in Alzheimer's disease-associated mutations using transgenic mice. Similarly, Frank Heppner will review transgenic mice as models for prion disorders.

Finally, Ralph Baumeister will discuss *C. elegans* as a model system for Parkinson's and Alzheimer's disease, allowing to study the consequences of disease-associated mutations in α-synuclein, parkin, presenilins and amyloid.

SYMPOSIUM 4

Thursday, June 12th 2003, 9.00–12.00, Lecture Hall 10

Chair: Jörg B. Schulz (Tübingen, Germany) and Christian Haass (Munich, Germany)

Transgenic animal models of neurodegenerative diseases

9.00 *Philipp Kahle, Munich, Germany*
Transgenic model systems for synucleinopathies

9.30 *Hélène Puccio, Illkirch, France*
Mouse models of Friedreich's ataxia – models for oxidative stress

10.05 *Bart de Strooper, Leuven, Belgium*
Presenilin biology in cells and transgenic model systems

10.40 **Coffee Break**

10.55 *Frank Heppner, Zürich, Switzerland*
Transgenic mouse models of prion disorders

11.30 *Ralf Baumeister, Munich, Germany*
Parkinson's and Alzheimer's disease in C. elegans

Introductory Remarks to Symposium 5

Signal integration in dendrites

Thomas Berger and Matthew Larkum

The last decade has seen a resurgence of interest in the properties of dendrites, spurred on by advances in techniques that have allowed researchers to probe their active nature. Up to the beginning of the 1990's it was fashionable to treat dendrites as passives structures in order to reduce their complexity and allow predictions of what computational advantage dendrites might provide. More and more since this time, researchers and theoreticians have had to face up to the additional complexity represented by dendrites with active conductances. Within this new framework there have been two paradigms: the one, treating dendritic conductances as mechanisms to compensate for the passive effects of dendrites and thereby normalize the efficacy of synaptic contacts over the whole tree, and the other, treating dendritic conductances as crucial for additional computational capabilities only possible with interactions in the dendritic tree.
Signal integration in active dendrites is enriched by two features that have been found in most neuronal cell types studied so far: a) action potentials can propagate actively along dendrites (the notable exception being Purkinje cell dendrites) and b) dendrites have regenerative regions which can produce local and/or forward propagating action potentials. Thus, signal integration from the modern perspective must embrace the concept that cells can send information about activity from one subcellular region to another. Furthermore, additional mechanisms can come into play for modulating this form of intracellular communication. This leads to a much more complicated view with signal integration being distributed in place and time throughout the whole cell.
The talks in this symposium will cover the recent research into these topics. Alex Reyes will demonstrate some of the properties of synaptic integration above and below threshold for action potentials. Then Jeff Magee and Matthew Larkum will introduce the topic of dendritic action potentials and active propagation in hippocampal and neocortical pyramidal cells. The second session will concentrate on the modulation of intracellular communication and the consequences for signal integration by inhibitory synaptic inputs (Michael Häusser) and leak conductances (Thomas Berger). Lastly, Greg Stuart will show how dendritic interactions can modulate calcium influx through NMDA channels.

SYMPOSIUM 5

Thursday, June 12th 2003, 9.00–12.00, Lecture Hall 11

Chair: Thomas Berger (Bern, Switzerland) and Matthew Larkum (Heidelberg)

Signal integration in dendrites

9.00 *Thomas Berger*
Introductory Remarks

9.05 *Alex Reyes, New York (USA)*
Integration of synaptic inputs: Summation in the subthreshold and suprathreshold range

9.30 *Sonia Gasparini and Jeff Magee, New Orleans (USA)*
Regulation of local dendritic spike initiation and propagation in CA1 pyramidal neurons

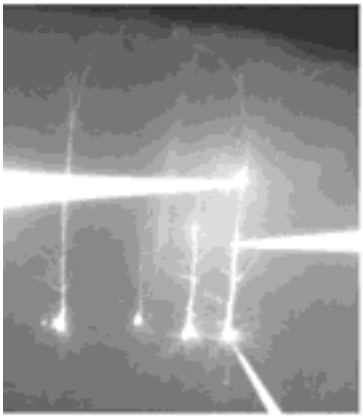
9.55 *Matthew Larkum, Heidelberg*
Dendritic interactions in layer 2/3 neocortical pyramidal cells

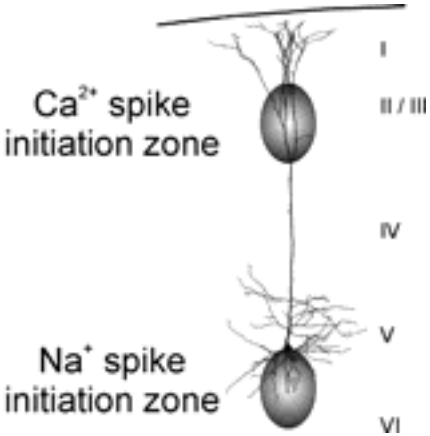
10.20 **Coffee Break**

10.45 *Michael Häusser, London (UK)*
Interactions of action potentials with somatic and dendritic IPSPs

11.10 *Thomas Berger, Bern (Switzerland)*
Electrotonic separation of two spike initiation zones in layer 5 pyramidal cells: Role of I_h

11.35 *Greg Stuart, Freiburg*
Dendritic mechanisms involved in spike-timing dependent plasticity





Introductory Remarks to Symposium 6

Neuronal death and neuroprotection: The role of glial cells

Andreas Reichenbach and Christian Steinhäuser

It is well known that glial cells in the brain undergo distinct and characteristic morphological alterations under pathological conditions. Evidence is now accumulating that the structural changes are accompanied by variations in glial functioning. These cells express a set of ion channels and receptors similar to their neuronal counterparts, and alterations of gating properties or expression levels of these channels and receptors might be involved in pathological processes of the CNS.

The Symposium aims at demonstrating that alterations in functional and molecular properties of microglia, oligodendrocytes and astrocytes can be causative of various CNS diseases, or, by contrast, exert protective effects. Six glia experts from four different countries will summarize latest knowledge indicating a critical role of glial cells in demyelinating disorders, epilepsy, and in the diseased retina.

Two lectures address the impact of glial cells in epilepsy. C. Steinhäuser describes functional and molecular changes of astroglial ionotropic glutamate receptors (AMPA subtype) and inwardly rectifying K^+ channels in the hippocampus of patients suffering from temporal lobe epilepsy. This subject is picked up by J. Gorter who provides a complementary report on altered expression of Na^+ channels and metabotropic glutamate receptors in microglia and astrocytes in a rat model of epilepsy. Both authors suggest a role for glial cells in seizure generation and/or seizure spread in the epileptogenic hippocampus. P. Kofuji shows that glial K^+ channels are crucial for oligodendrocyte development and myelination, supposing that glial cells are critically involved in the pathogenesis of demyelinating diseases. A. Buisson has identified glial factors secreted in response to TGF-beta stimulation which mitigate excitotoxic neuronal death. In his talk he will explain how these glial factors reduce the activation of glutamate receptors and why they might represent interesting candidates for alternative approaches to neuroprotection. A. Reichenbach summarizes recent findings on retinal Müller cells, delineating metabolic pathways enabling the cells to exert protective effects against glutamate-mediated neurotoxicity and free radical-induced injury. Finally, T. Reh presents evidence for the possibility of glia-derived neuronal repair. His data indicate that gliotic Müller cells constitute a source of neuronal transdifferentiation in the postnatal retina.

The speakers of this Symposium will provide compelling evidence that astrocytes, oligodendrocytes and microglia are highly plastic cell types that undergo various, parallel functional changes in the course of a disease. Promising approaches in analysing the role of glial cells in pathogenesis have to consider these multiple mechanisms, as well as sub-regional peculiarities defined by the cell's specific microenvironment.

SYMPOSIUM 6

Thursday, June 12th 2003, 9.00–12.00, Lecture Hall 105

Chair: Andreas Reichenbach (Leipzig) and Christian Steinhäuser (Bonn)

Neuronal death and neuroprotection: the role of glial cells

9.00 *Christian Steinhäuser, Gerald Seifert, Bonn*
Functional and molecular changes in astrocytes of human epileptic hippocampus: Relevance to seizure generation

9.25 *Jan A. Gorter, Amsterdam (The Netherlands)*
Molecular and immunocytochemical changes in macro- and microglia in a rat model of mesial temporal lobe epilepsy

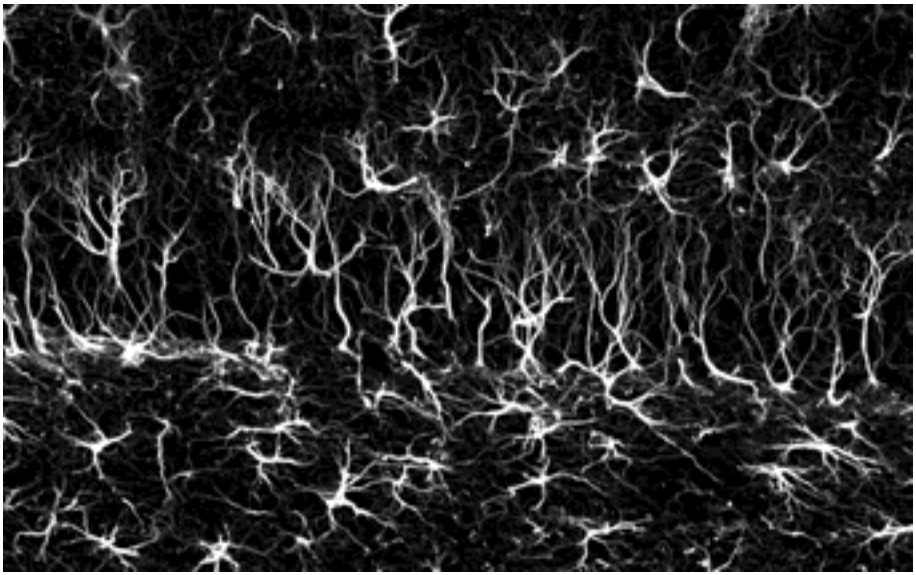
9.50 *Paulo Kofuji, Minnesota (USA)*
Demyelination and K^+ channels of oligodendrocytes

10.15 **Coffee Break**

10.45 *Alain L. Buisson, Caeu (France)*
Can glial cells modulate excitotoxic neuronal injury ?

11.10 *Andreas Reichenbach, Mike Francke, Leipzig*
Müller cells protect neurons by transfer of glutathione, and by control of extracellular glutamate

11.35 *Thomas Möller, Seattle (USA)*
Microglia: Friend or Foe?



The intriguing glial network. Murine hippocampus, GFAP immunohistohemistry.

Introductory Remarks to Symposium 7

Drug addiction: mechanisms and therapy

Volker Höllt

There is strong evidence that addictive drugs interact with an endogenous reward system in the brain. This system comprises a limbic circuitry of brain structures, such as amygdala, ventral tegmental area, nucleus accumbens, prefrontal cortex and other forebrain regions. Activation of these structures by addictive drugs or by other rewarding stimuli involves the modulation of the release of neurotransmitters, such as dopamine, glutamate, GABA, opioids and cannabinoids. Experimental interference with these neurotransmitter systems can alter the rewarding effects of addictive drugs. In addition, repeated administration of psychotropic drugs involves learning processes finally resulting in neuroplastic changes characterized as „addiction memory"". These adaptive processes are of long duration and may be responsible for the drug craving observed in long-term withdrawal/abstinence when all signs of somatic dependence have disappeared.

The present symposium will present molecular, behavioural and clinical aspects of drug addiction.

Neuroadaptive changes in gene expression in response to chronic opioid treatment will be addressed by V. Höllt. Using DNA microarrays the expression of about 8000 genes in the prefrontal cortex of rats chronically treated with morphine were analysed. Three persistently altered genes were found: *arc* and ania-3, proteins which are involved in synaptic function and *per*, a protein regulating circadian rhythmicity.

Cannabinoids and opioids are main neuromodulators of the reward system which have been proposed to act synergistically. Using mice with targeted deletions of the opioid peptide genes A. Zimmer will provide data which clearly show a close interaction of the cannabinoid and opioid system. Thus, cannabinoid withdrawal is attenuated in enkephalin knockout mice and conditioned place aversion of tetrahydrocannabinol is blocked in dynorphin-deficient animals.

Deprivation of alcohol in animals results in an enhanced ethanol consumption after reexposure to alcohol. Using this animal model for relapse and craving U. Schmitt will provide evidence that modulation of GABAergic neurotransmission clearly affects ethanol consumption and deprivation effects.

A detailed analysis of the glutamatergic mechanisms in addiction will be presented by W. Schmidt. Experimental evidence will be provided that interference with glutamatergic transmission alters the rewarding effect of addictive drugs. In addition, an increased reactivity of the glutamatergic system in long-term withdrawal is observed which appears to underly drug craving.

An overview of the reward system will be given by W. Hauber. By analysing the guidance of instrumental behaviour in rats he will provide evidence that signals related to expected natural reward are transmitted to the nucleus accumbens, a key region of the limbic cortico-striatal circuitry. The transmission of these signals in this structure involves NMDA- and AMPA glutamate receptors.

Clinically relevant neurobiological mechanisms of the development of drug addiction with an emphasis of the motivational aspects will be the topic of U. Havemann-Reinecke who will also provide an overview of the therapeutic stategies for dependence (substitution, anticraving drugs).

SYMPOSIUM 7

Thursday, June 12th 2003, 16.00–19.00, Lecture Hall 7

Chair: Ursula Havemann-Reinecke (Göttingen) and Volker Höllt (Magdeburg)

Drug addiction: mechanisms and therapy

16.00 *Volker Höllt, Magdeburg*
Gene expression profiles in rat brain after chronic morphine treatment

16.25 *Andreas Zimmer, Bonn*
Interaction between opioids and cannabinoids: Studies in knock-out mice

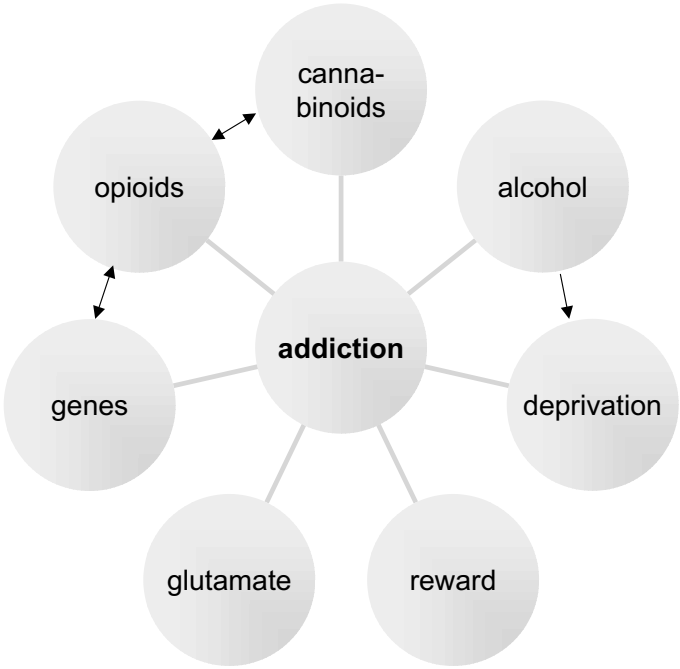
16.50 *Ulrich Schmitt, Mainz*
Alcohol deprivation and the development of addiction

17.25 **Coffee Break**

17.45 *Werner Schmidt, Tübingen*
Glutamatergic mechanisms in addiction

18.10 *Wolfgang Hauber, Stuttgart*
Control of behaviour by reward related stimuli

18.35 *Ursula Havemann-Reinecke, Göttingen*
Ecstasy, dependence and pharmacotherapy



Introductory Remarks to Symposium 8

Precise timing in the brain: linking neuronal activity and behavior

Detlef Heck and Fahad Sultan

Over the past decade evidence has accumulated that precisely timed neuronal activity in the neocortex is associated with various aspects of behavior. The role of these precisely timed activity patterns in the neocortex in the control of motor output is still unclear. Since synchronization occurs between distant parts of the neocortex, even bridging hemisphere boundaries, it is possible that synchronized activity is also used in the communication between different brain structures involved in the control of movement. The symposium shall bring together ideas about spatio-temporal activity patterns in neocortical neural networks with ideas about the function of the cerebellum and finally the neuronal control of precisely timed motor output.

