Neues aus der Forschung

6. Schweizer ALS Tag, REHAB Basel

25.10.2014

Prof. Dr. Markus Weber

Muskelzentrum/ALS Clinic, Kantonsspital St.Gallen
Neurologische Klinik, Universitätsspital Basel

Kantonsspital Basel
Universitätskliniken

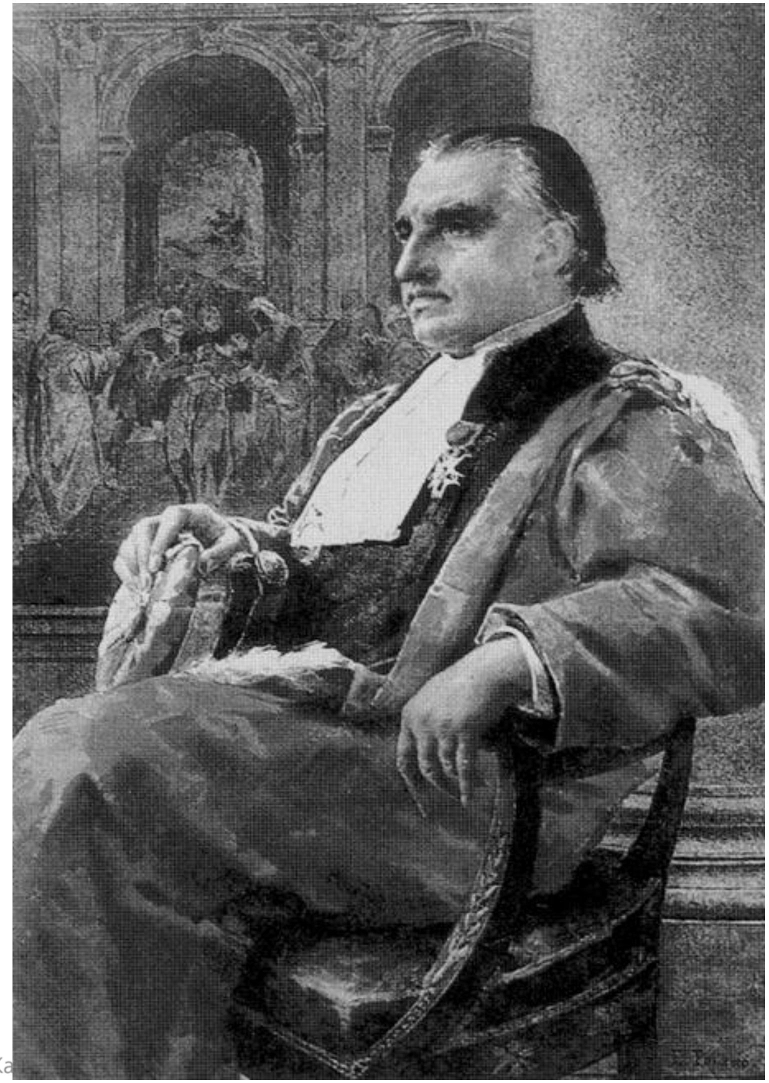
KSSG

Was ist ALS Forschung?

