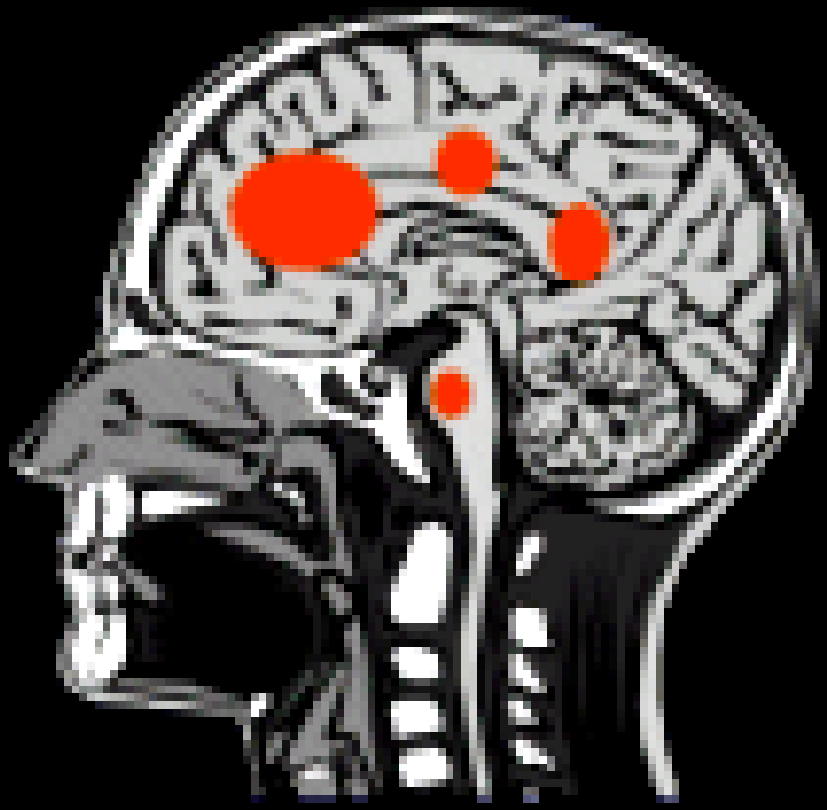
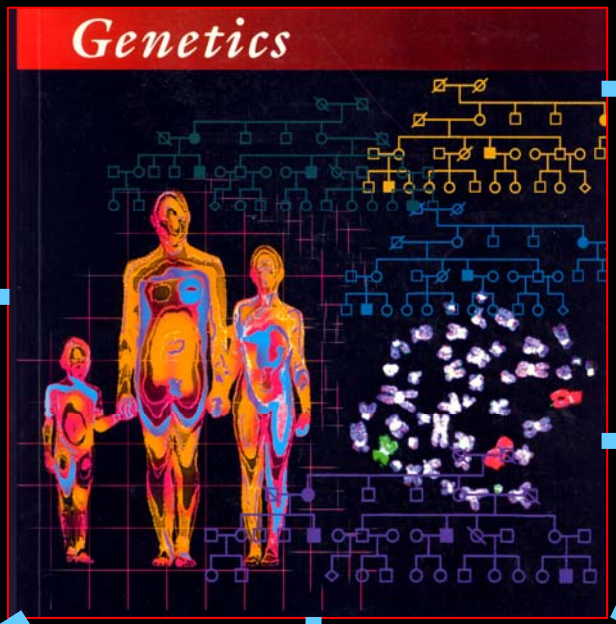
Was gibt es Neues bei der Multiplen Sklerose

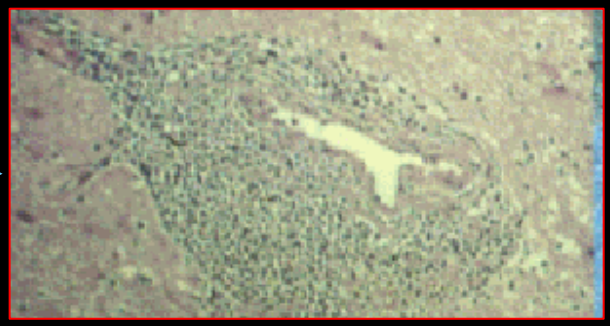
U.K. Zettl
Klinik für Neurologie Universität Rostock