Moshe Abeles has proposed that neocortical activity is organized in form of chains of synchronously active groups of neurons („synfire chains"). He later showed experimentally that precisely timed spike patterns – presumably reflecting a traveling „synfire chain" – occur during behavioral tasks in awake behaving monkeys.

Stefan Rotter and colleagues investigate and compare the contribution of single neurons and large populations of those to the control of various parameters of movement. They report that, when reconstructing movement trajectories on a single trial basis, the relevant time base of the underlying neuronal activity measures in tens of milliseconds rather than milliseconds.

Jeff Keating has used multiple electrode recordings in sensorimotor cortex to record activity during a reaching-grasping movement. He investigated the temporal correlation of neuronal activity and could show that successful and failure trials had identical changes in spike rate but were separable based on the dynamics of the correlation of neuronal activity prior to the grasp.

Peter Thier has shown using recordings from Purkinje cells in monkeys trained to do saccades of different amplitudes, that the population activity gives a precise temporal signature of saccade onset and offset. Further results suggest that the population response can be modified by changing the weights of the contribution of individual Purkinje cells, thus resulting in a change of saccade amplitude.

A cerebellar role in the precise timing of ball release in overarm throwing movements has been suggested by the ground breaking work of Jonathan Hore. His findings suggest that the combination of finger and hand muscle activity has to be controlled with millisecond precision in order to produce the desired motor output.

The cerebellum potentially detects precisely timed spike pattern generated by the neocortex and, triggered by their occurrence, produces the associated output. Precisely timed spike patterns may thus be a key to neocortical-cerebellar interaction (V. Braitenberg). Since the cerebellum is mostly involved in motor control we will try to build a bridge to temporally precise motor output.

SYMPOSIUM 8

Thursday, June 12th 2003, 16.00–19.00h, Lecture Hall 9

Chair: Detlef Heck (Freiburg) and Fahad Sultan (Tübingen)

Precise timing in the brain: linking neuronal activity and behavior

16.00 *M. Abeles, Jerusalem (Israel)*
Scales for computational elements in the cortex

16.30 *S. Rotter, Freiburg*
Decoding neuronal population activity associated with movement

17.00 *J. Keating, Pennsylvania (USA)*
Directional information flow in sensorymotor cortex during reaching as revealed by the gravitational transformation

17.30 *P. Thier, Tübingen*
Encoding of movement time by populations of cerebellar Purkinje cells

18.00 *J. Hore, Ontario (Canada)*
Precision and timing of motor output

18.30 *V. Braitenberg, Tübingen*
Spatio-temporal activity patterns as a key to cerebellar function

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Introductory remarks to Symposium 9

Ontogenetic cell death in the nervous system

Kerstin Krieglstein

Apoptotic cell death is a fundamental and essential process in development and tissue homeostasis of multicellular organisms. Roughly half of all neurons produced during neurogenesis die apoptotically before the nervous system matures. Apoptosis is a highly regulated biological process in which a cell is instructed to participate actively in its own demise. The signals identifying cells to be eliminated as well as the intracellular signaling events controlling apoptosis in the developing nervous system are far from being understood. This symposium will provide an overview on the current knowledge of cell-extrinsic and -intrinsic regulators of ontogenetic neural cell death.

Evidence for active triggering of neuronal death continues to accumulate. Death receptors such as p75, or FasR are thought to trigger cell death. Recent work by C. Henderson has provided new insights into the requirements for Fas signaling followed by a specific downstream pathway during motoneuron cell death.

The role of Fas ligand (FasL) is addressed by A. Martin-Villalba. Many neurological diseases involve neuronal degeneration and, consequently, cell death. Acute disorders, occurring within minutes and hours, e.g. brain trauma, or infarction involve injury-induced apoptosis. A. Martin-Villalba could demonstrate the neutralization of FasL is an essential step of her newly established experimental strategy to prevent ischemic neuron death in animal models of stroke.

Bcl-2 family members are important intracellular sensors that receive multiple signals from pathways upstream of irreversible cell damage. Bcl-family members play a pivotal role in deciding whether cells will live or die by either blocking or permitting the regulation of downstream cell death effectors at the mitochondrial level. J.-C. Martinou will address the molecular mechanisms underlying the mitochondrial involvement in apoptosis.

Cell death in the developing nervous system is already seen prior to neuronal differentiation and synaptogenesis. Early neural cell death is detected as early as neurulation and seems to affect proliferating neural precursor cells as well as young postmitotic cells during and following neurogenesis. Recent work by E. de la Rosa characterizes the molecular context in which cell death is permitted or prevented.

One of the still open questions relates to the cell extrinsic mechanisms that regulate cell death. Recent evidence provided by K. Krieglstein shows that the pleiotrophic molecule transforming growth factor-ß (TGF-ß) acts as a key regulator in the induction of developmental as well as lesion induced cell death.

SYMPOSIUM 9

Thursday, June 12th 2003, 16.00 -19.00, Lecture Hall 104

Chair: Kerstin Krieglstein (Göttingen, Germany)

Ontogenetic cell death in the nervous system

16.00 *Chris Henderson, Marseille (France)*
Active killing of neurons during development

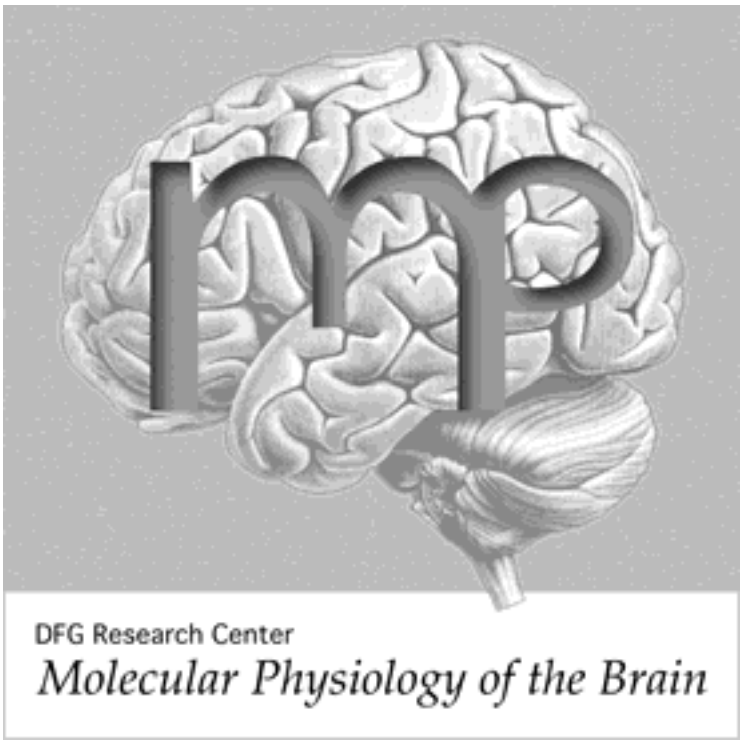
16.30 *Ana Martin-Villalba, Peter Krammer, Heidelberg*
Role of FasL in the nervous system

17.00 *Jean-Claude Martinou, Genf (Switzerland)*
Breaking the mitochondria barrier

17.30 **Coffee Break**

18.00 *Enrique de la Rosa, Ana Valenciano, Madrid (Spain)*
Regulation of programmed cell death during early neural development

18.30 *Kerstin Krieglstein, Göttingen*
TGF-ß is a key regulator in ontogenetic neuron death



Introductory remarks to Symposium 10

Insect neural and motor systems: from development to function and mechanics

Carsten Duch and Hans-Joachim Pflüger

The understanding of motor behavior and underlying circuitry requires a blend of many different research areas, making an integrative systems approach increasingly difficult. One of the strongholds of insect motor systems is that they can be analyzed at many different levels, and that bridging these levels is achieved in an increasing number of preparations. This symposium is intended to combine novel insights on the mechanisms underlying motor circuit development, the control of adult motor output by higher brain centers, the integration of motor output with muscle metabolism, and finally, the bionics underlying coordinated motor behavior.

Appropriate motor behavior relies on the integration of sensory information with the activity of central circuitry. However, isolated central networks can generate fictive locomotor rhythms in the absence of movement and sensory feedback. Therefore, the basic pattern of motor output is laid out by the intrinsic electrical properties and connectivity of neurons. Sensory input is required to adjust patterned motor output to changing environmental requirements. A central issue for our understanding of how locomotor circuits are specified and assembled is the extent to which sensory inputs are required as such systems develop. In his talk, Michael Bate (Cambridge, UK) will describe the effects of genetically eliminating sensory signaling or sensory structures on the embryonic and early postembryonic development of the peristaltic motor pattern of Drosophila.

Another aspect of genes being responsible for the formation of motor networks becomes apparent when postembryonic modifications of motor circuits follow a stereotypical developmental program with hormones acting as a timer. Hormonal control of postembryonic motor circuit remodeling is particularly apparent in holometabolous insects, such as Manduca and Drosophila. Among the genes that are directly activated by ecdysteroids is the Broad Complex (BRC). Christos Consoulas (Athens) will present recent data on the effects of BRC mutations on dendritic growth of an individually identified flight motoneuron during Drosophila metamorphosis.

Although ecdysteroids are the major player controlling motor circuit remodeling during insect metamorphosis, additional signals have important roles, too. A possible functional interplay between hormonal signals and activity-dependent mechanisms for structural and physiological changes of motoneurons will be addressed by Carsten Duch (Berlin). He will present data on the effects of selective electrical stimulations of identified motoneurons during Manduca development.

A particular feature of locomotory networks is their distribution over large parts of the central nervous system. ‘Higher locomotory centers’ in the brain may be important for the selection of motor patterns (‘motivation’), whereas segmental networks are important for controlling the rhythmical movements of limbs and joints with descending control necessary for a precise exertion of locomotory tasks. Roland Strauss (Würzburg) will show how mutations of different brain areas will differentially affect specific aspects of motor control inferring a modular control.

SYMPOSIUM 10

Thursday, June 13th 2003, 16.00–19.00, Lecture Hall 10

Chair: Carsten Duch and Hans-Joachim Pflüger (Berlin)

Insect neural and motor systems: from development to function and mechanics

16.00 *Michael Bate, Cambridge (UK)*
Embryonic assembly of neural circuitry underlying movement in *Drosophila*

16.25 *Christos Consoulas, Athens (Greece)*
A steroid-regulated gene is required for dendritic growth of motoneurons during metamorphosis of *Drosophila melanogaster*

16.50 *Carsten Duch, Berlin*
Stage-specific activity patterns affect motoneuron structure during *Manduca* metamorphosis

17.15 **Coffee Break**

17.45 *Roland Strauss, Würzburg*
Control of *Drosophila* walking and orientation behavior by functional subunits localized in different neuropils of the central brain

18.10 *Hans-Joachim Pflüger, Berlin*
Central modulatory neurons control fuel selection in flight muscle of migratory locust

18.30 *Fritz-Olaf Lehman, Ulm*
The control of vorticity in flying Drosophila

Neuromodulatory neurons are part of adult motor networks. Hans-Joachim Pflüger (Berlin) will report on their role as metabolic regulators. Combining electrophysiological with biochemical approaches reveals that octopaminergic neurons contribute to adjusting glycolytic flux in flight muscle to changing energy requirements.

The performance of a particular motor system is not only dependent on its neuronal components, but is to a great extent determined by mechanical properties. Fritz Olaf Lehmann (Ulm) studies the aerodynamics of flies' wing beats. The complex motion of insect wings produces fluid-mechanical forces that vary distinctly in both time and space. Flight mechanics, in turn, place distinct demands upon neural and muscular systems. These studies will also provide a link to what has become a most interesting application of fundamental studies on insect motor systems, robotics.

Introductory Remarks to Symposium 11

Adult neurogenesis

Gerd Kempermann

The adult brain generates new neurons throughout life. However, it seems to do so only in two privileged regions in the olfactory system and in the hippocampus. In astonishing contrast to this, stem or progenitor cells can be found in the entire adult brain. Potentially they could give rise to new neurons, because they do so after propagation in vitro. In the adult hippocampus, neurogenesis underlies a complex, activity-dependent regulation. First theories attempt to place adult neurogenesis into functional contexts. How can new neurons and thus neural stem cells contribute to hippocampal function? Is adult neurogenesis necessary for the function of the adult hippocampus? And what about the apparently quiescent stem or progenitor cells outside the neurogenic regions? There is increasing evidence that under certain conditions reactive neurogenesis is possible from these cells. However, the adult brain does regenerate poorly and does not seem to make use of the potential it harbors. Why is that so? Will it be possible to promote regeneration from these cells? And would they be functionally relevant?
Surprisingly, adult neurogenesis is linked to angiogenesis and bone marrow derived cells can form neurons in the adult brain. It is not clear whether bone marrow-derived brain cells reflect a fundamental biological principle or occur only under experimental conditions. In any case, adult neurogenesis and neural stem cell biology in general are much more complex than previously thought. The symposium is designed to address and discuss some of the topics of neural stem cell biology that have changed or will likely change fundamental neurobiological concepts.
The symposium begins with an overview on adult hippocampal neurogenesis, the role of neural progenitor cells in it and on how new neurons might contribute to hippocampal function. H. Georg Kuhn will introduce adult neurogenesis in the olfactory system. He will discuss, which role cell death plays in adult neurogenesis and will show new evidence, how intricately adult neurogenesis is linked to angiogenesis. Josef Priller will talk about the findings that at least under certain conditions bone-marrow derived cells can give rise to neurons and microglia in the adult brain. These data provoke very profound questions on the nature and origins of cellular plasticity in the adult brain. Among the human disorders which might be linked to stem or progenitor cell activity in the adult brain, temporal lobe epilepsy is of particular interest. Otmar Wiestler will present investigations on human hippocampal tissues, revealing that in the hippocampus of patients with temporal lobe epilepsy changes can be found that could be predicted from animal studies of adult hippocampal neurogenesis under the conditions of experimental seizures. This unique opportunity notwithstanding it remains difficult to study adult neurogenesis and neural stem cells in general in the adult human brain. In his presentation Mathias Höhn will explain new ideas on how new imaging technologies will allow us to visualize cellular plasticity in the living adult brain.