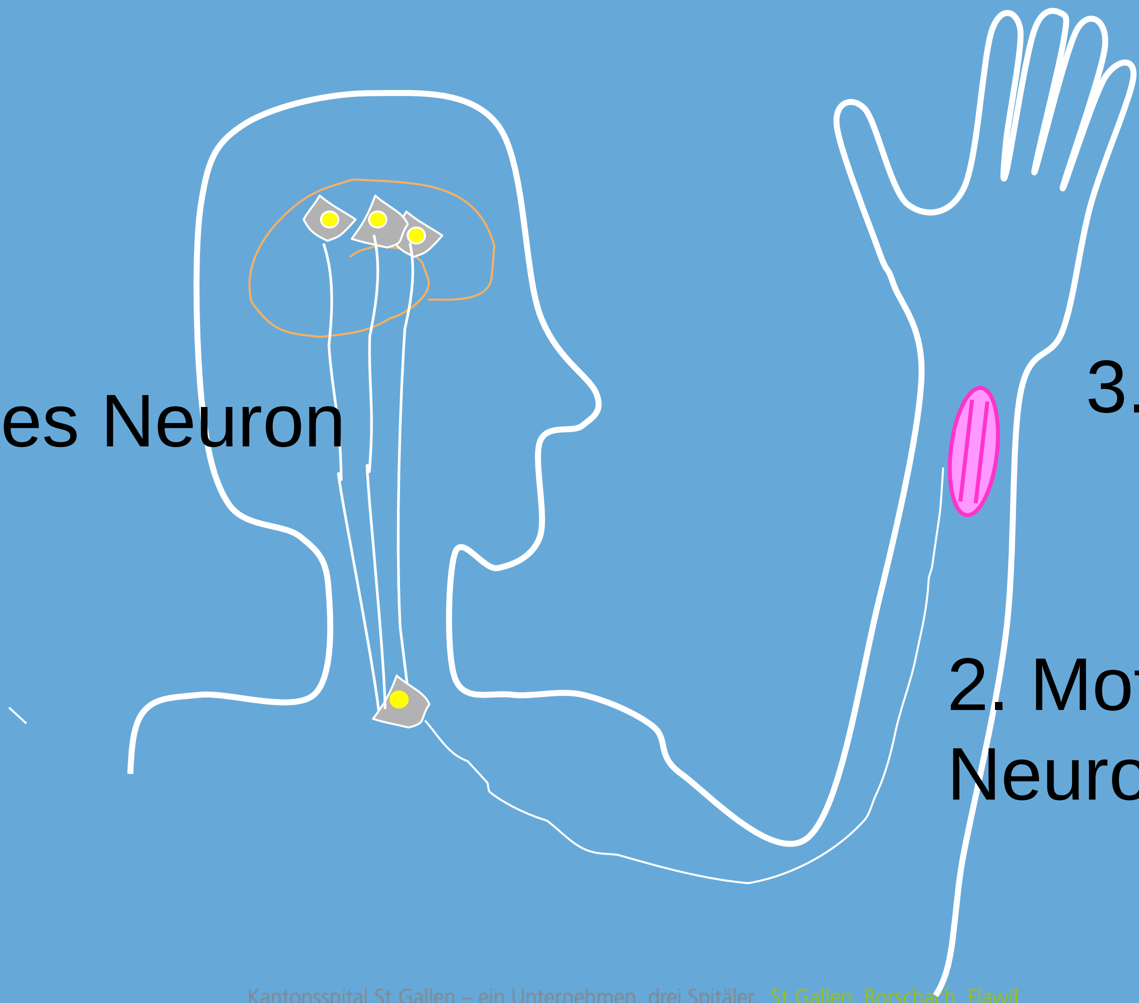
- Ursachenforschung
  - Genetik
  - Krankheitsmodelle
- Klinische (angewandte) Forschung
  - Lebensqualität
  - Versorgungsforschung
  - Biomarker
    - Messung des Krankheitsverlaufes
    - Messinstrumente zur Erfassung von kognitiven Störungen
- Therapiestudien

Was ist ALS?