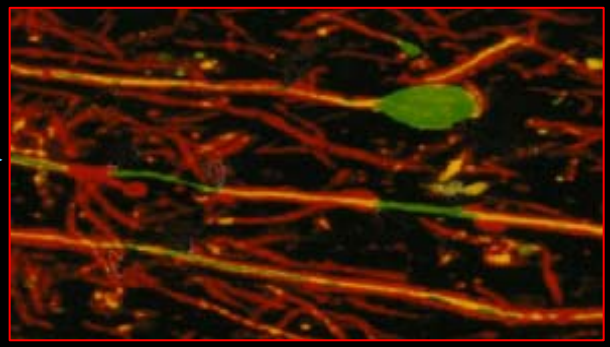
Ursachen der MS

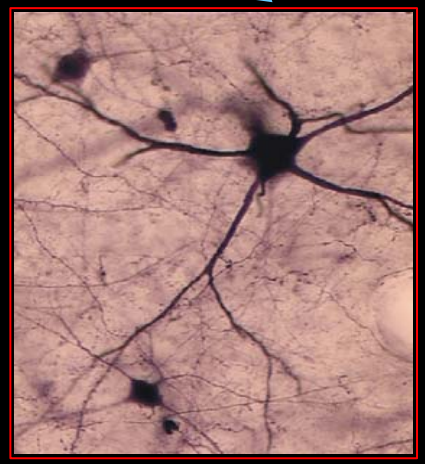


MS und Genetik

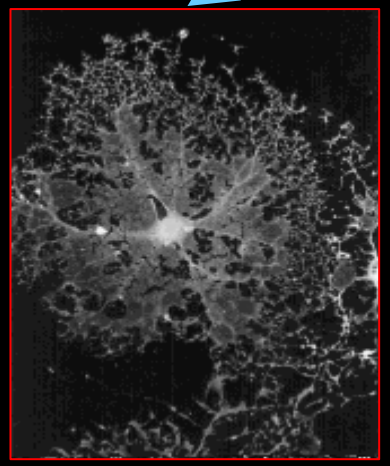




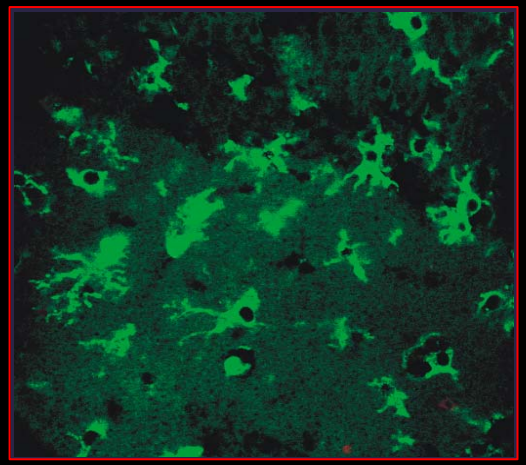




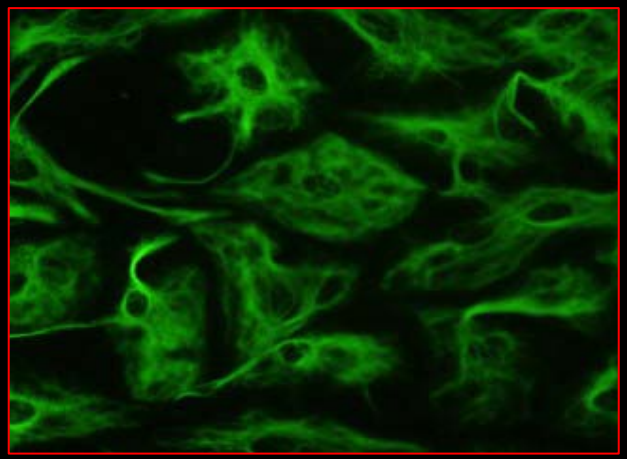
Neuron



Oligodendrozyt



Mikrogliazellen



Astrozyten

Infektion und MS

ARCHIVES OF NEUROLOGY

Vol. 63 No. 6, June 2006

Original Contribution

Epstein-Barr Virus and Multiple Sclerosis

Evidence of Association From a Prospective Study With Long-term Follow-up

Gerald N. DeLorenze, PhD; Kassandra L. Munger, MSc; Evelyn T. Lennette, PhD; Norman Orentreich, MD; Joseph H. Vogelman, DEE; Alberto Ascherio, MD, DrPH

Arch Neurol. 2006;63:839-844.

Tetanus – Impfung und MS

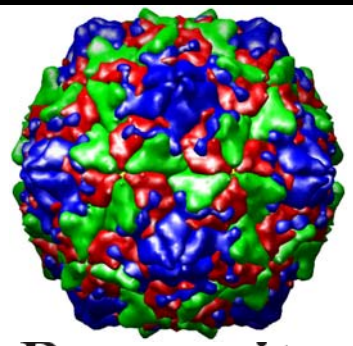
Tetanus vaccination and risk of multiple sclerosis

A systematic review

Miguel A. Hernán, MD; Alvaro Alonso, MD; and Sonia Hernández-Díaz, MD

Infektion und MS

2434 • The Journal of Neuroscience, March 2, 2005 • 25(9):2434–2444



Neurobiology of Disease

Coxsackievirus Targets Proliferating Neuronal Progenitor Cells in the Neonatal CNS

Ralph Feuer, Robb R. Pagarigan, Stephanie Harkins, Fei Liu, Isabelle P. Hunziker, and J. Lindsay Whitton
Department of Neuropharmacology, The Scripps Research Institute, La Jolla, California 92037