SYMPOSIUM 11

Thursday, June 12th 2003, 16.00–18.30, Lecture Hall 11

Chair: Gerd Kempermann (Berlin)

Adult neurogenesis

16.00 *Gerd Kempermann, Berlin*
From progenitor cells to new neurons in the adult brain: possible functions for adult hippocampal neurogenesis

16.30 *H. Georg Kuhn, Regensburg*
A link between neurogenesis and angiogenesis in the adult brain

17.00 *Josef Priller, Berlin*
Neurons and microglia from transplanted bone marrow

17.30 *Otmar D. Wiestler, Bonn*
Evidence for neurogenesis in human temporal lobe epilepsy

18.00 *Mathias Höhn, Köln*
How to track neurogenesis and stem cell activity in the adult brain

Introductory Remarks to Symposium 12

Invasive recording from the human brain – linking clinical applications with neurobiological research

Andreas K. Engel and Christian E. Elger

Currently, the vast majority of physiological data about the human brain are obtained by means of non-invasive methods, particularly functional MRI, EEG and MEG. These methods do not provide a sufficient resolution to permit the observation of physiological processes at the level of single cells or small cell assemblies. Therefore, our knowledge about physiological processes at the cellular level is largely inferential and based on comparative data from animal models. However, as part of therapeutical approaches it is in some cases possible, based on well defined clinical indications, to obtain data from invasive recordings in the human brain. This holds, e.g., for patients with neurodegenerative diseases of the basal ganglia (such as Parkinson's disease) or with epilepsies that are resistant to pharmacological treatment. In such cases, invasive recordings can be an indispensable means for both diagnosis of the respective disorder and for defining the appropriate therapeutical approach. Methodologically, this implies the use of electrodes for recording local field potientials reflecting the coherent activity of small cell assemblies, or even that of microelectrodes providing single-cell activity. In addition to their diagnostic relevance, such data are crucial for understanding the pathophysiology of the respective disorders and for linking animal models to the respective human disorders. Moreover, they can provide insights into basic mechanisms of normal brain functions such as movement control, sensory representation or memory formation. The goal of the symposium is to provide an overview of current developments in this field and to highlight approaches that link clinical applications with basic neurobiological research. Elger will introduce the approaches used in this research area and discuss the relevance of invasive methods for diagnosis of epilepsies. Subsequently, Engel et al. and Brown will focus on recordings in patients with movements disorders. The talk by Fernandez will review results on memory formation obtained in patients with epileptic disorders. Lachaux will present results on high-frequency oscillations during face perception in epileptic patients. Finally, Fried will discuss data that are relevant to understanding the role of single neurons in object representation in the human medial temporal lobe.

SYMPOSIUM 12

Thursday, June 12th, 16.00–19.00, Lecture Hall 105

Chair: Andreas K. Engel (Hamburg) and Christian E. Elger (Bonn)

Invasive recording from the human brain – linking clinical applications with neurobiological research

16.00 *Christian E. Elger, Bonn*
Introduction – the importance of invasive recording from the human brain

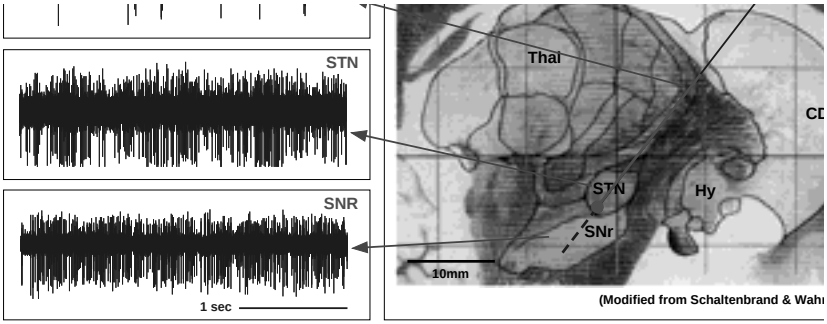
16.30 *Andreas K. Engel, Christian K. E. Moll, Christian Dohle, Niels Allert, Jürgen Voges, Ralf Lehrke, Hans-Joachim Freund and Volker Sturm, Hamburg, Jülich, Bonn, and Köln*
Microelectrode recordings from the human basal ganglia

17.00 *Peter Brown, London (UK)*
Task-related coupling in Parkinson's disease

17.30 *Guillen Fernandez, Jürgen Fell, Peter, Klaver, Susanne Weis and Christian E. Elger, Bonn and Nijmegen (The Netherlands)*
Rhinal-hippocampal coupling during human memory formation

18.00 *Jean-Philippe Lachaux, Paris (France)*
Increase of high-frequency (> 150 Hz) intracranial EEG activity during face perception in humans

18.30 *Itzhak Fried, Los Angeles (USA)*
Dynamics of single neurons during perception and memory tasks in the human medial temporal lobe



Targeting of the subthalamic nucleus by microelectrode recording in a patient with Parkinson's disease (Moll, Engel et al., unpublished)

Introductory Remarks to Symposium 13

Longterm potentiation and longterm depression of nociceptive CNS processing

Walter Magerl and Rolf-Detlef Treede

Longterm potentiation (LTP) and longterm depression (LTD) of synaptic transmission are well-accepted phenomena of cellular plasticity. Their prominent role in plasticity of the hippocampus and neocortex has prompted the generalization of these neurobiological mechanisms as general models of learning and memory in many species, including human. Due to the lack of convincing evidence of its contribution to acquisition and plasticity of complex behaviours, however, such a role is still disputed. Recent electrophysiological and functional evidence in animals and humans now suggest that LTP- and LTD-like plastic changes are also found in sensory and motor pathways. The symposium is centered around the role of LTP and LTD in the nociceptive system, which has long been known to display prominent plasticity.

The symposium will be opened by T. Bliss, who has first detected the phenomenon of LTP more than 30 years ago. His presentation will focus on development of the concept of LTP in the past decades. A. Artola will then focus on the role of LTP and LTD in hippocampus and visual neocortex, illustrating the central role of intracellular calcium concentration as a mechanism regulating a sliding balance of LTP and LTP. Beyond memory acquisition and consolidation additional mechanisms are imported in long-term storage of memories. In the absence of reinforcement, a resulting behavioural response will gradually diminish to be finally extinct. The importance of extinction, its cellular mechanisms and the role of the endocannabinoid system in extinction of aversive memory is highlighted by W. Zieglgänsberger.

The second half of the symposium is devoted to the role of LTP and LTD in sensory and motor systems. J. Sandkühler will demonstrate that synaptic LTP is a cellular mechanism of central sensitization in the nociceptive system. He will show that LTD and depotentiation can be induced in spinal cord that involve different signal transduction pathways. Eventually, the plasticity of spinal nociceptive processing may be paralled by analogues perceptual changes. W. Magerl will detail input-specific and heterosynaptic functional consequences of LTP- and LTD-inducing stimulus protocols on human pain perception demonstrating the diversity of these mechanisms. The symposium will be closed by U. Ziemann, who will illustrate using the method of transcranial magnetic stimulation that LTP-like plasticity of the human motor cortex is characterized by the principles of input-specificity, cooperativity and associativity. The induction of motor cortex LTP is modulated by dopaminergic, noradrenergic and cholinergic mechanisms.

SYMPOSIUM 13

Friday, June 13th 2003, 9.00–12.00, Lecture Hall 7

Chair: Walter Magerl (Mainz) and Rolf-Detlef-Treede (Mainz)

Long-term potentiation and long-term depression of nociceptive CNS processing

9.00 *Tim Bliss (London, UK)*
Long-term potentiation after 30 years – where do we stand?

9.25 *Alain Artola (Antwerp, Belgium)*
Use-dependent synaptic plasticities in the hippocampus and visual cortex

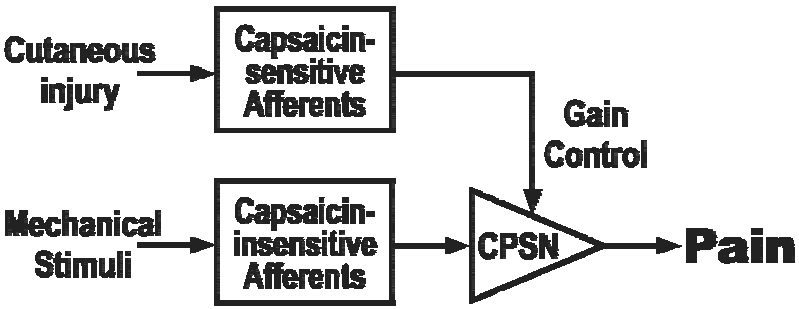
9.50 *Walter Zieglgänsberger (Munich)*
Extinction of aversive memory – a role for endocannabinoids?

10.15 **Coffee Break**

10.45 *Jürgen Sandkühler (Vienna, Austria)*
Synaptic LTP and LTD in spinal pathways

11.10 *Walter Magerl (Mainz)*
LTP- and LTD-like plasticity of human pain perception

11.35 *Ulf Ziemann (Frankfurt)*
LTP-like plasticity of the human motor cortex



Introductory Remarks to Symposium 14

Towards a molecular understanding of behavior

Ralf Heinrich and Edward A. Kravitz

Behavior is generated by the functional interplay of neurons within and between neural networks. The contribution of an individual neuron to the generation of a particular behavior is determined by the currently expressed molecular machinery (transmitters, receptors, channels, second messengers etc.) involved in transforming incoming stimuli into characteristically patterned electrical activity. The responsiveness of a neuron or a neural circuit is not fixed; rather it is dynamically modulated by previous activity to produce adaptive changes in the occurrence and performance of a particular behavior. Alterations in neural activity may persist for periods ranging from seconds to lifetime of an organism and may result from reversible modulation of intracellular signaling pathways, altered gene expression or changes in morphology and synaptic coupling.

This symposium presents studies using both invertebrate and vertebrate models in which recent progress has allowed investigators to directly link molecular mechanisms, neural functions and the control of specific behaviors.

Two presentations deal with long-term changes in central pattern generating circuits. Ron Harris-Warrick describes a homeostatic mechanism in crustacean stomatogastric ganglion neurons that compensates for the overexpression of a potassium channel encoded by the shal gene by upregulation of a hyperpolarization-activated inward current. The consequences of this change are that the firing properties of the neurons are not significantly changed. David Parker and Sarah Bevan demonstrate multiple effects of the neuropeptide substance P on second messenger pathways, RNA- and protein synthesis and ultrastructural changes in synaptic terminals. These cause characteristic changes in the activity of the lamprey spinal cord locomotor network.

The next two contributions deal with the functional analysis of second messenger pathways in insect brains. Ralf Heinrich focuses on the role of these pathways in specific arousal and the selection of situation-specific acoustic communication patterns in grasshoppers. Uli Müller describes, how a characteristic temporal activation of different signaling pathways contributes to distinct features of memory formation during associative learning of honeybees.

Social behaviors are considered to be controlled by multiple internal and external signals. Edward Kravitz introduces *Drosophila melanogaster* as a genetically accessible preparation for studies on the central nervous control of aggression. Conditions have been defined in which fighting behavior is routinely seen between pairs of male flies, the behavior has been quantitatively analyzed, and through the use of the GAL4/UAS system driving the expression of a temperature sensitive form of the protein dynamin, individual amine neuron types can be switched on and off while animals are fighting to observe effects on the behavior. In a comparative study on monogamous and nonmonogamous voles, Larry Young identified differences in a specific repetitive sequence in the promotor of the gene coding for the V1a vasopressin receptor. The presence or absence of this sequence determines both the expression pattern of the V1a receptor in the brain and the ability to form pair bonds following mating-induced release of arginine vasopressin in the central nervous system.

SYMPOSIUM 14

Friday, June 13th 2003, 9.00- 12.00, Lecture Hall 9

Chair: Ralf Heinrich (Göttingen) and Edward A. Kravitz (Boston, USA)

Towards a molecular understanding of behavior

9.00 *Ron M. Harris-Warrick, Ithaca (USA)*
Potassium channels and activity-independent homeostasis in the crustacean stomatogastric ganglion

9.25 *David Parker and Sarah Bevan, Cambridge (UK)*
Cellular and synaptic effects contributing to long-term neuropeptide-mediated modulation of a spinal cord locomotor network

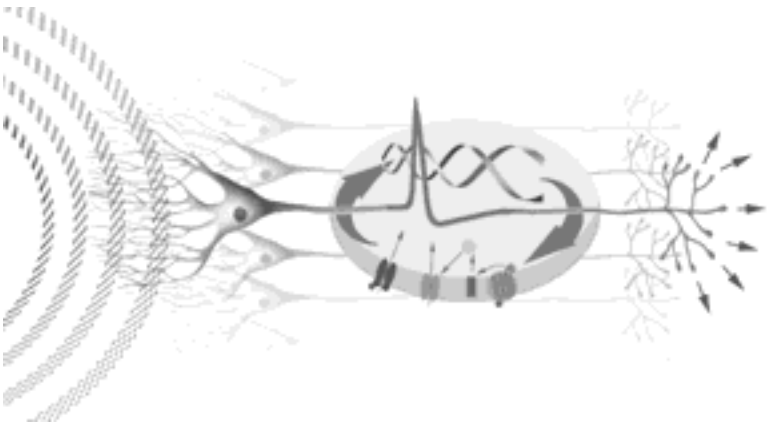
9.50 *Ralf Heinrich, Göttingen*
Selection and control of behavior by intracellular signaling pathways in the insect brain

10.15 **Coffee Break**

10.45 *Uli Müller, Berlin*
Second messenger cascades: Major mediators of memory formation

11.10 *Edward A. Kravitz, Boston (USA)*
Genetic studies on a fruit fly model of aggression

11.35 *Larry J. Young, Atlanta (USA)*
Vasopressin and social attachment in a monogamous mammal



Introductory Remarks to Symposium 15

Peptide co-transmitters in identified neurons

Petra Skiebe and Sabine Kreissl

The function of the nervous system not only depends on the connectivity of the neuronal networks, but also on the transmitters released by the neurons. Single neurons contain not one, but multiple transmitters, which often included neuropeptides. The number of isolated neuropeptides is large (more than one hundred) compared with the number of classical transmitters, and it is therefore necessary to study the effect caused by the release of a transmitter 'cocktail', in order to understand the plasticity of the brain. To investigate the role of peptide co-transmission, it is advantageous to work on identified neurons with known peptidergic co-transmitters, known function and known targets. These prerequisites are met in some crustacean systems in which peptides can be investigated on the level of single identified neurons using a combination of biochemical, anatomical and electrophysiological methods and by modeling.

Peptides are isolated by biochemical methods and their distribution can then be investigated by immunocytochemical methods in order to get a first idea concerning their function. Combining anatomical with genetic methods shows that a differential distribution of peptides can be due to a different distribution of precursors, resulting in putatively diverse physiological actions (Heiner Dircksen). The co-transmitters not only vary between neurons but also in a given neuron depending on the developmental stage (Valèrie Fénelon). Motoneurons, for example, transiently express FMRFamide-like peptides, suggesting a possible role in the establishment of the mature neuromuscular junction. Furthermore, different modulatory projecting neurons acquire FMRFamide-like immunoreactivity at different developmental stages, indicating different functional roles during development. Using only immunocytochemistry to identify peptides present in a given neuron can be misleading, since the antibody might recognize different peptides or different members of the same family. By combining immunocytochemistry with MALDI-TOF (Matrix-assisted laser desorption time-of-flight) mass spectrometry, it is possible to identify particular peptides or even multiple members of a peptide family in single identified neurons, which is necessary in order to investigate co-transmission or the role of different family members (Petra Skiebe).

One good model system to investigate the function of peptides is the neuromuscular junction. Sabine Kreissl and co-workers show that two peptides elicit antagonistic effects on muscles. This is interesting because they are likely to be co-released either as transmitters from a pair of identified motoneurons or as hormones from the pericardial organ, a major neurohaemal organ in crustaceans. That the co-transmitters of a particular modulatory interneuron have different effects depending on the circuit they are influencing has been shown using the stomatogastric nervous system (Wolgang Stein). These co-transmitters have a converging action in one circuit, while a diverging action on the second. Combining dynamic clamp experiments and modeling, the mechanisms through which the peptide Red Pigment-Concentrating Hormone (RPCH) shape the output of a network of identified neurons by acting on multiple cellular and synaptic targets have been analyzed (Prinz). RPCH has three different effects, and due to this combination of methods it is possible to judge the contribution of each of the effects on the output of the neural network.