J M Charcot



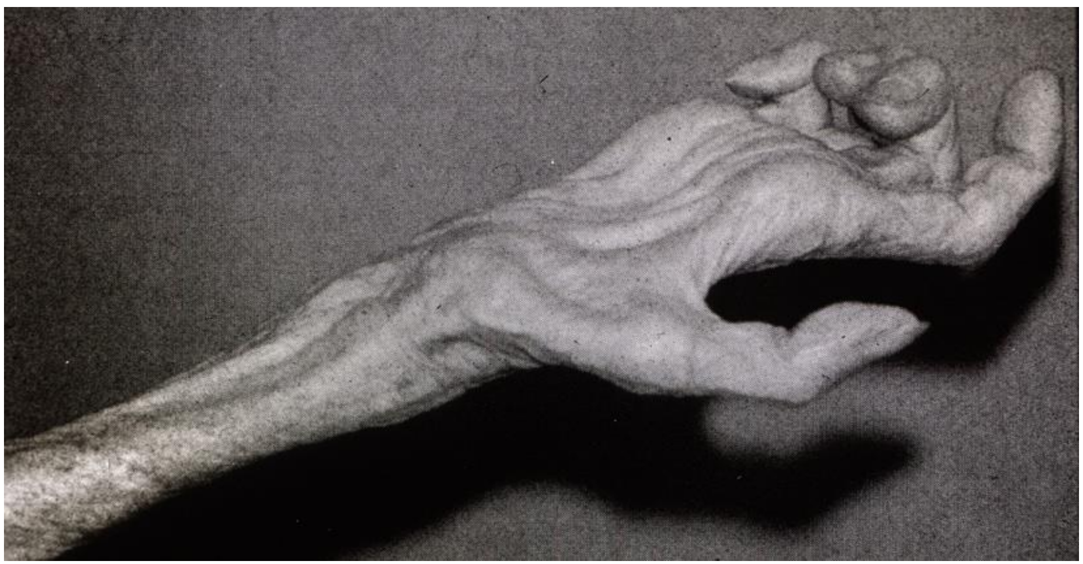
DAS MOTORISCHE SYSTEM

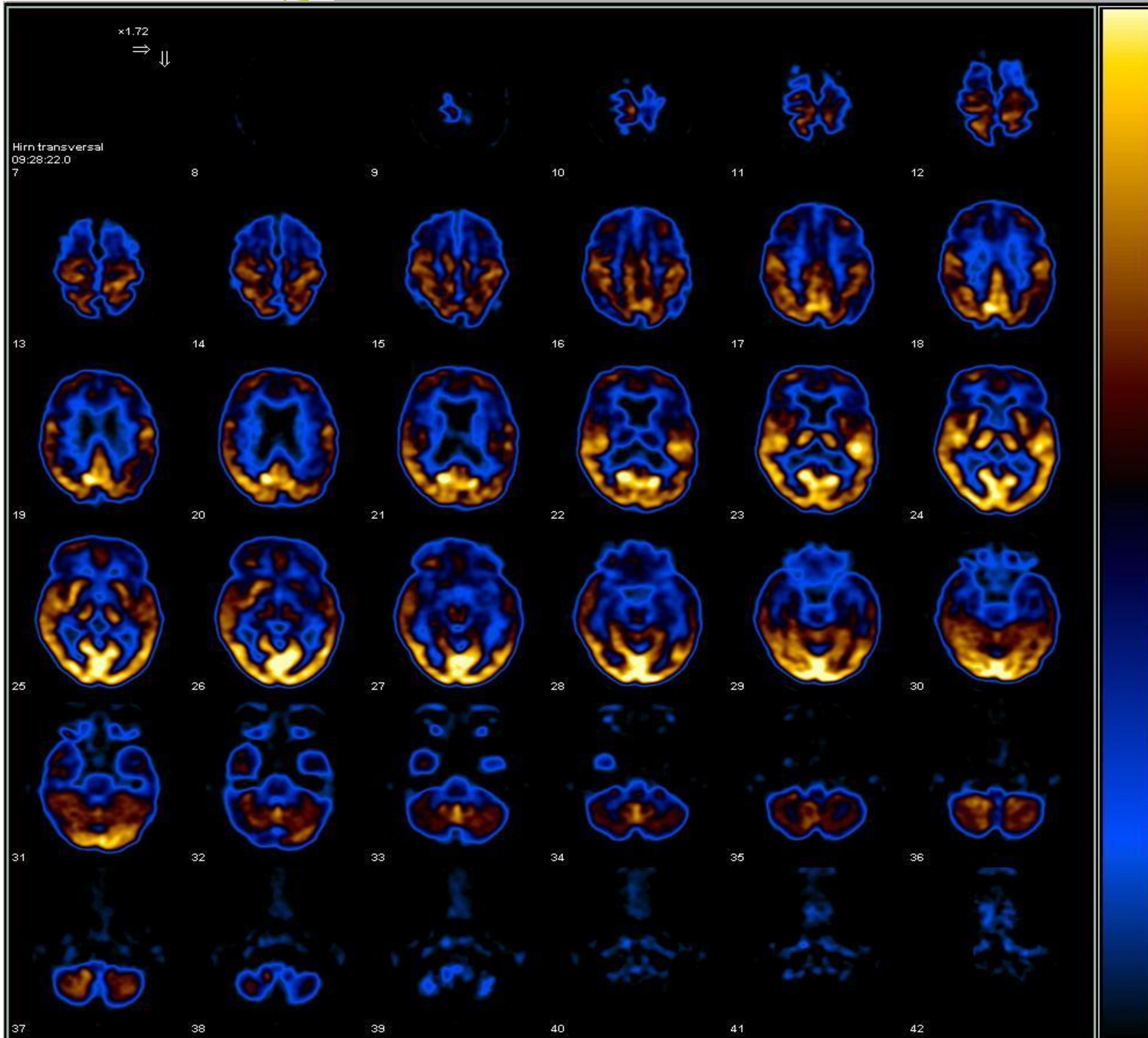


ひろた

Leitsysmptom

- Schädigung des 2. Motorisches Neuron führt zu Muskelschwund
- Normalerweise fokaler (asymmetrischer) Beginn
  - Spinale Verlaufsform (2/3)
    - Arme und Beine
  - Bulbäre Verlaufsform (20-25%)
    - Schluckstörung
    - Sprechstörung




×1.72
Hirn transversal
09:28:22.0
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42

Kognitive Störungen und Demenz- Wie viele sind betroffen und wann?