Coxsackie-Virus: Myelinschädigung bei Kindern

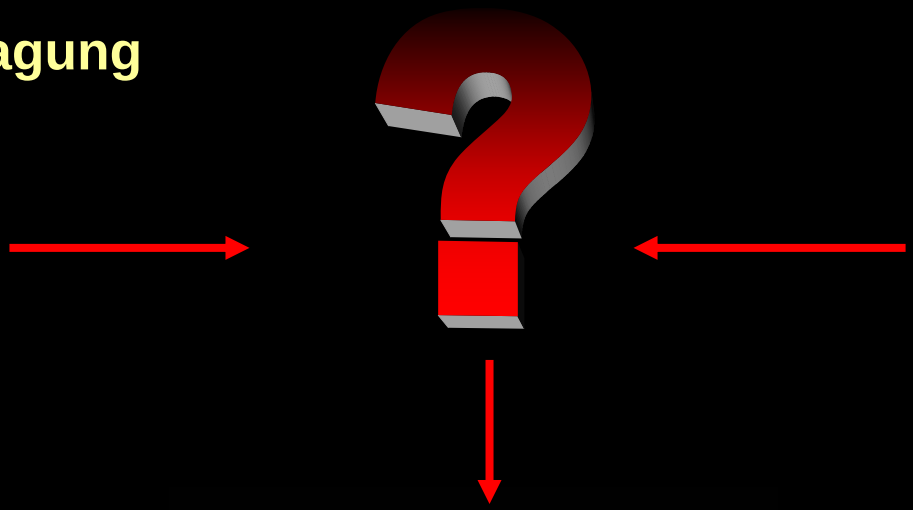
inaktiv in unreifen Zellen

Reduzierung der Vermehrung von NZ

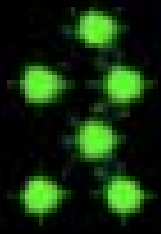
„schlafende Infektion“

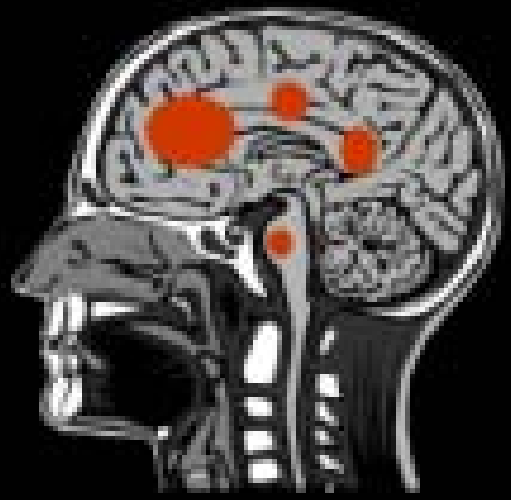
Ursachen der Multiplen Sklerose

Genetische Veranlagung



Umweltfaktoren





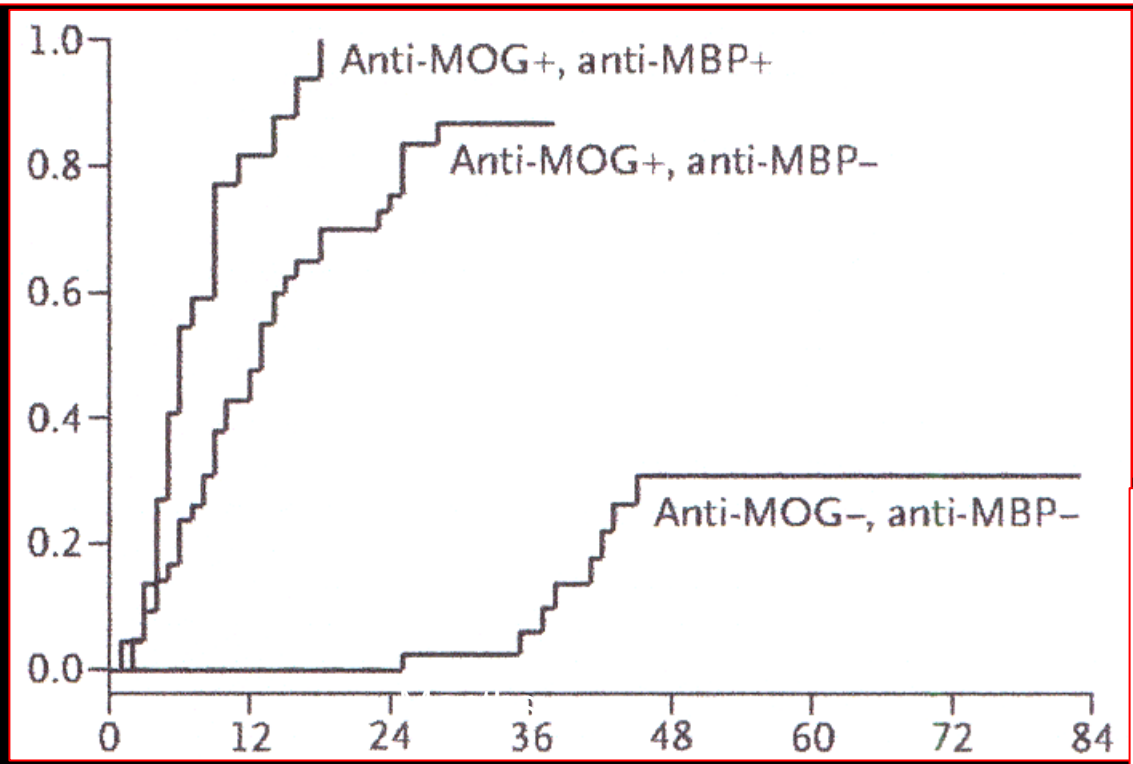
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Antimyelin Antibodies as a Predictor of Clinically Definite Multiple Sclerosis after a First Demyelinating Event

Thomas Berger, M.D., Paul Rubner, M.D., Franz Schautzer, M.D., Robert Egg, M.D., Hanno Ulmer, Ph.D., Irmgard Mayringer, M.D., Erika Dilitz, M.D., Florian Deisenhammer, M.D., and Markus Reindl, Ph.D.