SYMPOSIUM 15

Friday, June 13th 9.00–12.00, Lecture Hall 104

Chair: Petra Skiebe (Berlin) and Sabine Kreissl (Konstanz)

Peptide co-transmitters in identified neurons

9.00 *Heiner Dircksen, Bonn*
Differential distributions and functions of orcokinins and orcomyotropin, novel partially co-localized peptides in crayfish sensory, motor, interneuronal and neurosecretory cells

9.25 *Valèrie Fénelon, Bordeaux (France)*
Ontogeny of modulatory systems

9.50 *Petra Skiebe, Berlin*
Combining MALDI-TOF MS and immunocytochemistry to identify peptide co-transmitters in single identified neurons

10.15 **Coffee Break**

10.45 *Sabine Kreissl, Torsten Weiss, Werner Rathmayer, Konstanz*
Antagonistic modulation of neuromuscular efficacy by two co-localized peptides

11.10 *Wolfgang Stein, Ulm*
Convergence and divergence of peptide co-transmitter actions: Functional consequences in a multifunctional network

11.35 *Astrid Prinz, Watham (USA)*
Dissecting and modeling the actions of neuromodulatory peptides on multiple targets in a network of identified neurons

Introductory Remarks to Symposium 16

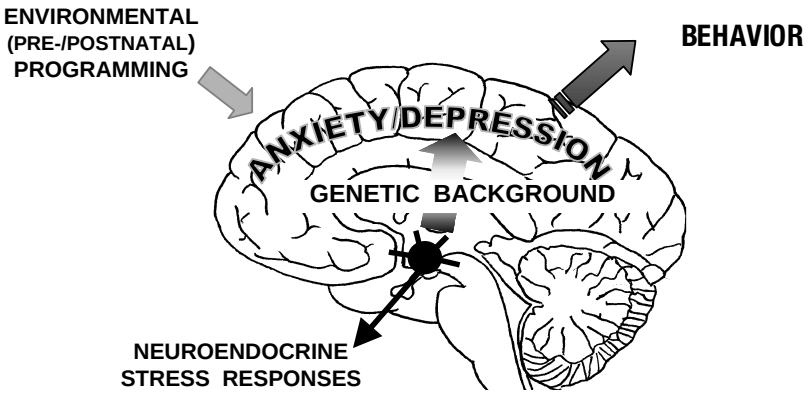
Early environmental programming: Molecular, neuroanatomical, neuroendocrine and behavioural effects

Inga Neumann and Katharina Braun

There is profound evidence that the development and manifestation of various psychiatric diseases, such as major depression or anxiety disorders as well as cognitive deficits are dependent on prenatal and immediate postnatal factors. Whereas the basic wiring of the mammalian central nervous system is genetically programmed, its fine tuning throughout different phases of infancy, childhood, and adulthood are highly dependent on environmental conditions. Early experiences which occur during phases of elevated neuronal and synaptic plasticity appear to „imprint"" patterns of synaptic connectivity, neural circuitries, and neuronal and neuroendocrine activity in the infant brain. In particular the wiring patterns of limbic circuits, which are relevant for learning and memory, emotional behavior and regulation of neuroendocrine stress responsiveness are modified by early emotional experiences.

The symposium will present evidence that exposure to pre- or postnatal stress permanently modifies brain functions at various levels. In the prenatal period, pharmacological or physiological exposure to excessive levels of glucocorticoids, which can easily cross the placentar-barrier, is known not only to reduce birth weight but also to be associated with an increased risk of cardiovascular, metabolic, neuroendocrine and emotional pathophysiologies in adulthood. Jonathan Seckl will demonstrate the importance of the placentar glucocorticoid „buffer"" enzyme 11β-hydroxysteroid dehydrogenase in preventing the effects of prenatal stress relevant for rodents and humans. With respect to stressful emotional experiences during the early postnatal period, Katharina Braun will present data from two rodent models which indicate that repeated or chronic parental separation affects the synaptic reorganization in the anterior cingulate cortex, hippocampus and amygdala.

New insights into the influence of early postnatal maternal deprivation versus handling on the development of neuroendocrince function including the behavioural corre-



SYMPOSIUM 16

Friday, June 13th 2003, 9.00–12.00, Lecture Hall 10

Chair: Inga Neumann (Regensburg) and Katharina Braun (Magdeburg)

Early environmental programming: Molecular, neuroanatomical, neuroendocrine and behavioural effects

9.00 *Jonathan Seckl, Edinburgh (UK)*
Prenatal glucocorticoid programming of adult pathophysiology

9.30 *Katharina Braun, Magdeburg*
Effects of parental separation on the maturation of limbic circuits

10.00 **Coffee Break**

10.30 *Paul Plotsky, Atlanta (USA)*
Molecular and neuroendocrine consequences of postnatal separation versus handling

11.00 *Mathias Schmidt, Leiden (Netherlands)*
Molecular and neuroendocrine effects of maternal deprivation in mice lacking the CRH receptor type 1

11.30 *Inga Neumann, Regensburg*
Effects of early life stress: dependency on gender and the genetic predisposition to high and low anxiety

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lates will be presented by Paul Plotsky. With the aim to directly link the behavioural and neuroendocrine effects of postnatal stress to the activity of the corticotropin releasing hormone (CRH) system in the brain, in particular within the hypothalamus, Mathias Schmidt will show that the CRH receptor is essential for the dysregulation of the corticosterone response, of hypothalamic CRH expression and hippocampal mineralocorticoid receptor expression following maternal deprivation using CRH receptor type 1 deficient mice. Such complex pathological adaptations of neuronal wiring, emotionality and neuroendocrine function as a result of prenatal stress or postnatal maternal deprivation may be dependent on the genetic predisposition of the individual animal. Inga Neumann will report on differences in the efficacy of stressful events very early in life between male and female offspring with males being more vulnerable to postnatal stress. Furthermore, the direction of effects of perinatal manipulation differs markedly between animals with genetically determined differences in emotionality.
The data presented in this symposium will support the hypothesis that impoverished or stressful environmental stimulation and traumatic socio-emotional experience permanently impair the formation of functional brain pathways, neuroendocrine functions and behaviour, which may eventually lead to a variety of mental disorders. The analysis of the neurobiology of such self-organizing plastic systems may begin to change our conceptual approaches to psychopathology and open new avenues of therapeutics for psychiatric diseases.

Introductory Remarks to Symposium 17

New forms of cerebellar signaling

Jana Hartmann and Arthur Konnerth

Recent years have revealed unexpected mechanisms of neuronal signaling in the cerebellum. Particularly retrograde signaling at Purkinje cell synapses has triggered a wide interest among neuroscientists. Depolarization-induced suppression of inhibition (DSI) is a calcium-dependent transient suppression of transmitter release following postsynaptic depolarization at the interneuron-Purkinje cell synapse. Previous work of Alain Marty and colleagues indicated that DSI is induced by the release of a retrograde messenger. Alain Marty will present new data, which allow a quantitative estimate of the changes in interneuron excitability and the probability of release at interneuron-Purkinje cell synapses connected with DSI. Wade Regehr and coworkers have demonstrated that DSI is not restricted to inhibitory synapses but presents a more general type of synaptic regulation. They identified endogenous cannabinoids as the retrograde messenger responsible for DSI and depolarization-induced suppression of excitation (DSE) in the hippocampus and cerebellum, respectively. In his talk Wade Regehr will focus on new results from his laboratory about the role of the endocannabinoid system at Purkinje cell synapses. At excitatory Purkinje cell synapses either calcium influx following postsynaptic depolarization or calcium release from intracellular stores due to activation of postsynaptic metabotropic glutamate receptors (mGluRs) is sufficient to induce the production of endocannabinoids. This putative synergistic action of both pathways in the regulation of transmitter release is suggested by recent findings from Masanobu Kano's laboratory and will be the topic of his presentation. The role of presynaptic calcium stores in synaptic transmission at inhibitory interneuron-Purkinje cell synapses will be discussed by Isabel Llano. She and her colleagues detected for the first time spontaneous ryanodine receptor-mediated calcium transients in presynaptic terminals, a mechanism that will be covered in her presentation. Jana Hartmann will review recent findings about neurotrophin-mediated signaling in Purkinje cells. Brain-derived neurotrophic factor (BDNF) has a rapid excitatory action in Purkinje cells and is capable of LTD-induction. Thus, in the cerebellum BDNF is a transmitter-like signaling molecule that modifies persistently synaptic transmission even in response to a single brief exposure. The cerebellar cortical output is shaped by the interplay between Purkinje cell somata and dendrites. Mechanisms for coincidence detection between parallel fiber and climbing fiber inputs in dendrites of Purkinje cells will be described by Michael Häusser. Based on results from *in vivo* dendritic patch-clamp recordings he will show the relation between patterns of synaptic activity and specific patterns of spiking.

SYMPOSIUM 17

Friday, June 13th 2003, 9.00–12.00, Lecture Hall 11

Chair: Arthur Konnerth and Jana Hartmann (Munich)

New Forms of Cerebellar Signaling

9.00 *Alain Marty, Paris (France)*
Mechanisms of retrograde synaptic modulation at interneuron-Purkinje cell synapse

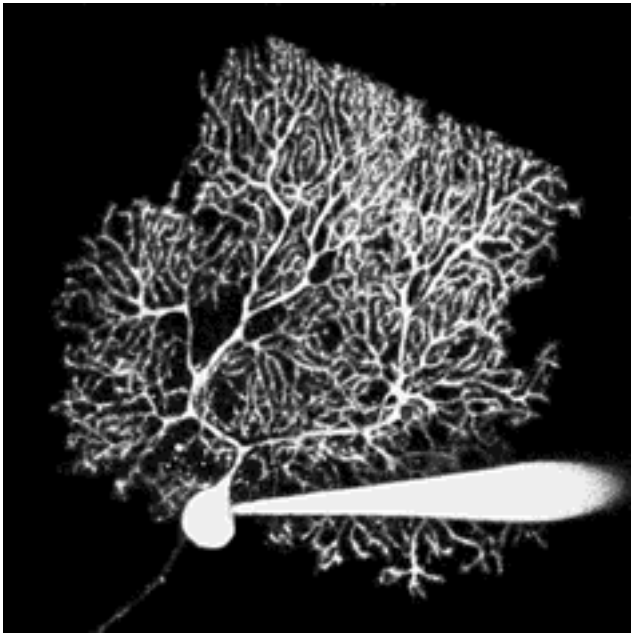
9.30 *Wade Regehr, Boston (USA)*
Retrograde modulation of synapses by endocannabinoids

10.00 *Masanobu Kano, Kanazawa (Japan)*
Endocannabinoid-mediated retrograde signaling triggered by activation of postsynaptic metabotropic glutamate receptors in cerebellar Purkinje cells

10.30 *Isabel Llano, Paris (France)*
Probing the role of intracellular calcium stores in presynaptic calcium signalling

11.00 *Jana Hartmann, München*
BDNF-mediated rapid signaling in cerebellar Purkinje cells

11.30 *Michael Häusser, London (UK)*
Dendritic integration in cerebellar Purkinje cells



Introductory Remarks to Symposium 18

Complex sensory processing in the vertebrate midbrain

Bernhard Gaese and Harald Luksch

Analyzing sensory information and creating useful behavioral output from it is, in short, the general function of the brain. Although all parts of the brain are involved in this task, the production of complex behavior in avian and mammalian vertebrates is usually thought of as being strongly related to forebrain structures. The midbrain, on the other hand, is mainly considered to be involved in the production of simple orienting behavior, e.g. saccadic eye movements. The aim of this symposium is to broaden the scope by taking together recent results showing how midbrain structures are involved in rather complex processing, how these structures integrate information from different sensory systems, and how the processing is connected to that in other sensorimotor loops.

We will start with an overview of the key elements for visual processing in the pigeon optic tectum. Based on a detailed analysis of the anatomical connections this will show how the retinotopically mapped input to the tectum is transformed into topographies more closely related to specific functions such as movement and looming detection (Güntürkün). It will then be shown how the processing of one important aspect of visual stimuli, visual motion, is carried out at the cellular level in the optic tectum of birds, and how the biophysical features of neurons and local networks are optimized for such a specific processing (Luksch). The mammalian counterpart of the avian optic tectum, the superior colliculus, has been analysed in comparable detail. Examples of neuronal circuits and related molecular structures involved in visual processing will be presented, with an emphasis on the inhibitory activity and its importance for the processing within and in between midbrain nuclei (Schmidt). The visual input into these structures is, among others, used to generate orienting responses towards objects of interest. Usually large sets of distributed neurons are involved in this visuo-motor transformation. Principles of processing underlying the integration of such distributed activity was analyzed in the cat and rat (Engler, Kang, Brecht, and Engel). In addition to the predominant visual afferents, the superior colliculus is a centre for the integration of input from different sensory modalities. As an example, the processing of auditory spatial activity and its organization into a map of auditory space in the mammalian superior colliculus making use of the virtual space technique will then be presented (King). Finally, the integration of orienting behavior into the actual behavioral context is discussed. This is done by presenting new data on the visual-auditory integration subserving the spatial orienting of attention in barn owls and rats. The attention-related activity, found already at the midbrain level, is seen as one of several mechanisms ensuring a consistent behavioral output by integrating sensory input from different modalities and from different levels of analysis (Gaese).

Taken together, these presentations demonstrate how complex the processing of information at the midbrain level is, and how the resulting activity is integrated into the entire network of the brain to produce adaptive behavior.

SYMPOSIUM 18

Friday, June 13th 2003, 9.00–12.00, Lecture Hall 105

Chair: Bernhard Gaese (Frankfurt) and Harald Luksch (Aachen)

Complex sensory processing in the vertebrate midbrain

9.00 **Introductory Remarks**

9.05 *Onur Güntürkün, Bochum*
From retinotopy to functionotopy: Structural organization of parallel information processing within the tectofugal visual system of pigeon

9.30 *Harald Luksch, Aachen*
The biophysical and cellular basis of object-motion detection in the avian optic tectum

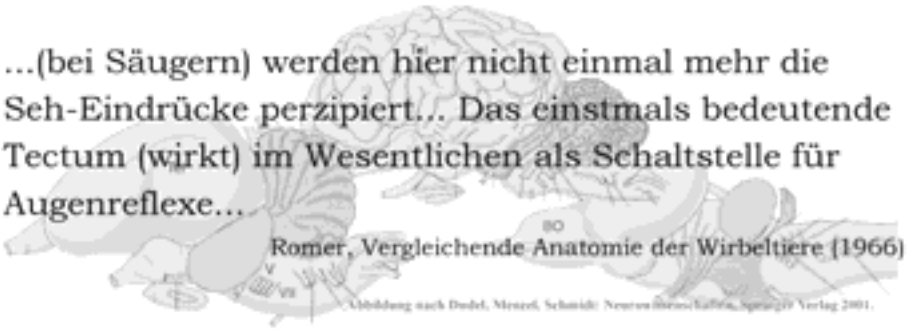
9.55 *Matthias Schmidt, Bochum*
Local inhibitory mechanisms control information flow in the mammalian superior colliculus

10.20 Coffee Break

10.45 *Gerhard Engler, Jun-Suk Kang, Michael Brecht and Andreas K. Engel, Hamburg, Frankfurt and Heidelberg*
Role of neural synchrony for response selection in the superior colliculus

11.10 *Andrew J. King, Oxford (UK)*
Computing a neural representation of auditory space in the mammalian superior colliculus

11.35 *Bernhard H. Gaese (Frankfurt a.M.)*
Cognitive influences on auditory processing in the vertebrate midbrain



Introductory Remarks to Symposium 19

Function and dysfunction of the amygdala: fear and epilepsy

Deniz M. Yilmazer-Hanke and Oliver Stork

Amygdala research currently focusses on two related and clinically relevant aspects of amygdalar function and dysfunction, fear and epilepsy. These appear to interact in human and experimental temporal lobe epilepsy (TLE), as intracerebral recordings have suggested a contribution of the amygdala not only to generation and propagation of focal seizures but also to the elicitation of ictal fear during temporal lobe seizure activity. The detailed morphological and physiological characterization of the amygdala's extrinsic and intrinsic connectivity in recent years now allows to approach the mechanisms underlying these (dys-)functions (Pitkänen et al., 1997; 2000). In essence, it is believed that the basolateral complex of the amygdala acts as a „sensory gate"" and integrates exteroceptive and interoceptive sensory information of different modalities, which is then relayed via the central nucleus to vegetative centres in the hypothalamus and brainstem. Much physiological work has focussed on the glutamatergic transmission at sensory afferents to the lateral amygdala, such as the NMDA-receptor dependent and long term potentiation-like enhancement of neural activity after fear conditioning training, and the metabotropic glutamate receptor-dependent epileptogenesis (McKernann and Shinnick-Gallagher, 1997; Neugebauer et al., 1997). The importance of GABAergic interneurons in the basolateral complex, that regulate the activity of pyramidal projection neurons by feed-forward and feed-back inhibition, is also widely appreciated. Evidence suggest that, in fact, a loss of perisomatic inhibition in the amygdala may relate to the enhanced excitability of glutamatergic projection neurons in human TLE. Various subpopulations of GABAergic and peptidergic neurons in the basolateral complex and central amygdala further play important and specific roles in the regulation of stress and fear responses. The prominent monoaminergic innervation of the amygdala has been implicated in the control of many of these neurons. Amygdala-hippocampal interactions are particularly important for cognitive aspects of fear conditioning, as well as stress-related effects on hippocampal synaptic plasticity and hippocampus-dependent memory consolidation (Akirav and Richter-Levin, 1999). On a molecular level it has become evident that the activation of various protein-kinase pathways leads to an activation of gene-expression from cyclicAMP-responsive elements and subsequent induction of immediate early gene transcription factors, as well as signal transduction and structural reorganisation factors in the amygdala.