- Verlaufsstudie (Phukan et al. JNNP, 2010)
  - 87 Patienten
  - 32% kognitive Störungen zum Zeitpunkt der Erstvorstellung
  - 8% Verhaltensauffälligkeiten
  - 18% Demenz
- Vor, während oder nach dem Auftreten motorischer Symptome

Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2014; Early Online: 1–8

informa healthcare

ORIGINAL ARTICLE

The Edinburgh Cognitive and Behavioural Amyotrophic Lateral Sclerosis Screen: A cross-sectional comparison of established screening tools in a German-Swiss population

DOROTHÉE LULÉ[1], CHRISTIAN BURKHARDT[2], SUSANNE ABDULLA[3,4], SARAH BÖHM[1], KATJA KOLLEWE[3], INGO UTTNER[1], SHARON ABRAHAMS[5], THOMAS H. BAK[5], SUSANNE PETRI[3], MARKUS WEBER[2] & ALBERT C. LUDOLPH[1]

[1]*Department of Neurology, University of Ulm, Germany,* [2]*Kantonsspital St. Gallen, Neuromuscular Disease Centre/ ALS Clinic, Switzerland,* [3]*Department of Neurology, Hannover Medical School, Germany,* [4]*Department of Neurology, Otto-von-Guericke University Magdeburg, German Centre for Neurodegenerative Diseases, Magdeburg, Germany, and* [5]*Psychology-PPLS, Euan MacDonald Centre for Motor Neuron Disease Research and Anne Rowling Regenerative Neurology Clinic, University of Edinburgh, UK*

Woher kommt ALS ?

ALS ist ein komplexe Erkrankung

Genetische Faktoren

Risikofaktoren und Umweltfaktoren-
grösstenteils unbekannt

Figure 1 Timeline of gene discoveries in familial and sporadic ALS. Values represent the proportion of ALS explained by each gene in populations of European ancestry. References are provided in the main text.

Renton et al. Nature Neuroscience, 2014

C9Orf72 bei Schweizer Patienten (unpubliziert)

- 20/43 FALS (46%)
- 13/182 SALS (7%)
- Krankheitsdauer: 1-11 Jahre
- Eineiige Zwillinge
  - Einer verstarb 2007, im Alter von 55 Jahren, Krankheitsdauer 21 Monate
  - Der andere keine Symptome

Joint Programme - Neurodegenerative Disease Research
Transnational research proposals 2013

"European research projects for the identification of genetic, epigenetic and environmental risk and protective factors for Neurodegenerative Diseases"

JPND research
EU Joint Programme – Neurodegenerative Disease Research

Proposal Application Form

I. General information

Acronym (10 characters max.): STRENGTH

Project Title: Survival, Trigger and Risk, Epigenetic, eNvironmental and Genetic Targets for motor neuron Health

Project Coordinator: **Ammar Al-Chalabi**

Name	Ammar Al-Chalabi
Institution/Department	King's College London, Department of Clinical Neuroscience
Address	London SE5 8AF
Country	UK
Phone + Fax	+44 20 7848 5187 (P) +44 20 7848 5190 (F)
Email	ammar.al-chalabi@kcl.ac.uk

Total funding applied for: 4,253,547 €

Muskelzentrum/ALS Clinic

Total Funding: To Be Finalised*

Start Date: February, 2014

Duration: 3 years

Coordinator: Ammar Al-Chalabi

T: +44-20784-85187

E: ammar.al-chalabi@kcl.ac.uk

Project Partners:

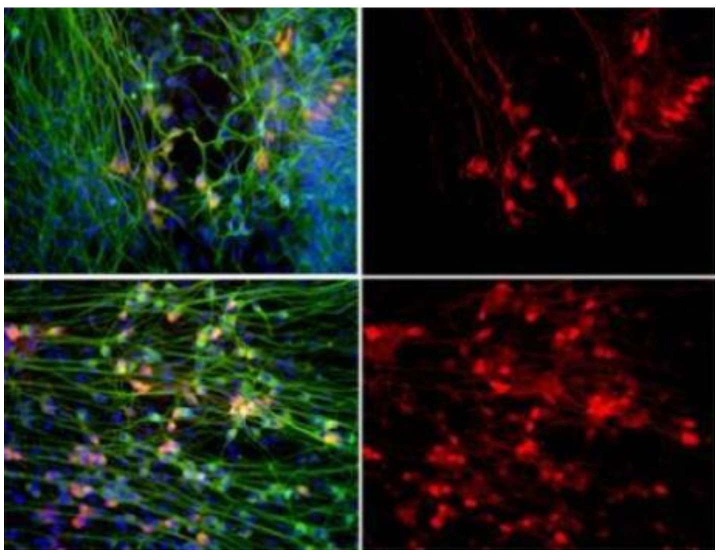
COORDINATOR I AMMAR AL-CHALABI

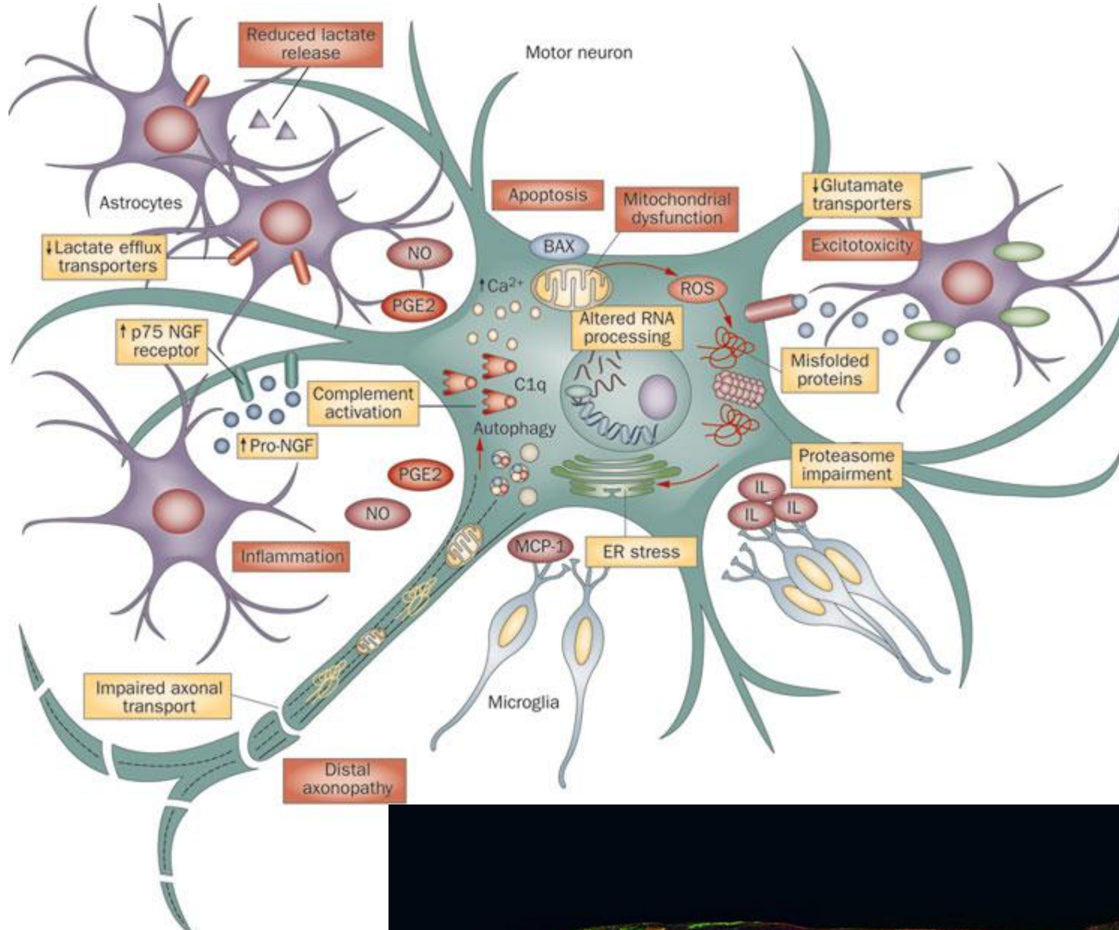
- **Ammar Al-Chalabi**, King's College London, United Kingdom
- **Pamela Shaw**, University of Sheffield, United Kingdom
- **Leonard van den Berg**, University Medical Centre Utrecht, Netherlands
- **Wim Robberecht**, Katholieke Universiteit Leuven, Belgium
- **Adriano Chio**, University of Turin, Italy
- **Vincenzo Silani**, Istituto Auxologico Italiano, Italy
- **Philippe Corcia**, INSERM U930 - Université François Rabelais, France
- **Markus Weber**, Kantonsspital St. Gallen, Switzerland
- **Weimin Ye**, Karolinska Institutet, Sweden
- **Jochen Weishaupt**, University of Ulm, Germany
- **Orla Hardiman**, Trinity College Dublin, Ireland