Risk of Clinically Definite Multiple Sclerosis



International Immunology, Vol. 16, No. 3, pp. 559–565
doi: 10.1093/intimm/dxh056

Anti-MOG autoantibodies in Italian multiple sclerosis patients: specificity, sensitivity and clinical association

Renato Mantegazza[1], Piercarlo Cristaldini[1], Pia Bernasconi[1], Fulvio Baggi[1], Rosetta Pedotti[1], Ilaria Piccini[1], Nerina Mascoli[2], Loredana La Mantia[2], Carlo Antozzi[1], Ornella Simoncini[1], Ferdinando Cornelio[1] and **Clara Milanese[2]**

[1]Immunology and Muscular Pathology – Neurology IV, [2]Multiple Sclerosis Center – Neurology IV, Istituto Nazionale Neurologico 'Carlo Besta', Milan, Italy

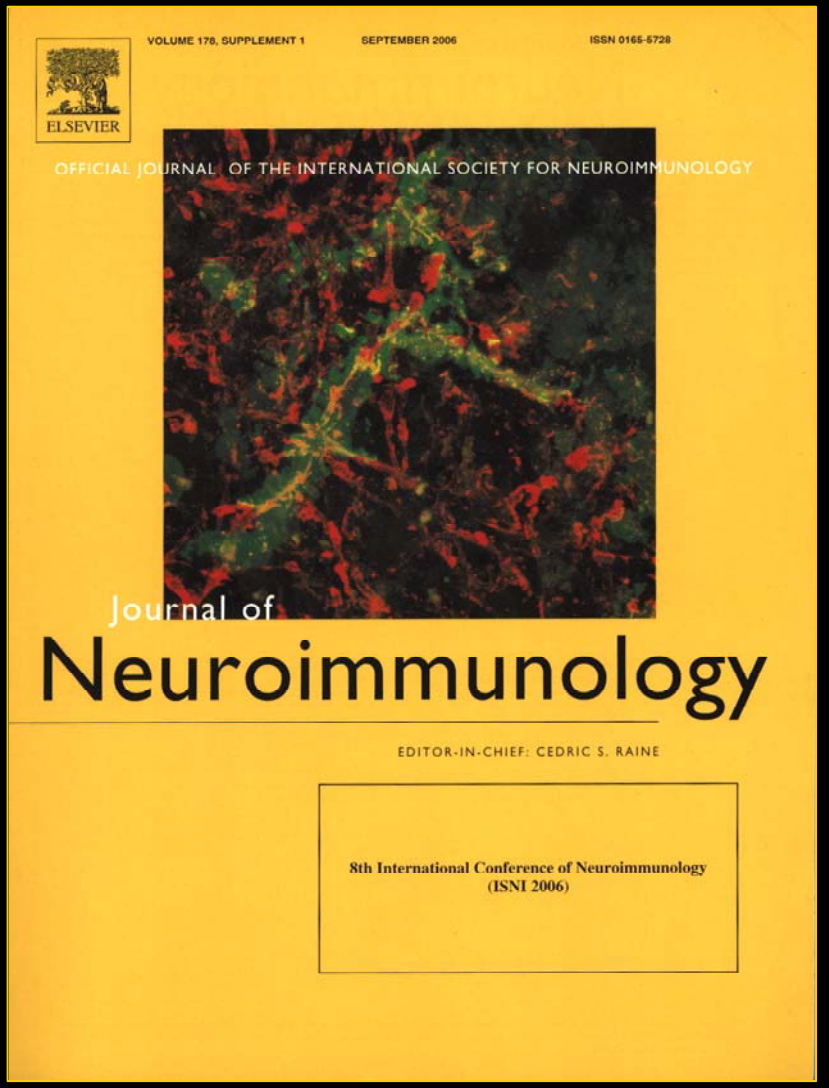
Keywords: antibody, anitbody index, autoimmunity, multiple sclerosis, myelin oligodendrocyte glycoprotein

Similar low frequency of anti-MOG IgG and IgM in MS patients and healthy subjects

V. Lampasona, BSc; D. Franciotta, MD; R. Furlan, MD; S. Zanaboni, BSc; R. Fazio, MD; E. Bonifacio, PhD; G. Comi, MD; and G. Martino, MD

Abstract—The authors used a liquid-phase radiobinding assay to measure serum anti-myelin oligodendrocyte protein (MOG) immunoglobulin (Ig) G in 87 patients with multiple sclerosis (MS), in 12 patients with encephalomyelitis, and in 47 healthy subjects. Anti-MOG IgM was determined in samples obtained at onset from 40 of 87 patients with MS and in control subjects. The frequency of positive samples with low titers of anti-MOG IgG (≤5.7%) and IgM (≤8.3%) was similar in all the groups and subgroups. Binding competition experiments showed that these antibodies had low affinity. Anti-MOG antibodies are not disease specific.

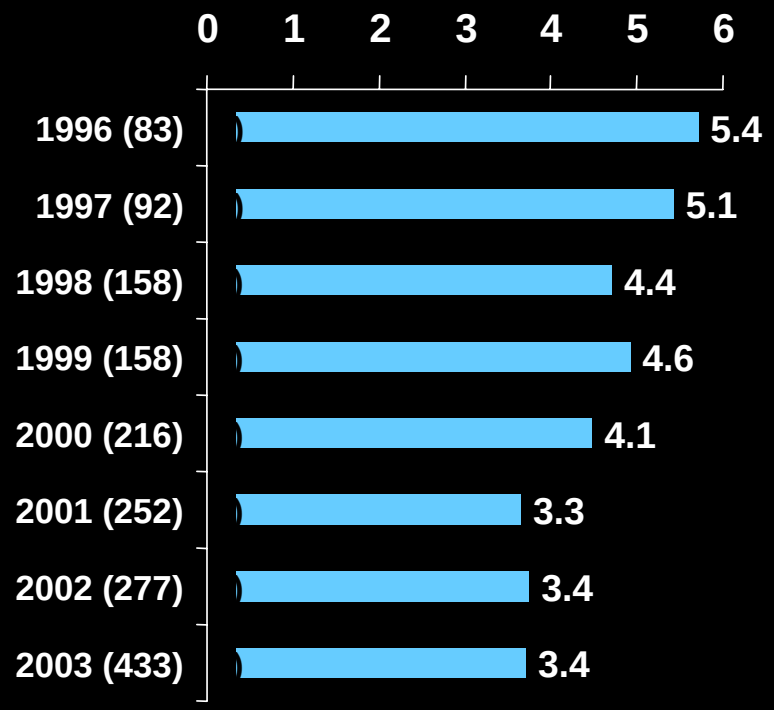
NEUROLOGY 2004;62:2092–2094

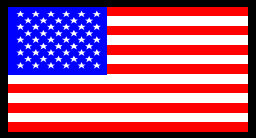


Kuhle J. et al.: Prognostic relevance of antimyelin antibodies for the progression to multiple sclerosis after a first demyelinating event: Results of the BENEFIT trial. *J Neuroimmunol* 2006; 55