Akirav I, Richter-Levin G (1999) J Neurosci 19: 10530–35.
McKernan MG, Shinnick-Gallagher P (1997) Nature 390:607–611
Neugebauer V, Keele NB, Shinnick-Gallagher P (1997) J Neurosci 17:983–95.
Pitkänen A, Savander V, LeDoux JE (1997) Trends Neurosci: 11:517–23.
Pitkanen A, Pikkarainen M, Nurminen N, Ylinen A (2000) Ann NY Acad Sci 911:369–91.

SYMPOSIUM 19

Saturday, June 14th 2003, 15.00–18.00, Lecture Hall 7

Chair: Deniz M. Yilmazer-Hanke and Oliver Stork

Function and dysfunction of the amygdala: fear and epilepsy

15.00 *Deniz M. Yilmazer-Hanke*
Introductory Remarks

15.05 *Deniz M. Yilmazer-Hanke*
Cellular und structural alterations leading to increased excitability of the amygdala in human temporal lobe epilepsy

15.30 *Patricia Shinnick-Gallagher*
The amygdala in conditioned fear and epilepsy

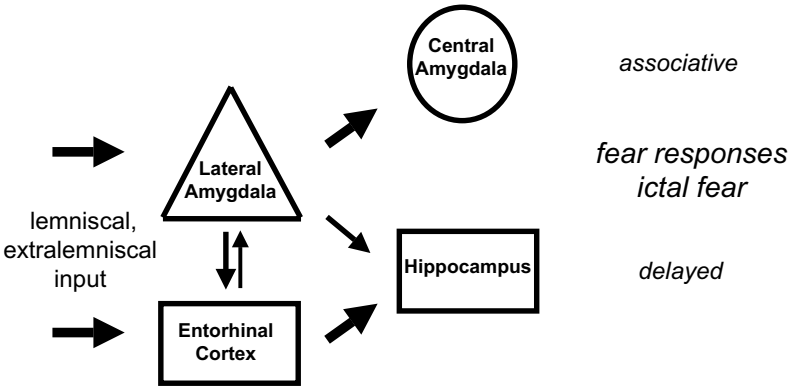
15.55 *Doris Albrecht*
Effects of amygdaloid kindling on post-ictal plasticity in the lateral nucleus of the amygdala

16.20 *Katarzyna Majak*
Amygdalo-hippocampal connectivity and its activation during fear conditioning

16.45 *Esther Asan*
Monoaminergic afferents and their targets in the rat amygdala: implications for stress and fear responses

17.10 *Gal Richter-Levin*
Emotional modulation of memory – Stress modulation of plasticity in the hippocampus and amygdala

17.35 *Oliver Stork*
Molecular mechanisms of fear memory: gene expression and transgenic approaches



Introductory Remarks to Symposium 20

Transsynaptic signalling at central glutamatergic synapses

Volkmar Leßmann and Kurt Gottmann

Central synapses are intercellular junctions dedicated to fast presynaptic neurotransmitter release and to sensitive postsynaptic transmitter receptiveness. Exact alignment of pre- and postsynaptic specializations is fundamental to synaptic function. Transsynaptic signalling in both anterograde and retrograde direction is thought to control and regulate synaptic organization and plasticity. One class of molecular mechanisms involves the release of protein factors, binding to their cognate receptors and triggering of downstream signalling cascades. Prominent examples for such a mechanism are neurotrophins, in particular brain-derived neurotrophic factor (BDNF).

Other mechanisms consist of transsynaptic interaction of membrane-bound molecules in a ligand-receptor mode triggering asymmetric signal transduction pathways. Ephrins and eph receptors are a particularly well studied, prototypic pair of this type. Symmetric transsynaptic interactions are mediated by classical cell adhesion molecules, such as N-cadherin. These adhesion molecules bind to each other in a homophilic way, giving rise to symmetric signalling in the pre- and postsynaptic neuron. Last not least, glial cells have recently been found to release factors that are essential for the formation and maintenance of functional synapses, thus supporting the emerging concept of a tripartite synapse.

This symposium concentrates on glutamatergic synapses, because transsynaptic signalling in long-term synaptic plasticity has been a major focus of neurobiological research at these excitatory central synapses. Tobias Bonhoeffer will introduce the essential role of neurotrophins, such as BDNF, in long-term potentiation and will present recent data on the receptor mechanisms involved. Volkmar Lessmann will continue by presenting work on the sites and mechanisms of neurotrophin secretion from neurons, a topic that has become a major focus in the field. Finally, Arthur Konnerth will show exciting new data on the mechanism of a fast, transmitter-like postsynaptic action of BDNF that appears to play an unexpected role in long-term potentiation.

In the second part of the symposium, Rüdiger Klein will focus on transsynaptic interaction of membrane-bound molecules and will present new work in the rapidly expanding field of the synaptic role of ephrins and eph receptors. This will be followed by a talk (Kay Jüngling/Kurt Gottmann) on the classical cell adhesion molecule N-cadherin regulating presynaptic function. Work employing in vitro differentiation of N-cadherin-deficient embryonic stem cells will be presented. Concluding the symposium, Frank Pfrieger will present the recent discovery, that cholesterol released by glial cells is an essential player in synapse formation and functional plasticity.

SYMPOSIUM 20

Saturday, June 14th 2003, 15.00–18.00, Lecture Hall 9

Chair: Volkmar Leßmann (Mainz) and Kurt Gottmann (Bochum)

Transsynaptic signalling at central glutamatergic synapses

15.00 *Tobias Bonhoeffer, Munich-Martinsried*
Neurotrophins and synaptic plasticity

15.25 *Volkmar Leßmann, Mainz*
Synaptic targeting and secretion of neurotrophins

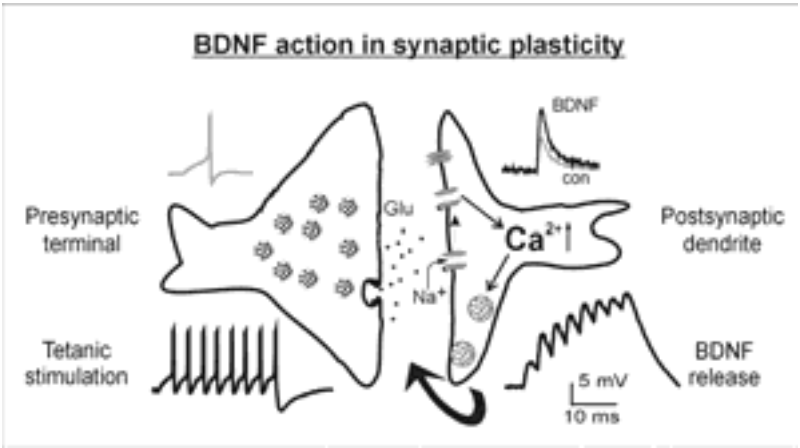
15.50 *Arthur Konnerth, Munich*
Regulation of glutamatergic transmission through BDNF-evoked dendritic depolarization

16.15 **Coffee Break**

16.45 *Rüdiger Klein, Munich-Martinsried*
Ephrin and Eph receptor functions in development and synaptic plasticity

17.10 *Kay Jüngling and Kurt Gottmann, Bochum*
Regulation of presynaptic function by synaptic adhesion molecules: role of N-cadherin

17.35 *Frank W. Pfrieger, Strasbourg (France*
Role of cholesterol in synapse development



Introductory Remarks to Symposium 21

Molecular basis of axonal damage in inflammatory and degenerative CNS diseases

Harald Neumann and Mathias Bähr

Selective early loss of axons, dendrites and synapses is a common feature of several neurodegenerative and neuroinflammatory diseases including multiple sclerosis, traumatic or ischemic brain injury and Alzheimer's disease. Secondary degeneration of neurons is often the consequence of this primary neurite damage, resulting in permanent neurological deficits in patients.

The exact molecular mechanisms of primary axonal damage are not exactly known, but significant progress has been achieved in our understanding of the respective pathologies, as will be highlighted in the symposium:

In multiple sclerosis demyelination and inflammatory reactions are involved in axonal damage and loss of neurites (Brück and Nave). Axonal damage in multiple sclerosis is responsible for the permanent deficits of the patients. The damage of axons is mainly observed in the acute demyelinating multiple sclerosis lesions and is associated with the number of infiltrating macrophages and cytotoxic CD8+ T-lymphocytes.

In Alzheimer's disease intracellular polymerisation of axonal (mutated) proteins and extracellular amyloid aggregates appear to be the cause of axonal damage (Götz and Perry). Beta-amyloid plaques or polymerized beta-amyloid peptide can promote the formation of neurofibrillary tangles and loss of axonal teminals. Activation of the innate immune response (e.g. microglia, complement) is linked to the lesion site in Alzheimer's disease and might perpetuate the disease process.

Recently, experimental repair strategies have been designed to prevent further damage and functionally improve deficits caused by axonal and secondary neuronal degeneration: One exciting strategy aims at stimulating axonal outgrowth from lesioned neurons by interference with inhibitory signalling molecules such as Nogo (Kerschensteiner). Furthermore, stem cells that differentiate into oligodendrocytes are applied locally to induce remyelination (Brüstle).

In summary, the symposium will highlight the emerging research on the molecular mechanism of axonal damage and elucidate new ways for protective and cell replacement therapy.

SYMPOSIUM 21

Saturday, June 14th 2003, 15.00–18.00, Lecture Hall 104

Chair: Harald Neumann (Göttingen) and Mathias Bähr (Göttingen)

Molecular basis of axonal damage in inflammatory and degenerative CNS diseases

15.00 *Jürgen Götz, Zürich (Switzerland)*
Linking beta-amyloid plaques to neurofibrillary tangle formation in an Alzheimer's disease mouse model

15.25 *Hugh Perry, Southampton (UK)*
Inflammation in the CNS and its potential to trigger an axon 'self-destruct' programme

15.50 *Martin Kerschensteiner, Zürich (Switzerland)*
Early aspects of axonal damage in spinal cord injury

16.15 **Coffee Break**

16.45 *Wolfgang Brück, Göttingen*
Axonal pathology in multiple sclerosis

17.10 *Klaus-Armin Nave, Göttingen*
Axon-glia interactions in transgenic models of myelin disease

17.35 *Oliver Brüstle, Bonn*
Stem cell based therapy of demyelinating diseases

Introductory Remarks to Symposium 22

Neurotrauma: a trigger for schizophrenia?

Hannelore Ehrenreich, Anna-Leena Sirén and Eckart Rüther

Despite general agreement on the significance of a genetic predisposition, the etiology of schizophrenic psychosis remains obscure. There is, however, strong evidence for a number of co-factors (e.g. neurotrauma, drug abuse) that influence manifestation and course of schizophrenia. These findings point to a dual origin of the disease-determining processes: neurodevelopmental and neurodegenerative.

Epidemiological studies have established a connection between head injury and psychosis. As will be demonstrated by D. Malaspina, traumatic brain injury (TBI) significantly increases the risk for schizophrenia. Progressive deterioration of cognitive functions together with progressive ventricular enlargement in imaging studies are typical features of post TBI and schizophrenia supporting a neurodegenerative component in the pathophysiology of schizophrenia. The possible mechanisms underlying this connection will be further addressed in the presentation of A.-L. Sirén and H. Ehrenreich demonstrating distinct and selective behavioral deficits and significant enlargement of the ventricle system months after application of a discrete lesion of the right parietal cortex in mice. The presentation of J. Price will address the role of possible reactivation of developmental gene expression after brain damage. His contribution will provide evidence for a role of OCT-6 as a pathophysiological marker that is turned on in both schizophrenia and after neurotrauma. Using high-resolution MRI scans, Giedd will provide evidence for continuous degenerative processes in schizophrenia. His studies demonstrate dynamic patterns of accelerated gray matter loss in the brains of childhood-onset schizophrenia with earliest defects in the parietal association cortex, an area where gray matter loss is known to be strongly associated with environmental risk factors such as TBI. A reduction in interneuronal neuropil in the prefrontal cortex that has been shown to be a prominent feature of cortical pathology in schizophrenia suggesting that subtle changes in cellular architecture and brain circuitry may have a devastating impact on cortical function. T. Falkai will address the role of inflammatory mechanisms in these events. T. Pollmächer will further elaborate on involvement of cytokines in the pathophysiology of schizophrenia.

SYMPOSIUM 22

Saturday, June 14th 2003, 15:00–18:00, Lecture Hall 10

Chair: Hannelore Ehrenreich and Eckart Rüther (Göttingen)

Neurotrauma: a trigger for schizophrenia?