Muskelzentrum/ALS Clinic

Muskelzentrum/ALS Clinic

Ursachenforschung





Klinische Forschung

Lebensqualität

(auch der Angehörigen)

frontiers in PSYCHOLOGY

ORIGINAL RESEARCH ARTICLE
published: 25 October 2012
doi: 10.3389/fpsyg.2012.00443

Attitudes toward assisted suicide and life-prolonging measures in Swiss ALS patients and their caregivers

Ralf Stutzki[1]*, *Ursula Schneider*[2], *Stella Reiter-Theil*[1] *and Markus Weber*[2]

[1] *Clinical Ethics Support and Accompanying Research, University Hospital Basel/Psychiatric University Hospitals Basel, Institut für Bio- und Medizin Ethik, University of Basel, Basel, Switzerland*
[2] *Muskelzentrum/ALS Clinic, Kantonsspital St. Gallen, St. Gallen, Switzerland*

Edited by:
Francesco Pagnini, Catholic University of Milan, Italy

Reviewed by:
Gian Mauro Manzoni, Istituto Auxologico Italiano IRCCS, Italy
Francesco Pagnini, Catholic University of Milan, Italy

***Correspondence:**
Ralf Stutzki, Clinical Ethics Support and Accompanying Research, Universitäre Psychiatrische Kliniken, Schanzenstrasse 13, 4056 Basel, Switzerland.
e-mail: ralf.stutzki@unibas.ch

Objectives: In Switzerland, assisted suicide (AS) is legal, provided that the person seeking assistance has decisional capacity and the person assisting is not motivated by reasons of self-interest. However, in this particular setting nothing is known about patients' and their caregivers' attitudes toward AS and life-prolonging measures. **Methods:** Data was retrieved through validated questionnaires and personal interviews in 33 patients and their caregivers covering the following domains: physical function according to the revised Amyotrophic Lateral Sclerosis Functional Rating Scale (ALSFRS-R), demographic data, quality of life, anxiety, depression, social situation, spirituality, burden of disease, life-prolonging, and life-shortening acts. **Results:** In patients the median time after diagnosis was 9 months (2–90) and the median Amyotrophic Lateral Sclerosis (ALS) FRS-R score was 37 (22–48). The majority of patients (94%; $n = 31$) had no desire to hasten death. Patients' and caregivers' attitudes toward Percutaneous Endoscopic Gastrostomy (PEG) and Non-Invasive Ventilation (NIV) differed. Significantly more patients than caregivers (21.2 versus 3.1%) stated that they were against NIV ($p = 0.049$) and against PEG (27.3 versus 3.1%; $p = 0.031$). Answers regarding tracheotomy were not significantly different ($p = 0.139$). Caregivers scored significantly higher levels of "suffering" ($p = 0.007$), "loneliness" ($p = 0.006$), and "emotional distress" answering the questionnaires ($p < 0.001$). Suffering ($p < 0.026$) and loneliness ($p < 0.016$) were related to the score of the Hospital Anxiety and Depression Scale (HADS) in patients. **Conclusion:** A liberal legal setting does not necessarily promote the wish for AS. However, the desire to discuss AS is prevalent in ALS patients. There is a higher level of suffering and loneliness on the caregivers' side. A longitudinal study is warranted.