Wann beginnen wir mit der Behandlung? Anzahl der Schübe vor dem Behandlungsbeginn

Durchnittliche Anzahl der Schübe:





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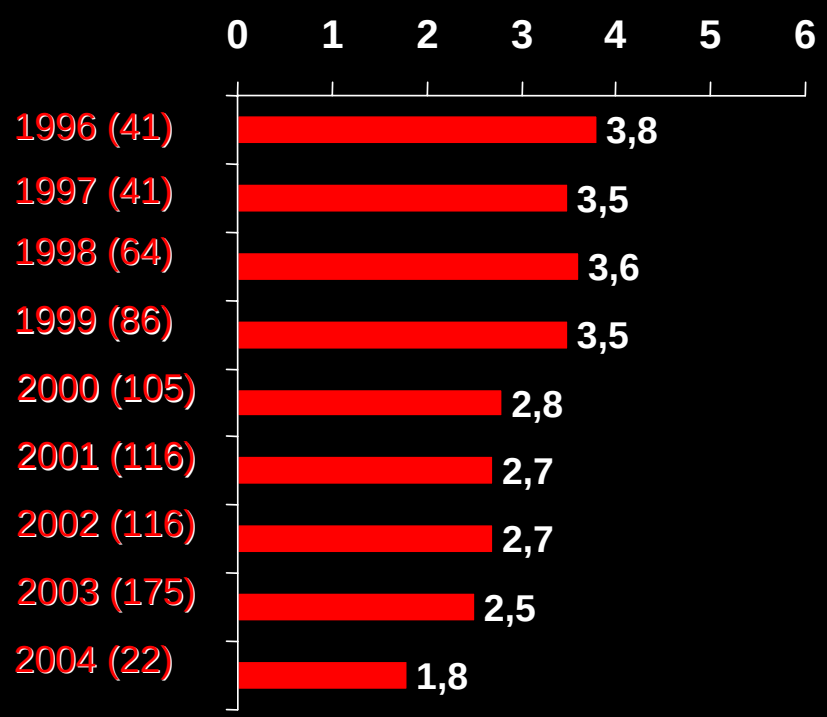
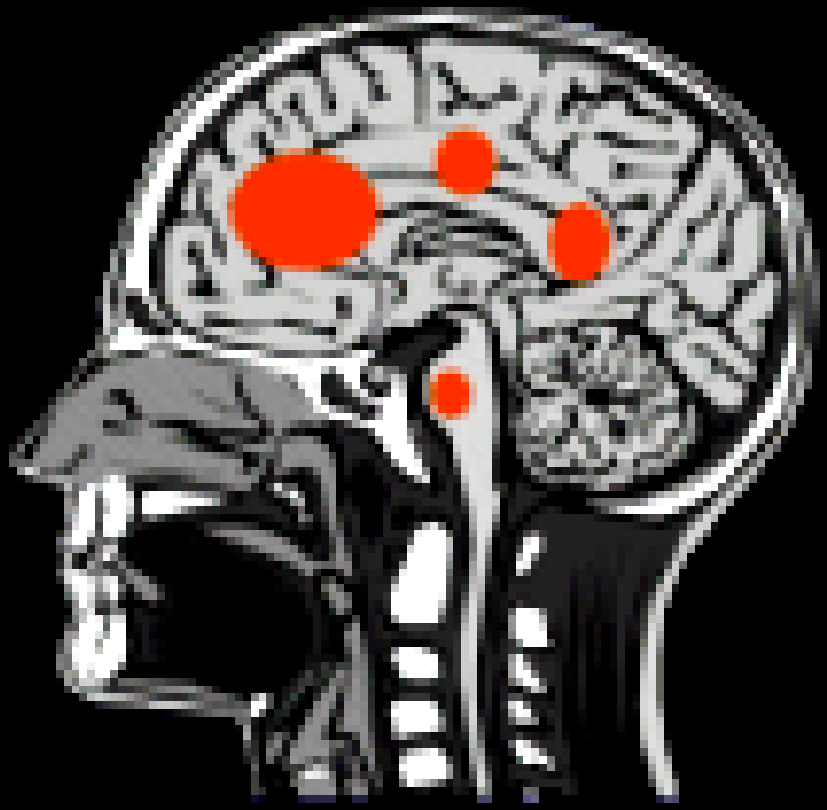


ISIS Research plc – MS Therapy Monitors 2004

Therapien der MS



Nervenarzt 2002 · 73:556–563
DOI 10.1007/s00115-002-1328-x

Aktuelles

Multiple-Sklerose-Therapie-Konsensus-Gruppe*

Immunmodulatorische Stufentherapie der Multiplen Sklerose

Neue Aspekte und praktische Umsetzung, März 2002

*Dies ist ein Beitrag der „Multiple-Sklerose-Therapie-Konsensus-Gruppe".
Die beteiligten Autoren sind in alphabetischer Reihenfolge aufgeführt:
Schweiz: C. Bassetti (Zürich), K. Beer (St. Gallen), S. Beer (Valens), U. Buettner (Aarau), M. Chofflon (Genf), M. Götschi-Fuchs (Knoblisbühl), K. Hess (Zürich), L. Kappos (Basel), J. Kesselring (Valens), H.P. Ludin (St. Gallen), H. Mattle (Bern), M. Schluep (Lausanne), C. Vaney (Montana-Vermala).
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Würzburger Mitarbeiter der MSTKG: A. Bayas, A. Chan, P. Flachenecker, B. Gold, B. Kallmann, V. Leussink.