15.00 *Hannelore Ehrenreich, Göttingen*
Opening remarks

15.05 *Dolores Malaspina, New York*
Neurotrauma and schizophrenia: Epidemiology

15.30 *Anna-Leena Sirén, Göttingen*
Late consequences of neurotrauma

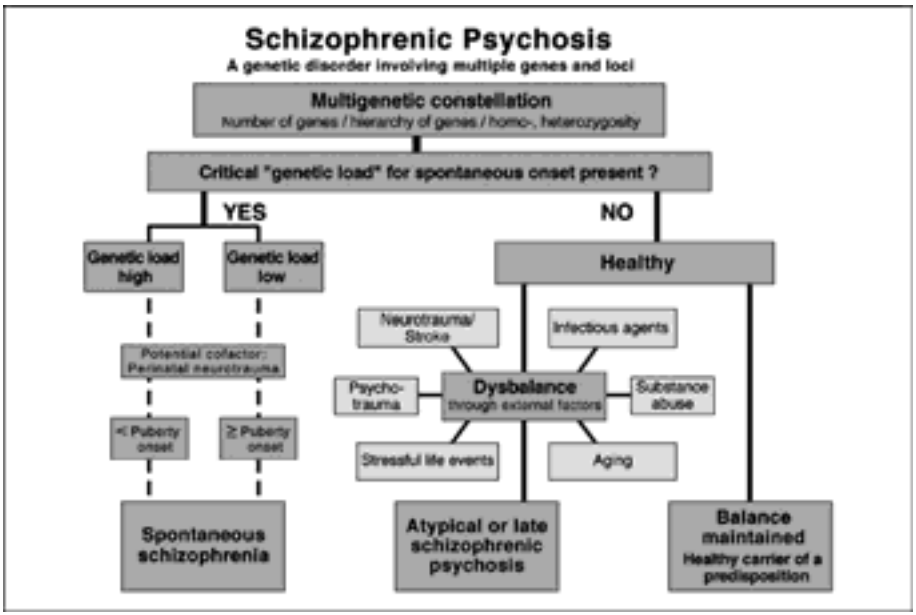
15.55 *Jack Price, London*
Oct-6, Neural Damage, and Schizophrenia

16.20 **Coffee break**

16.45 *Jay Giedd, Bethesda*
Cortical gray-matter deficits in schizophrenia

17.10 *Peter Falkai, Homburg*
Inflammatory mechanisms of schizophrenia

17.35 *Thomas Pollmächer, Munich*
Involvement of cytokines in the pathophysiology of schizophrenia



Introductory Remarks to Symposium 23

German-Israeli cooperation in neuroscience

Bert Sakmann, Heinz Beck and Marlies Dorlöchter

There is a long tradition of cooperation between German and Israeli scientists. The German Max-Planck-Society and the Israeli Weizmann Institute were the first to initiate official contacts between scientists from Israel and Germany in 1959, long before diplomatic relations between both countries were possible. Following these early contacts, the German Federal Ministry of Education and Research (BMBF) and the Israeli Ministry of Science, Culture and Sport (MOS) established a cooperation in various fields of medical research. Molecular and cellular mechanisms of brain function and neurological diseases are the main research areas of the current projects.

Using a combination of electrophysiological, molecular biological and optical imaging methods in addition to behavioral studies, A. Grinvald and B. Sakmann examine the functional architecture of the mammalian cortex. They address the development of cortical maps, their function and dysfunction with regard to glutamate receptor channels and other neuronal messenger systems.

H. Bergmann and A. Engel investigate the pathophysiological relevance of synchrony and temporal patterning in Parkinson's disease in animal models. Abnormalities in these characteristics of neuronal assemblies are critical for both the tremor and the negative motor signs of the disease such as akinesia and rigidity. Recording of basal ganglia and cortical activity in monkey and rat allows to study effects of pharmacological manipulations of the symptoms.

Y. Yaari and H. Beck examine activity-dependent molecular and functional changes in different classes of voltage-dependent ion channels, and how these changes affect the intrinsic firing behavior of neurons. Altered expression of voltage-dependent Na^+ and Ca^{2+} channels following epileptic activity was shown to cause a dramatic and long-lasting switch from regular to burst firing mode. This change may be critical for initiation of seizures and suggest new drug targets in the treatment of epilepsy.

Irreversible injury to neuronal structures in patients with multiple sclerosis (MS) is the topic of F. Zipp's and S. Brocke's work. They present evidence that tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) plays a detrimental role for neuronal injury in autoimmune encephalomyelitis, an animal model of MS. Functional blockade of the death pathway may open up a new therapeutic avenue for MS patients.

Nerve growth factor and the related neurotrophins are secreted by target cells in the projection fields of responsive neurons, which are dependent on this trophic support for survival and maintenance of phenotype. The cellular mechanism by which the neurotrophin signal is propagated from the axon terminal to the cell body is probably retrograde axonal transport of activated neurotrophin-receptor complexes. Work by M. Fainzilber and T. Jovin supports this „signaling endosome" hypothesis.

GABA, the main neurotransmitter in the suprachiasmatic nucleus (SCN) has a dual effect on SCN neurons, excitatory during the day, and inhibitory at night. This has been attributed to circadian changes in intracellular chloride concentration ($[Cl^-]i$). Indeed, Y. Yarom and F. Nürnberger demonstrate that GABA induced current in SCN neurons can significantly alter $[Cl^-]i$. Slow recovery from Cl^- depletion along with a reduced activity of the GABA transporter, GAT-1, can explain a lower $[Cl^-]i$ during the night phase of the circadian cycle.

SYMPOSIUM 23

Saturday, June 14th 2003, 15.00–18.00, Lecture Hall 11

Chair: Bert Sakmann (Heidelberg)

German-Israeli cooperation in neuroscience

15.00 *Bert Sakmann, Heidelberg*
Introduction

15.10 *Amiram Grinvald and Bert Sakmann, Rehovot and Heidelberg*
Structure and function of cortical maps in genetically manipulated mice

15.35 *Hagai Bergman and Andreas Engel, Jerusalem and Hamburg*
Role of neural dynamics in Parkinson's disease – comparative physiological studies in the primate and rodent model

16.00 *Yoel Yaari and Heinz Beck, Jerusalem and Bonn*
Plasticity of intrinsic membrane properties in epilepsy

16.25 **Coffee Break**

16.45 *Frauke Zipp and Stefan Brocke, Berlin and Jerusalem*
Regulation of neuronal apoptotic cell death in autoimmune inflammatory disorders of the central nervous system

17.10 *Michael Fainzilber and Thomas Jovin, Rehovot and Göttingen*
Retrograde transport of trophic signaling complexes in healthy and injured neurons

17.35 *Yosef Yarom and Frank Nürnberger, Jerusalem and Frankfurt*
GABA, chloride and circadian rhythm

Introductory Remarks to Symposium 24

Attentional modulation of sensory information processing in man and monkey

Stefan Treue

The visual system of man and other highly evolved animals supplies a wealth of detailed information about the visual environment. Yet at any given moment much of this information is behaviorally irrelevant. If evolution would not have also endowed the nervous system with mechanisms to control the flow of information, only a small fraction of our processing capabilities could be devoted to critical aspects of the incoming sensory signals. In addition to bottom-up mechanisms the visual system uses attention as a powerful top-down influence to optimize the use of its processing resources by allowing us to concentrate processing on a very small proportion of the incoming information.

In recent years a combination of psychophysical and functional cortical imaging studies in humans, with computational modeling and electrophysiological recordings in non-human primates have brought an explosive growth in our understanding of the mechanisms and perceptual effects of attention.

Such studies have shown that the allocation of attention enhances the processing of attended locations or stimulus features and suppresses those from unattended locations or features. The effect of such attentional selection is dramatic, leading to severe perceptual deficits for unattended aspects of visual scenes and playing a crucial role in the control of goal-directed movements (H. Deubel). Psychophysical experiments in humans have helped to quantify the perceptual effects of attention, providing the constraints needed for realistic models of attentional mechanisms (J. Braun). Cortical imaging techniques have elucidated the networks of cortical areas that underlie the deployment of attention (S. Kastner). Single cell recordings in awake behaving monkeys have provided important information as to where and how the interaction between bottom-up sensory signals and top-down influences takes place (P. Fries, S. Treue). Attentional influences have now been demonstrated throughout visual cortex but with an increase as one ascends the hierarchy of visual areas in primate cortex, ultimately resulting in a neural representation of the visual world that is dominated by the behavioral relevance of the information rather than being primarily designed to provide an accurate and complete description of it.

The symposium will give an overview of the state-of-the-art in attentional research covering a range of approaches, but all aimed at a central area of research in cognitive neuroscience.

SYMPOSIUM 24

Saturday, June 14th 2003, 15.00–18.00, Lecture Hall 105

Chair: Stefan Treue (Göttingen)

Attentional modulation of sensory information processing in man and monkey

15.00 *Jochen Braun, Devon (UK)*
Attention as a bottom-up process

15.30 *Stefan Treue, Göttingen*
The physiology of attention in the „where" pathway: location, features and objects

16.00 *Pascal Fries, Nijmegen (The Netherlands)*
The physiology of attention in the „what" pathway: oscillatory neuronal synchronization and firing rates

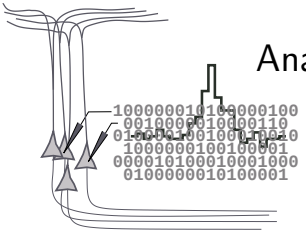
16.30 **Coffee Break**

17.00 *Sabine Kastner, Princeton (USA)*
Mechanisms of visual attention in the human brain

17.30 *Heiner Deubel, Munich*
Attention and awareness in goal-directed eye and hand movements

Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seems several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others.

William James, The Principles of Psychology, 1890



Course on

Analysis and Models in Neurophysiology

6. - 9. October 2003
at the Albrecht-Ludwigs-University, Freiburg
presented by the
German Neuroscience Society
organized by
S. Grün, A. Aertsen, U. Egert, S. Rotter

The course is intended to provide graduate students and young researchers from neuroscience with approaches for the analysis of electrophysiological data and the theoretical concepts behind them. The course includes topics such as

- Neuron models and point processes
- Spike train statistics and correlation measures
- Systems and signals
- Local field potentials and synaptic plasticity

Lectures and exercises (using Matlab and Mathematica) will be given by the organizers. Experience with the software packages would be very helpful but is not required.

The course takes place at the Inst. for Biology, Albrecht-Ludwigs-University, CIP-Pool, Hauptstr. 1, 79104 Freiburg.

Participation fee: 50 EUR (NWG-members), 125 EUR (non-members).

Number of participants limited to 12. Deadline for registration: 30. June 2003.

For further information please contact:

Dr. Sonja Grün, tel: 030-838-56635 (new!), nwg-course@biologie.uni-freiburg.de

PhD or Postdoc Position
in Computational Neuroscience/Neuroinformatics

In a combined experimental and theoretical project in cooperation with the groups of Dr. M. Munk, Max-Planck-Institute for Brain Research in Frankfurt/M and Prof. A. Engel, University Hamburg, funded by the Volkswagen foundation, we study neuronal interactions and test their functional significance for sensorimotor integration and short time memory.

The theoretical project focuses on the development of new tools for the observation of assembly dynamics in massively parallel recordings, involves the investigation of algorithms for data reduction and analysis, and the visualization of high-dimensional data sets. Access to advanced software tools and computer systems is provided.

Applicants should have a background in physics, mathematics, statistics or related fields. Skills in signal- and time series analysis as well as in numerical methods would be helpful.

Contact and further information:

Dr. Sonja Grün, Inst. Biologie (Neurobiologie), Freie Universität Berlin, Königin-Luise Str. 28/30, 14195 Berlin, tel: 030-838-56635, gruen@neurobiologie.fu-berlin.de

Symposia

Poster Contributions

Number of contribution	
0235–0253	Mechanoreception and somatosensory systems
0254–0279	Muscle, motor and sensorimotor systems
0280–0287	Rhythmogenesis and motor pattern generation
0288–0311	Audition, vibration and communication in invertebrates
0312–0317	Audition and vocalization in lower vertebrates
0318–0329	Audition and vocalization in birds and mammals: Periphery
0330–0371	Audition and vocalization in birds and mammals: CNS and perception
0372–0387	Lateral line systems; Vestibular systems
0388–0442	Chemosensory and thermosensory systems
0443–0468	Visual systems of invertebrates: Periphery
0469–0500	Visual systems of invertebrates: Central areas and perception
0501–0526	Visual systems of vertebrates: Periphery
0527–0560	Visual systems of vertebrates: Central areas and perception
0561–0581	Visual systems of vertebrates: Development and regeneration
0582–0606	Cortex and Cerebellum
0607–0634	Hippocampus and Limbic system
0635–0676	Learning and Memory
0677–0692	Neuroanatomical studies
0693–0702	Neurohistochemical studies
0703–0710	Neurochemistry
0711–0760	Synapses and transmitters
0761–0785	Neuropeptides and neuromodulation
0786–0843	Ion channels and receptors
0844–0867	Neuropharmacology and -toxicology
0868–0881	Cell and tissue cultures
0882–0900	Glia cells; Myelin
0901–0936	Neuronal development
0937–0976	Regeneration and plasticity
0977–0983	Neurogenetics
0984–1001	Neuropathology
1002–1006	Neural-immune interactions
1007–1012	Neuroendocrinology
1013–1030	Neuropsychology and psychophysics
1031–1058	Neuronal networks theory and modeling
1059–1087	Methods and demonstrations

Poster Contributions Part I

Symposium: Adaptation: the psychophysicist's microelectrode

No. 1–6: Lectures at the symposium

7 A. Werner, Tübingen
Stereo disparity and chromatic adaptation

Symposium: Juvenile hormone as a mediator of behavioural plasticity in adult insects

No. 8–13: Lectures at the symposium

14 Y. Gaubard, C. Gadenne, G. D. Prestwich, C. Löfstedt and J.-F. Picimbon, Lund (Sweden), Villenave Ornon (France) and Salt Lake City, UT (USA)
Juvenile hormone binding proteins and neuronal plasticity

15 R. Spieß and U. Rose, Ulm
Effects of juvenile hormone on the abdominal motor system of adult Locusta migratoria

16 S. Anton and R. Ignell, Alnarp (Sweden)
Olfactory-guided aggregation behaviour and olfactory processing in desert locusts are regulated by juvenile hormone

Symposium: Cytokines as mediators of neuroglial interactions

No. 17–22: Lectures at the symposium

23 N. Jeliaznik and J. Mey, Aachen
Activation of retinoic acid signaling after sciatic nerve injury: Upregulation of cellular retinoid binding proteins

24 K. Schrage, V. Johann and J. Mey, Aachen
Cytokine expression in schwann cell primary cultures after retinoic acid treatment

25 H. Siebert and W. Brück, Göttingen
The influence of different cytokines and proteases on sciatic nerve degeneration – a study in different knockout mice

26 S. J. Haas, A. Ahrens, O. Schmitt and A. Wree, Rostock
Quinolinic acid lesions of the caudate putamen in the rat lead to an increase of Ciliary Neurotrophic Factor

Symposium: Transgenic animal models for neurodegenerative diseases

No. 27–31: Lectures at the symposium

32 U. Ueberham, E. Ueberham, R. Gebhardt and T. Arendt, Leipzig
Inducible neuronal expression of TGF-β1 in transgenic mice

33 E. Ramminger, U. Ueberham, A. G. Beck-Sickinger, R. Heumann and T. Arendt, Leipzig and Bochum
Altered expression of plasticity-related genes in syn-ras transgenic mice

34 S. Cambridge, B. Cürten and T. Bonhoeffer
A caged doxycycline analog for photoactivated gene expression with high spatiotemporal resolution

Symposium: Signal integration in dendrites

No. 35–40: Lectures at the symposium

41 N. Benhassine and T. Berger, Bern (Switzerland)
Biophysical properties and distribution of large-conductance calcium-dependent potassium channels in neocortical layer 5 pyramidal neurons.

42 W. Senn, H.-R. Lüscher and M. E. Larkum, Bern (Switzerland) and Heidelberg
The gain of L5 pyramidal neurons is larger for distal than for somatic input

43 B. M. Kampa and G. J. Stuart, Freiburg
Dendritic mechanisms involved in spike-timing dependent plasticity

44 E. H. van den Burg, J. Bacelo, L. Gómez, J. Engelmann and K. Grant, Gif sur Yvette (France)
Inhibition of back-propagating spikes in a cerebellum-like sensory structure in the weakly electric fish Gnathonemus petersii*, by a general anaesthetic*

Symposium: Neuronal death and neuroprotection: The role of glial cells

No. 45–50: Lectures at the symposium

51 A. Wallraff, K. Hüttmann and C. Steinhäuser, Bonn
Complete lack of gap junctional coupling in a subpopulation of astrocytes, termed GluR cells, in the hippocampus.

52 C. Krebs, H. Fernandes, C. Sheldon, A. Huxtable, A. El-Husseini, L. Raymond and K. Baimbridge, Bonn
Functional NMDA receptors in post-ischemia astrocytes – a possible synaptic target?

53 G. Seifert, K. Hüttmann, K. Matthias, C. Knott, G. Wilkin, C. Neusch, H. Lester and C. Steinhäuser, Bonn, London (UK), Göttingen and Pasadena, CA (USA)
Kir channels in the hippocampus: Different expression in distinct types of astrocytes and alterations under pathophysiological conditions

54 S. Walter, S. Kühl, Y. Liu, F. Mühlhäuser, K. Beyreuther and K. Faßbender, Göttingen
Alzheimer's disease. Beta-amyloid induces neuroinflammation via lipopolysaccharide receptor (CD14)

55 A. El Emmam Dief, C. Redecker, G. Metz, A. Aschoff, O. Witte, K. El Sabah and G. Jirikowski, Jena and Alexandria (Egypt)
Histochemical monitoring of apoptosis after cerebral ischemia and reperfusion in rat brain

56 M. Francke, I. Goczalik, D. Schwarze, M. Raap and A. Reichenbach, Leipzig
Neuronal glutathione supply by Müller cells during oxidative stress

57 T. Pannicke, B. Biedermann, O. Uckermann, M. Weick, A. Bringmann, S. Wolf, P. Wiedemann, E. Buse and A. Reichenbach, Leipzig and Münster
Physiological properties of retinal Müller glial cells from the monkey Macaca fascicularis – *comparison to human Müller cells*

Symposium: Drug addiction: Mechanisms and therapy

No. 58–63: Lectures at the symposium

Symposium: Precise timing in the brain: Linking neuronal activity and behaviour

No. 64–69: Lectures at the symposium

70 H. R. Dinse and I. van der Berg, Bochum
What is simultaneous? Tactile coactivation in human subjects reveals requirement for millisecond precision for induction of plastic changes

71 K. H. Kreikemeier, I. van den Berg and H. R. Dinse, Bochum
Effects of timing: Switching cortical map reorganization and perceptual learning

72 C. Oreja-Guevara, R. Gobbelé, F. Darvas, A. Dieckhoefer, H. Buchner and K. P. Hoffmann, Bochum, Aachen and Recklinghausen
Electrical source activity and interregional coherences of the human brain during visuomotor tasks

73 P. Ragert, B. Pleger, M. Tegenthoff, A.-F. Foerster, V. Nicolas and H. R. Dinse, Bochum
rTMS elicits tactile discrimination improvement and parallel plastic reorganization in human SI

74 B. Pleger, P. Ragert, A.-F. Förster, H. Dinse, V. Nicolas and M. Tegenthoff, Bochum
Functional magnetic resonance imaging of the human brain: Cortical reorganization controls somatosensory short-term learning.

75 B. Hedwig, Cambridge (UK)
Coding of pattern recognition

76 D. Suchanek, F. Kuemmell, A. Aertsen and D. Heck, Freiburg
Investigating cortical network dynamics with combined intracellular and multi-electrode extracellular recordings

77 F. Sultan and D. Heck, Tübingen and Freiburg
Detection of sequences in the cerebellar cortex: Numerical estimate of the possible number of sequences represented

78 F. Sultan and S. Rotter, Tübingen and Freiburg
Simulating the cerebellar tidal-wave – variability in axonal conduction velocity constrains noisy inputs

Symposium: Ontogenetic cell death in the nervous system

No. 79–83: Lectures at the symposium

84 N. Dünker and N. Schuster, Göttingen and Homburg
TGF-β modulated programmed cell death in the developing retina

85 E. Aden, Hamburg
Apoptosis determines the ontogenetic regression of the cave fish eye

86 J. Dorszewska and Z. Goncerzewicz, Poznan (Poland)
The oxidative DNA damage and repair (p53) in rat brain aging.

Symposium: Arthropod neural and motor systems: From development to function and mechanics

No. 87–92: Lectures at the symposium

93 S. Schönknecht, C. Duch, M. Scholz, J.-F. Evers and K. Obermayer
Multi compartment model *of developmental changes in dendritic shape during postembryonic motoneuron development*

94 P. Burkert and C. Duch, Berlin
Changes in CaM kinase II activity and localization correlate with distinct phases of motoneuron dendritic growth during Manduca *metamorphosis*

95 J. F. Evers, D. Münch and C. Duch, Berlin
Metric analysis of growth-cones during dendritic remodeling of an identified flight motoneuron in Manduca sexta

96 D. Münch, S. Schmitt, M. Scholz and H.-J. Pflüger, Berlin
Postembryonic growth of a first order interneuron in a developing sensory-motor circuit – A morphometric analysis

97 E. Heidel and H.-J. Pflüger, Berlin
Transient potassium currents in identified subtypes of octopaminergic dorsal unpaired median (DUM-) neurons isolated from locust thoracic ganglia

98 S. Schmitt, J. F. Evers, M. Scholz, K. Obermayer and C. Duch, Berlin
From voxels to model: Automatic reconstruction of neurons from confocal images

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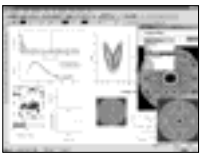

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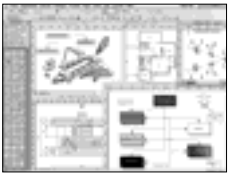

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99 M. C. Göpfert, H. Stocker and D. Robert, Bristol (UK) and Zurich (Switzerland)
Genetically linked formations of sensory and accessory components in the auditory system of Drosophila

100 M. C. Göpfert and D. Robert, Bristol (UK)
Mechanical activity of Drosophila mechanosensory neurons

101 A. Prokop, G. M. Technau, B. Küppers, R. Löhr, K. Lüer, M. Mende and N. Sánchez-Soriano, Mainz
From the NMJ into the CNS – Synapse and circuit formation in fruitflies

102 S. Pick and R. Strauss, Würzburg
Towards the neuronal substrates underlying insect climbing behavior – a high-speed 3D-video analysis of normal and mutant fruit flies

Symposium: Adult neurogenesis

No. 103–107: Lectures at the symposium

108 N. Braun, J. Sévigny, S. K. Mishra, S. C. Robson, S. W. Barth, R. Gerstberger, K. Hammer and H. Zimmermann, Frankfurt am Main, Sainte-Foy, Quebec (Canada), Boston, MA (USA), Karlsruhe and Gießen
The ecto-ATPase NTPDase2 is expressed in the germinal zones of the developing and adult rat brain

Symposium: Invasive recording from the human brain: Linking clinical applications with neurobiological research

No. 109–113: Lectures at the symposium

Symposium: Longterm potentiaton and longterm depression of nociceptive CNS processing

No. 114–119: Lectures at the symposium

120 A. J. Artola, Antwerp (Belgium)
Use-dependent synaptic plasticities in hippocampus and visual cortex

121 U. Ziemann, Frankfurt am Main
LTP-like plasticity in intact human motor cortex. Investigations with transcranial magnetic stimulation.

122 A. Tappe, D. Hirlinger, J. Benrath and R. Kuner, Heidelberg
Selective induction of Homer1a in spinal neurons during pathological pain states via activation of NMDA receptors and Erk1/2

123 E. P. Kostyuk, Kiev (Ukraine)
Changes in neuronal calcium signalling during diabetic pathology

Symposium: Towards a molecular understanding of behavior

No. 124–129: Lectures at the symposium

130 E. A. Kravitz and S. Chen, Boston, MA (USA)
Untitled

131 K. Hoffmann, B. Wenzel, C. Günther, N. Elsner and R. Heinrich, Göttingen
The potency of acetylcholine to activate muscarinic receptors in the brain of grasshoppers

132 B. Wenzel, C. Günther, R. Lakes-Harlan, N. Elsner and R. Heinrich, Göttingen
Grasshopper acoustic communication behavior is inhibited by activation of the NO-/cGMP- signaling pathway in the brain

133 H. Rolf and M. Hoerner, Göttingen and Hong Kong SAR (China)
Fight or flight? Octopamine effects on the cricket escape pathway

134 M. Seifert, M. Gewecke and T. Roeder, Hamburg and Würzburg
The tyramine receptor of Caenorhabditis elegans

135 V. Dyakonova, A. Kruschinski and D. Sakharov, Moscow (Russian Federation)
To mate or to fight? Effects of flight on male-female relationships in cricket gryllus bimaculatus

136 U. Werner, K. Volkmann and H. Scholz, Würzburg
Functional dissection of the octopaminergic neurotransmitter system in ethanol tolerance in Drosophila

Symposium: Peptide co-transmitters in identified neurons

No. 137–142: Lectures at the symposium

143 V. Fenelon, Y. Lefeuvre and P. Meyrand, Talence (France)
Ontogeny of modulatory systems

Symposium: Early environmental programming: Molecular, neuroanatomical, neuroendocrine and behavioural effects

No. 144–148: Lectures at the symposium

149 A. Avital, G. Richter-Levin, M. Matar, J. Zohar, K. Zohar and H. Cohen, Haifa (Israel)
Setting apart the affected: The use of behavioral criteria in animal models of Acute Stress Response and Post Traumatic Stress Disorder

150 W. Ovtscharoff jr and A. K. Braun, Magdeburg
Quantitative analysis and 3D-reconstruction of neuronal and synaptic structures from serial sections

151 M. Gruss and K. Braun, Magdeburg
Consequences of maternal separation during different stages of early development on HPA axis activity in three week old rats.

152 L.-T. Boenke, J. Bock and A. K. Braun, Magdeburg
Early traumatic experience alters metabolic brain activity in thalamic, hypothalamic and prefrontal cortical brain areas of Octodon degus

Symposium: New forms of cerebellar signaling

No. 153–158: Lectures at the symposium

159 H. Heuer and C. A. Mason, New York, NY (USA)
Role of thyroid hormone in Purkinje cell dendritic development

160 J. Chavas and A. Marty, Paris (France)
Mixed excitatory/inhibitory effect of $GABA_A$ synapses in the cerebellum

Symposium: Complex sensory processing in the vertebrate midbrain

No. 161–166: Lectures at the symposium

167 B. Mönig and H. Luksch, Aachen
Primary Culture of Cells from the optic tectum of the Chick: Establishment and characterisation

168 H. Luksch, Aachen
Neuronal computation in the avian optic tectum: A compilation of neuron types, their connections and transmitters

169 H. Luksch and R. Wessel, Aachen and Saint Louis, MO (USA)
Synaptic depression in motion-sensitive SGC-neurons of the chick optic tectum: Physiological data and modelling

170 M. Manns, B. Hellmann and O. Güntürkün, Bochum
Separation of ascending and descending tectal projections within the tectofugal pathway of the pigeon

171 S. Moeller and B. H. Gaese, Aachen
Auditory attention and spatial selection behaviour effect the neuronal activity in the superior colliculus in rats.

Symposium: Function and dysfunction of the amygdala: Fear and epilepsy

No. 172–178: Lectures at the symposium

179 K. Hüttmann, D. Yilmazer-Hanke, G. Seifert, R. Jabs, J. Schramm, H.-C. Pape and C. Steinhäuser, Bonn and Magdeburg
Functional and molecular characterization of neurons in the human lateral amygdala

180 A. Dityatev, J. Tang, S. Wagner, M. Schachner and C. T. Wotjak, Hamburg
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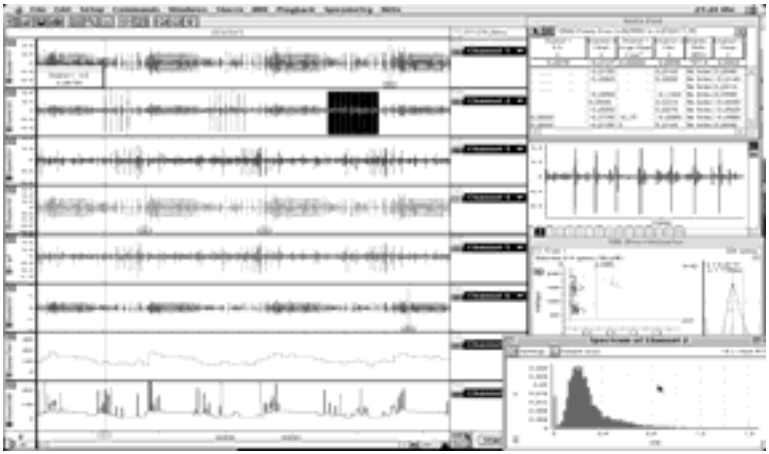

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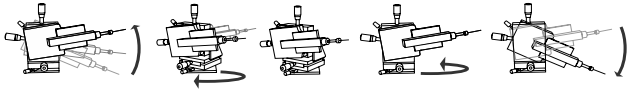

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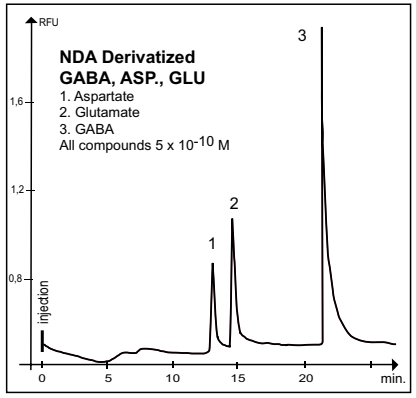

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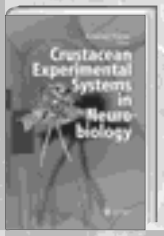

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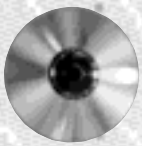

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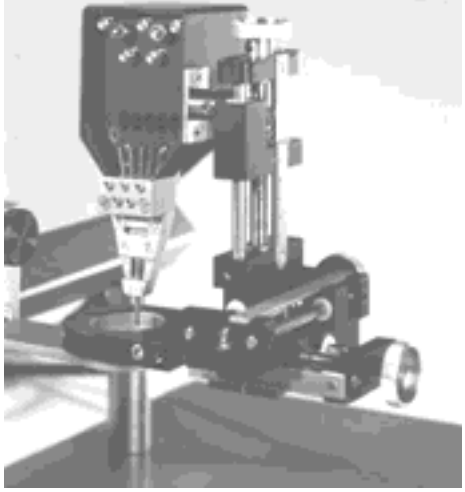

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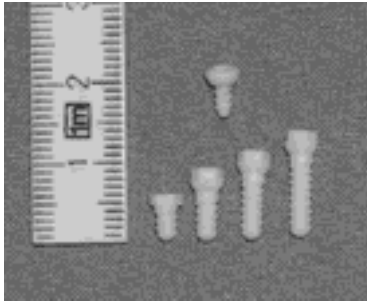

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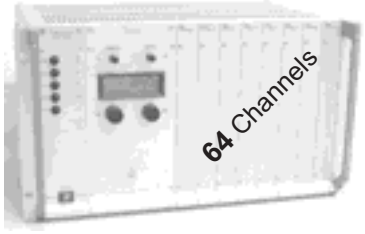

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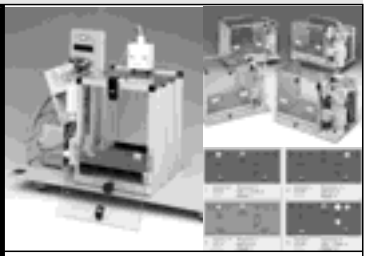

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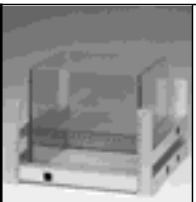

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Poster Contributions Part II