J Neurol (2004) 251 : 1329–1339
DOI 10.1007/s00415-004-0537-6

ORIGINAL COMMUNICATION

Multiple Sclerosis Therapy Consensus Group

Escalating immunotherapy of multiple sclerosis

New aspects and practical application

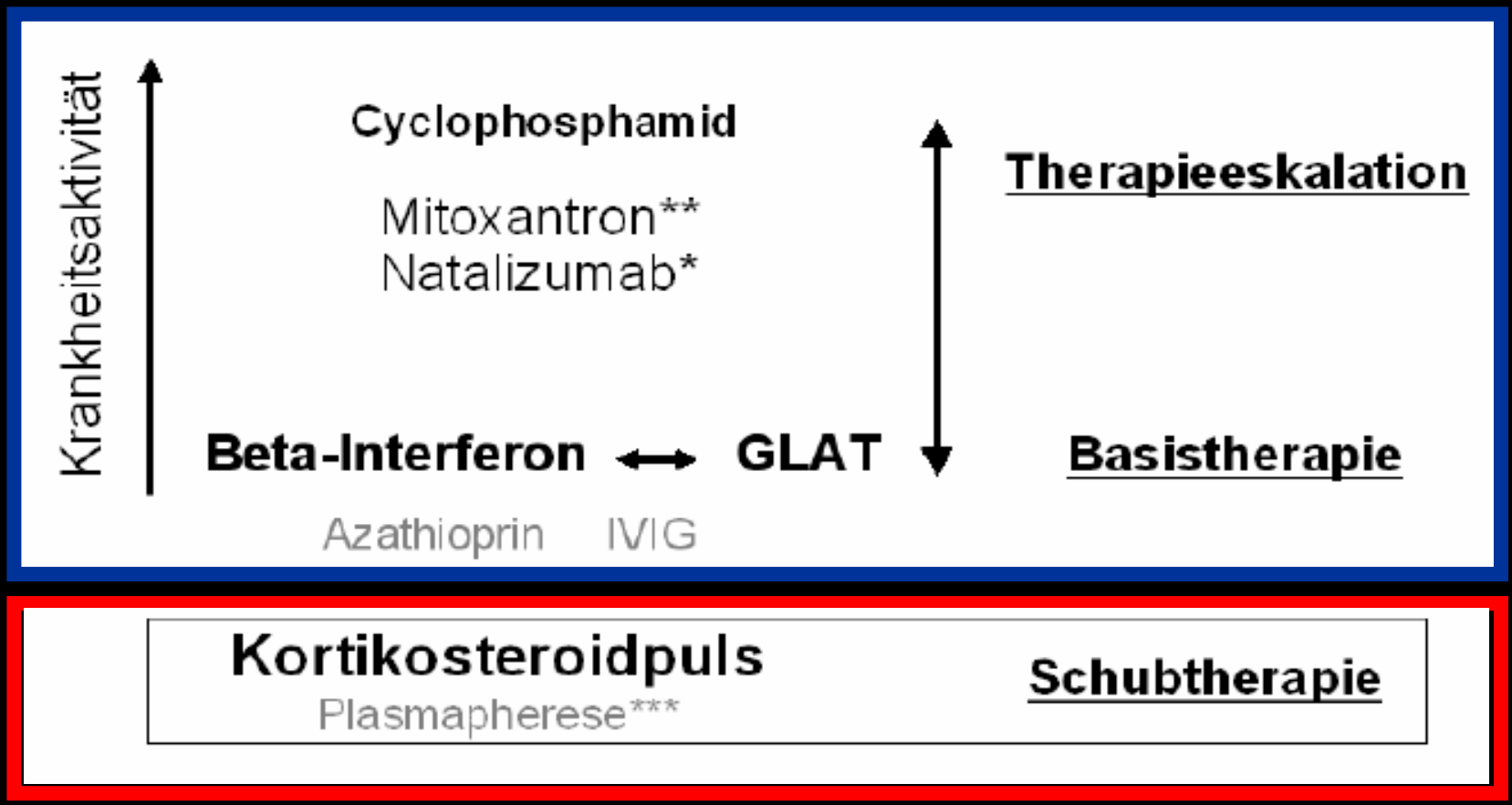
Immunmodulatorische Stufentherapie der Multiplen Sklerose

Aktuelle Therapieempfehlungen (September 2006)
Multiple Sklerose Therapie Konsensus Gruppe (MSTKG)

Escalating immunomodulatory therapy of multiple sclerosis
Update (September 2006)

Immunmodulatorische Stufentherapie der schubförmigen MS

Update 2006



* Bei ≥ 2 schweren Schüben pro Jahr auch als Primärtherapie möglich
** Therapiewechsel auf dieser Eskalationsstufe noch nicht erprobt
*** Option bei schweren, Steroid-resistenten Schüben

Relation between humoral pathological changes in multiple sclerosis and response to therapeutic plasma exchange

Mark Keegan, Fatima König, Robyn McClelland, Wolfgang Brück, Yazmín Morales, Andreas Bitsch, Hillel Panitch, Hans Lassmann, Brian Weinshenker, Moses Rodriguez, Joseph Parisi, Claudia F Lucchinetti

Early, active multiple sclerosis lesions show four immunopathological patterns of demyelination. Although these patterns differ between patients, multiple active lesions from a given patient have an identical pattern, which suggests pathogenic heterogeneity. Therapeutic plasma exchange (TPE) has been successfully used to treat fulminant demyelinating attacks unresponsive to steroids. We postulated that patients with pattern II would be more likely to improve after TPE than those with other patterns since pattern II lesions are distinguished by prominent immunoglobulin deposition and complement activation. We retrospectively studied 19 patients treated with TPE for an attack of fulminant CNS inflammatory demyelinating disease. All patients with pattern II (n=10), but none with pattern I (n=3) or pattern III (n=6), achieved moderate to substantial functional neurological improvement after TPE ($p<0\cdot0001$). Patients with multiple sclerosis with pattern II pathology are more likely to respond favourably to TPE than are patients with patterns I or III.

Lancet 2005; 366: 579–82

See Comment page 526

Acknowledgments

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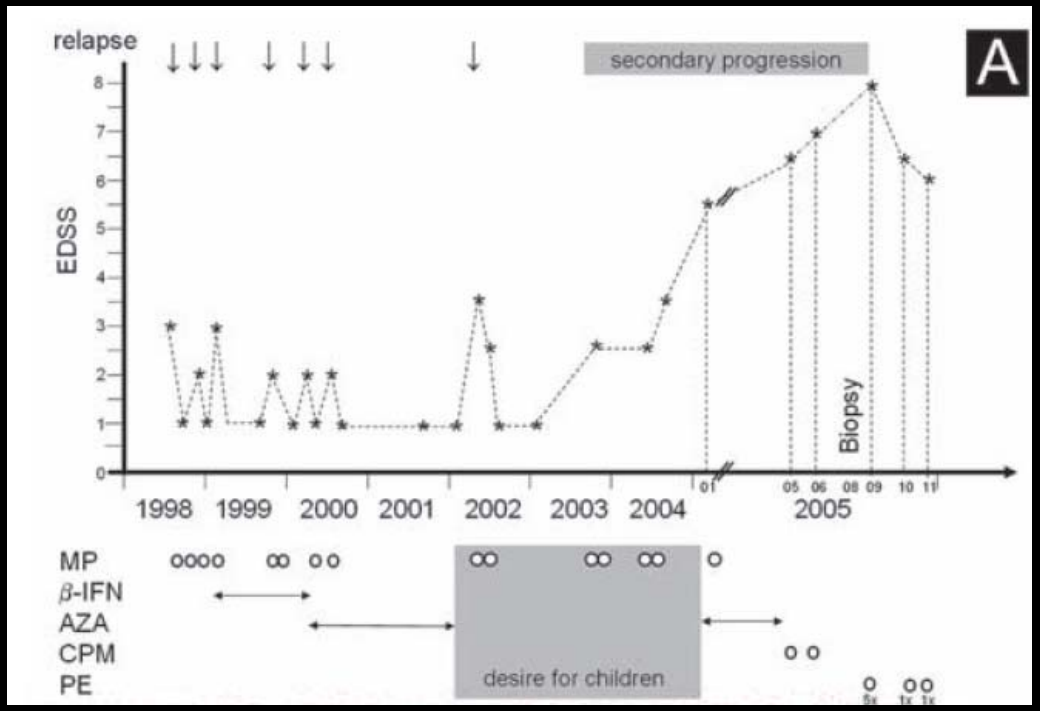
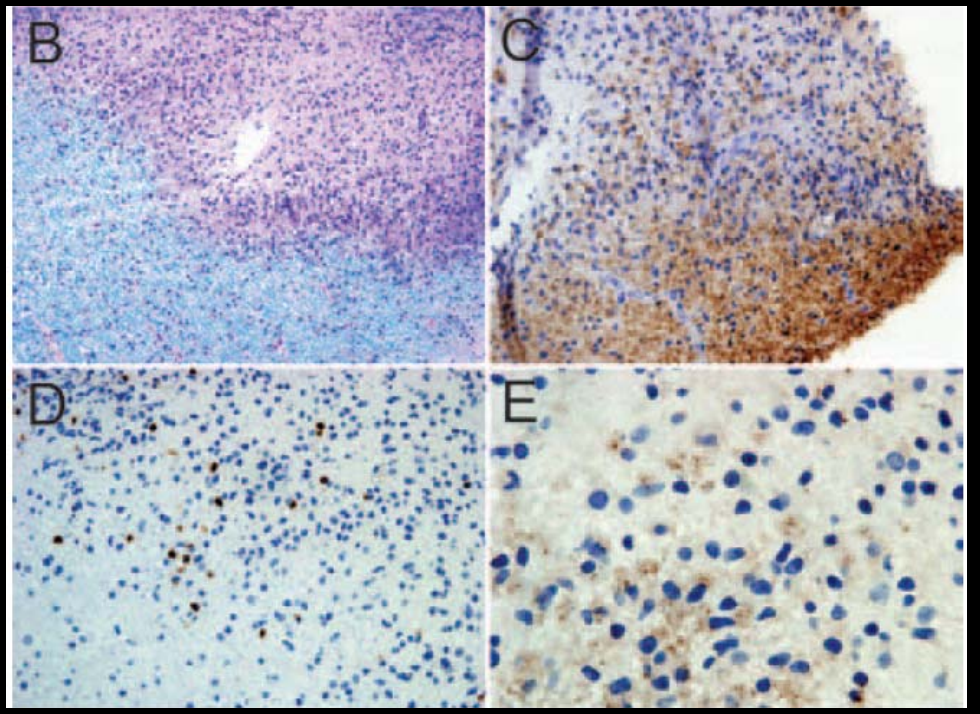
	Treatment failure (n=9)	Treatment success (n=10)	Total (n=19)	p*
Multiple sclerosis pathological pattern [n (%)]				<0·0001
Pattern I	3 (33%)	0 (0%)	3 (16%)	
Pattern II	0 (0%)	10 (100%)	10 (53%)	
Pattern III	6 (67%)	0 (0%)	6 (32%)	
Expanded disability status scale [median (range)]†				
Pre-TPE	7·0 (3·0 to 9·5)	7·3 (4·0 to 9·5)	7·3 (3·0 to 9·5)	0·63
1 month post-TPE	6·8 (3·0 to 9·5)	4·0 (2·0 to 8·0)	4·5 (2·0 to 9·5)	0·08
Change pre-to-post	0·0 (-0·5 to 0·0)	−2·0 (−5·5 to 0·0)	−0·75 (-5·5 to 0·0)	<0·0001
Neurological deficit [n/N]‡				
Brainstem/cranial nerve	0/5	4/4	9 (47%)§	0·66
Cerebellar	0/3	1/4	7 (37%)§	1·00
Cerebral impairment	0/9	5/6	15 (79%)§	0·09
Motor weakness	0/5	7/7	12 (63%)§	0·65
Sensory	0/4	4/6	10 (53%)§	0·66