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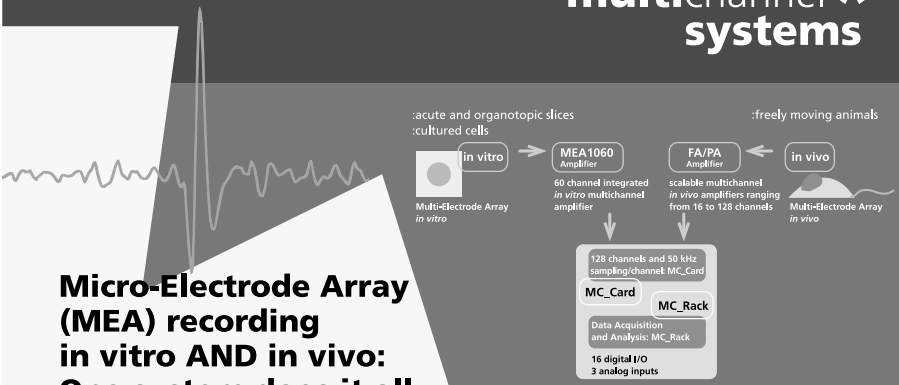

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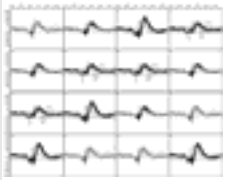

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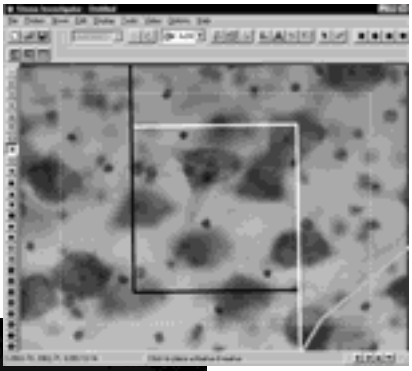

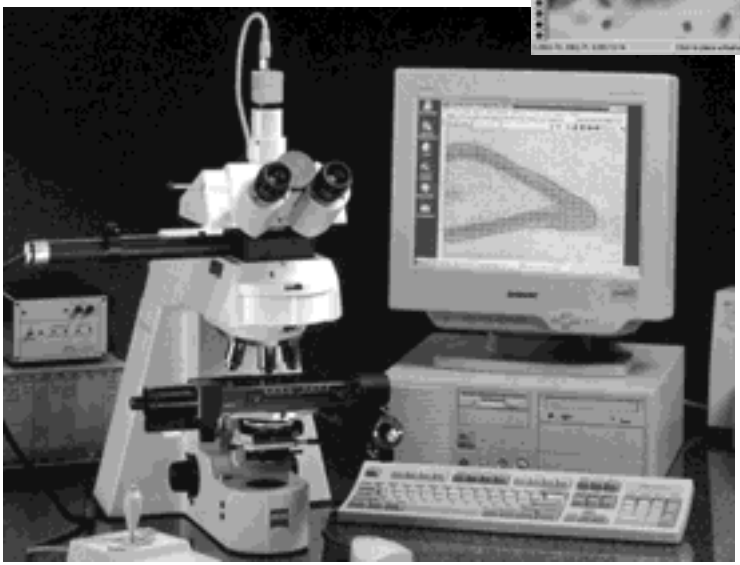

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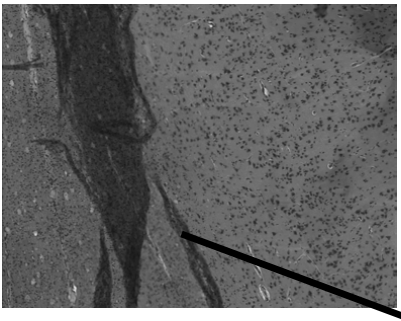

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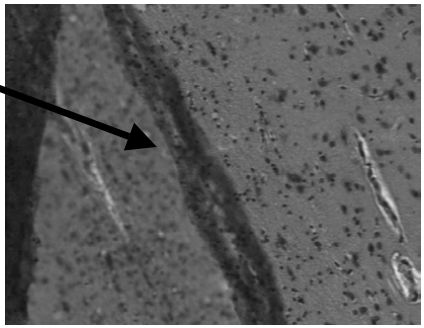

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Allocation of secretory organelle proteins to EGFP-expressing astrocytes in vitro *and* in situ

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gp130-mediated activation of the JAK/STAT-pathway is necessary for activation of glial cells following optic nerve lesion.

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Gap junctions serving intercellular communications are stabilized under the plasma membrane by direct interactions with drebrin

889 C. B. Braun, B. Fuss, G. Raivich and K. Frank, Göttingen, Richmond, VA (USA) and London (UK)
Oligodendrocytes during myelination and trauma in PLP-DSRED transgenic mice

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Dysmyelination caused by cre-mediated inactivation of cholesterol biosynthesis in oligodendrocytes

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Two-photon imaging of glial cells in the intact neocortex

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47 Keddy Bridge Road
R.R. #2
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Canada
phone +1 902 624 0606
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USA
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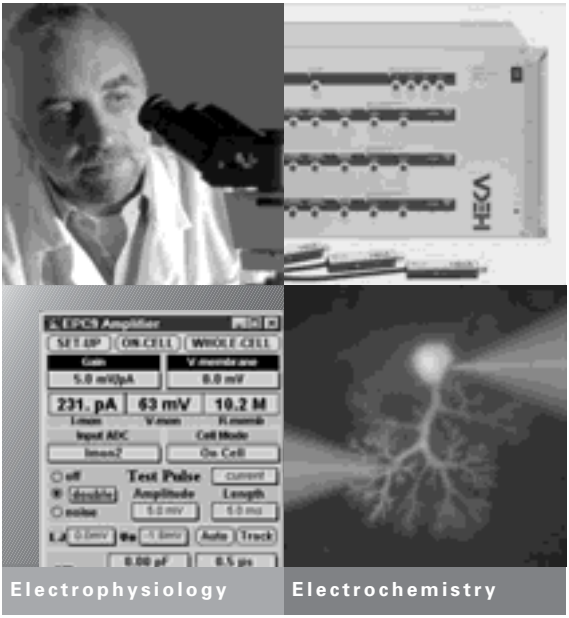

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How likely is recovery after a stroke? Implications from descriptive movement analysis after focal cerebral ischemia in adult rats

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A link between the nervous system and the immune system in insects?

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Affective properties of 1,|25-(OH)$_2$-vitamin D$_3$ and other steroid hormones in rats with or without exposure to endocrine disruptors

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1012 S. A. Krömer, W. Jacob and R. Landgraf, München
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1018 D. Davis and G. von der Emde, Seattle, WA (USA) and Bonn
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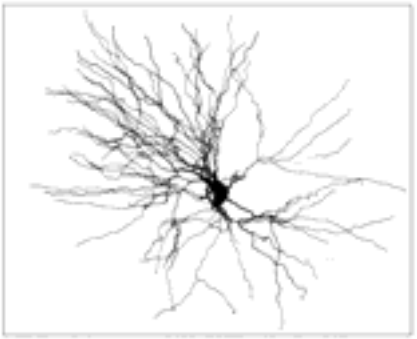

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1028 M. C. Kittel, O. Gleich, I. Hamann and J. Strutz, Regensburg
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1049 S. Dodel, J. M. Herrmann, J.-B. Poline and T. Geisel, Orsay (France) and Göttingen
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1056 A. Knoblauch and G. Palm, Ulm
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1063 H.-G. Schlosser, K. Druen, A. Clarke, W. Lanksch and A. Unterberg, Berlin
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1064 W. Horstmann, S. Lorenz and M. Egelhaaf, Bielefeld
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1065 J. P. Lindemann, N. Böddeker and M. Egelhaaf, Bielefeld
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1066 S. Kutluk, E. Bodur, S. Akar and A. N. Cokugras, Ankara (Turkey) and Darmstadt
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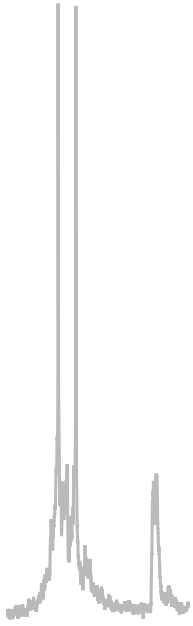

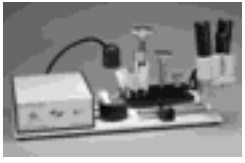

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1082 C. Pouzat, M. Delescluse and J. Diebolt, Paris (France) and Champs-sur-Marne (France)
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1084 H. R. Polder, J. Planck, M. Weskamp, H. Terlau and M. Ferber, Tamm and Göttingen
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1096 M. Egorova, G. Ehret and I. Vartanyan, Saint Petersburg (Russian Federation) and Ulm
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1097 M. Schmäh and H. Wolf, Ulm
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1098 M. Schmäh, H. Wolf and P. Bräunig, Ulm and Aachen
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1099 C. Sommer, R. Kollmar, S. Schwab, M. Kiessling and W.-R. Schäbitz, Ulm and Heidelberg
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1191 M. Sandrini, S. F. Cappa, S. Rossi, P. M. Rossini and C. Miniussi, Brescia (Italy), Milano (Italy), Siena (Italy) and Roma (Italy)
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1194 M. S. Nitsche, M. A. Nitsche, C. C. Klein, F. Tergau, J. Rothwell and W. Paulus, Göttingen and London (UK)
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1196 A. Schlitterlau, U. Henschke, K. Fricke, M. A. Nitsche, D. Liebetanz, F. Tergau and W. Paulus
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1197 M. Nitsche and K. Fricke, Göttingen
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1198 K. Matsunaga, M. A. Nitsche and J. C. Rothwell, London (UK) and Göttingen
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1200 P. Krause, S. Foerderreuther and A. Straube, München
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1201 P. Krause and A. Straube, München
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1202 A. Struppler, B. Angerer and P. Havel, München
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1203 B. Angerer, P. Havel and A. Struppler, München
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1204 A. Peinemann, B. Reimer, C. Löer, B. Conrad and H. R. Siebner, München and Kiel
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1207 N. G. Muggleton, J. O Shea, C.-H. Juan, A. Cowey and V. Walsh, Oxford (UK) and London (UK)
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1208 O. Bártfai, T. Z. Kincses, A. Antal, M. A. Nitsche and W. Paulus, Pécs (Hungary) and Göttingen
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1210 J. Horacek, L. Skrdlantova, B. Paskova, J. P. Prasko, M. Kopecek, C. Hoschl and O. Belohlavek, Prague (Czech Republic)
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1211 L. Skrdlantova, J. Horacek, M. Kopecek, M. Klirova, P. Jezil and J. P. Prasko, Prague (Czech Republic)
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1212 E. Fernandez, A. Alfaro, J. Tormos, R. Climent, H. Vilanova, M. Bongard, J. Peris and A. Pascual-Leone, San Juan de Alicante (Spain)
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1213 E. A. Feredoes, P. S. Sachdev and W. Wen, Sydney (Australia)
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1215 E. A. Feredoes and P. S. Sachdev, Sydney (Australia)
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1216 A. Gerdelat, D. Tombari, I. Loubinoux, F. Chollet and M. Simonetta-Moreau, Toulouse (France)
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1217 B. Tomasino, R. Rumiati, P. Borroni and A. Isaja, Trieste (Italy) and Milano (Italy)
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1218 A. A. Karim, M. Lotze, T. Kammer, T. Hinterberger, B. Godde, L. G. Cohen and N. Birbaumer, Tübingen
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1220 G. F. Wittenberg, S. Smith, E. P. Bastings, T. P. Pons and D. C. Good, Winston Salem, NC (USA)
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1221 A. Wolters, F. Sandbrink, A. Schlottmann, E. Kunesch, K. Stefan, L. G. Cohen, R. Benecke and J. Classen, Rostock, Bethesda, MD (USA) and Würzburg
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1222 M. Wycislo and J. Classen, Würzburg
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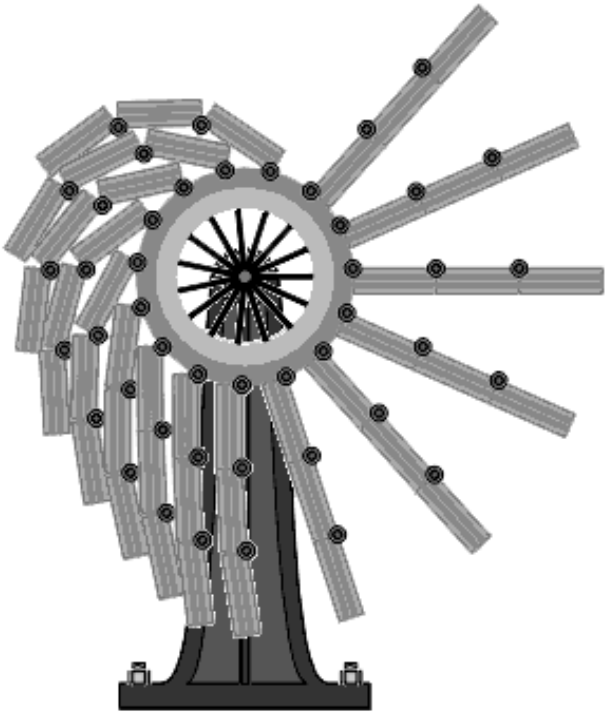
Satellite symposium: Novel Channels and Activation Mechanisms

No. 1223–1227: Lectures at the symposium



Die Wissenschaft sucht das Perpetuum mobile.
Sie hat es gefunden: sie selbst ist es.

Victor Hugo *L'Art et la Science*

Cover illustrations

Front cover: Photomontage of a DiI-labelled neuron from human prefrontal cortex and a human brain. The postmortem specimen was kindly provided by Dr. D. Senitz, University of Würzburg. About 100 optical sections were scanned through the tissue with a confocal laser scanning microscope and volume rendered using a 3D image software.

Back Cover: What appears like a dark ancient forest is actually the immunocytochemical distribution of two different proteins in pyramidal neurons of the rat hippocampus. The microtubule-associated protein MAP2 (bluish) is a marker for the dendritic tree of hippocampal neurons, the proline-rich synapse associated protein ProSAP1 (green) is a recently identified multi-domain protein that may play a crucial role in the assembly of the postsynaptic apparatus. For details see: Böckers et al. 1999, J. Neuroscience 19, 6506–6518.

NOTE OF THANKS

The organisers and the participants of the Göttingen Neurobiology Conference 2003 are deeply indebted to Dipl. Biol. Ralf Schünemann who developed the software for on-line registration of the participants and electronic submission of abstracts as well as the computer aided production of the programme brochure and the conference proceedings. Without his creativity and initiative the conference would not have been possible. Another "Dankeschön" for their substantial organisational help goes to Uli Conrad, Hannelore Elsner, Ralf Heinrich, Reinhard Lakes-Harlan, Stephanie Pauls, Matthias Schink, Andreas Stumpner, Wolfram Zarnack and numerous students of the Department of Neurobiology at the Göttingen Institute for Zoology and Anthropology.

IMPRESSUM

5. Konferenz der Neurowissenschaftliche Gesellschaft
– **29 . Göttinger Neurobiologentagung** –

Leitung
Prof. Dr. Herbert Zimmermann, Biozentrum der Universität Frankfurt am Main, Marie-Curie-Straße 9, D-60439 Frankfurt am Main

Prof. Dr. Norbert Elsner, Abteilung Neurobiologie, Institut für Zoologie und Anthropologie der Universität Göttingen, Berliner Straße 28, D-37073 Göttingen

Die Göttinger Tagung 2003 wird in Verbindung mit der Neurowissenschaftlichen Gesellschaft e. V. unter der Schirmherrschaft des Präsidenten der Georg-August-Universität Göttingen vom Förderkreis Göttinger Neurobiologentagung veranstaltet. Der Förderkreis ist vom Finanzamt Göttingen mit Bescheid vom 9. Juli 2002 im Sinne der §§ 51 ff. AO als gemeinnützigen Zwecken dienend anerkannt worden.