Lesion pathology predicts response to plasma exchange in secondary progressive MS

U.K. Zettl, MD; H.P. Hartung, MD; A. Pahnke, MD; W. Brueck, MD; R. Benecke, MD; and J. Pahnke, MD, PhD





Neurology (2006) 1515

Natalizumab

VLA-4-Ab, Antegren, Tysabri

The effect of anti-α4 integrin antibody on brain lesion activity in MS

N. Tubridy, MD; P.O. Behan, FRCP; R. Capildeo, FRCP; A. Chaudhuri, FRCP; R. Forbes, MD; C.P. Hawkins, FRCP; R.A.C. Hughes, FRCP; J. Palace, MRCP; B. Sharrack, MD; R. Swingler, MD; C. Young, MRCP; I.F. Moseley, FRCR; D.G. MacManus, MSc; S. Donoghue, PhD; D.H. Miller, FRCP; and The UK Antegren Study Group

Acta Neuropathol (2002) 103: 131–136

REGULAR PAPER

I. Leussink · U. K. Zettl · S. Jander · R. B. Pepinsky
R. Lobb · G. Stoll · K. V. Toyka · R. Gold

Blockade of signaling via the very late antigen (VLA-4) and its counterligand vascular cell adhesion molecule-1 (VCAM-1) causes increased T cell apoptosis in experimental autoimmune neuritis

Received: 23 May 2001 / Revised, accepted: 10 July 2001 / Published online: 31 October 2001
Springer-Verlag 2001

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

A Controlled Trial of Natalizumab for Relapsing Multiple Sclerosis

David H. Miller, M.D., Omar A. Khan, M.D., William A. Sheremata, M.D., Lance D. Blumhardt, M.D., George P.A. Rice, M.D., Michele A. Libonati, M.S., Allison J. Willmer-Hulme, Ph.D., Catherine M. Dalton, M.B., Katherine A. Miszkiel, M.B., and Paul W. O'Connor, M.D., for the International Natalizumab Multiple Sclerosis Trial Group*

The NEW ENGLAND JOURNAL of MEDICINE

BRIEF REPORT

Progressive Multifocal Leukoencephalopathy after Natalizumab Therapy for Crohn's Disease

Gert Van Assche, M.D., Ph.D., Marc Van Ranst, M.D., Ph.D., Raf Sciot, M.D., Ph.D., Bénédicte Dubois, M.D., Ph.D., Séverine Vermeire, M.D., Ph.D., Maja Noman, M.D., Jannick Verbeeck, M.Sc., Karel Geboes, M.D., Ph.D., Wim Robberecht, M.D., Ph.D., and Paul Rutgeerts, M.D., Ph.D.

The NEW ENGLAND JOURNAL of MEDICINE

BRIEF REPORT

Progressive Multifocal Leukoencephalopathy in a Patient Treated with Natalizumab

Annette Langer-Gould, M.D., Scott W. Atlas, M.D., Andrew W. Bollen, M.D., and Daniel Pelletier, M.D.

The NEW ENGLAND JOURNAL of MEDICINE

BRIEF REPORT

Progressive Multifocal Leukoencephalopathy Complicating Treatment with Natalizumab and Interferon Beta-1a for Multiple Sclerosis

B.K. Kleinschmidt-DeMasters, M.D., and Kenneth L. Tyler, M.D.

14. ROSTOCKER

MULTIPLE SKLEROSE-SYMPOSIUM

Natalizumab (Tysabri) im klinischen Alltag

Rostock, 13.12.2006

Anerkanntes MS-Zentrum
Regionales MS-Zentrum

Das Zertifikat der DMSG
für Akutkliniken, Rehabilitationskliniken,
Schwerpunktpraxen und
neurologische Praxen/Praxisverbund

DMSG

DEUTSCHE MULTIPLE SKLEROSE GESELLSCHAFT
BUNDESVERBAND E.V.