Keywords: ALS, motor neuron disease, quality of life, depression, end of life

Symptombehandlung z. B. Krämpfe

Chininsulfat (Chininsulfat Hänseler®, Limptar N®)

- Studie in St.Gallen
- Placebo-kontrolliert
- Tägliche Muskelkrämpfe
- Dauer 8 Wochen
- Kontakt: christoph.neuwirth@kssg.ch

Table 1. ALS clinical trials.*

Drug	Size†	Year
Acetylcysteine	110	1995[193]
Amantadine / guanidine	20	1981[194]
Arimoclomol	84	2008[195]
β-1a interferon	61	2000[196]
Baclofen	23	1979[197]
BCAA	22	1988[198]
BCAA	126	1993[199]
BCAA / threonine	95	1996[200]
BDNF	1135	1999[201]
BDNF	25	2000[202]
Celecoxib	300	2006[203]
Celecoxib / minocycline	60	2008[204]
CNTF	730	1996[205]
Coenzyme Q10	185	2009[206]
Creatine	175	2003[207]
Creatine	104	2004[208]
Creatine	107	2008[209]
Cyclosporine	74	1988[210]
Dexpramipexole	102	2011[19]
Dexpramipexole	616	2013[18]
Dextromethorphan	14	1993[211]
Dextromethorphan	49	1996[212]
Dextromethorphan	45	1997[213]
Dextromethorphan / quinidine	140	2004[214]
Gabapentin	152	1996[215]
Gabapentin	231	1998[216]
Gabapentin	204	2001[217]
Gangliosides	40	1984[218]
Gangliosides	40	1984[219]
G-CSF	24	2011[220]
Glatiramer	30	2006[221]
Glatiramer	366	2009[222]
Glutathione	32	1998[223]
Growth hormone	40	2012[224]
Guanidine	24	1974[225]
IGF-1	75	1993[226]
IGF-1	266	1997[227]
IGF-1	183	1998[228]
IGF-1	9	2005[229]
IGF-1	330	2008[230]
Indinavir	46	2005[231]
Inosiplex	16	1974[232]
Isoprinosine	25	1971[233]
Lamotrigine	67	1993[234]
Lamotrigine	30	2003[235]
Levamisole	59	1985[236]
Lithium	84	2010[237]
Lithium	133	2012[238]
Lithium	214	2013[239]
L-threonine	23	1992[240]
L-threonine	30	1992[241]
Memantine	63	2010[242]
Methionine / antioxidants	28	2001[243]
Methylcobalamin	24	1998[244]
Minocycline	19	2004a[245]
Minocycline	23	2004b[245]
Minocycline	412	2007[246]
Nimodipine	87	1996[247]
Pentoxifylline	400	2006[248]
Physostigmine	25	1993[249]
Riluzole	155	1994[13]
Riluzole	959	1996[14]
Riluzole	15	1998[250]
Riluzole	168	2002[251]
Selegiline	111	1994[252]
Selegiline	10	1994[253]
Selegiline	133	1998[254]
Talampanel	59	2010[255]
TCH346	553	2007[256]
Thalidomide	37	2008[257]
Thyrotropin	30	1986[258]
Thyrotropin	12	1986[259]
Thyrotropin	41	1986[260]
Thyrotropin	25	1987[261]
Thyrotropin	23	1991[262]
Thyrotropin	36	1992[263]
Tilorone	16	1978[264]
Topiramate	296	2003[265]
Transfer factor	66	1979[266]
Ursodeoxycholic acid	64	2012[267]
Valproic acid	163	2009[268]
Vitamin E	289	2001[269]
Vitamin E	160	2005[270]
Xaliproden	78	2004[271]
Xaliproden	867	2004a[272]
Xaliproden	1210	2004b[272]

BCAA, branched-chain amino acids; BDNF, brain-derived neurotrophic factor; CNTF, ciliary neurotrophic factor; G-CSF, granulocyte colony-stimulating factor; IGF-1, insulin-like growth factor-1; TCH346, omigapil.

**Modified from Beghi et al.*[16] *(2011) with permission of the publisher.*

†*Total number of patients in study.*

3%. All genes found to be mutated in FALS can also be found to be mutated in SALS, albeit at low frequencies.[38] Even in cases with known genetic etiology, it is unclear how the genetic insult, present in all somatic cells, induces selective vulnerability in motor neurons.[39,40]

An understanding of genes involved in ALS may be essential in developing effective therapeutics. In what follows is a discussion of specific genes linked to ALS. This discussion is not exhaustive, but rather focuses on genes more commonly linked to ALS in epidemiologic studies and also includes rare genetic alterations if they shed light on novel pathways or phenotypes. Genetic mutations that are very rare or those with contributions to ALS that are less well documented are included and referenced in the tables of causative genes (Table 2) or modifiers of disease risk or progression (Table 3), but are not discussed in detail.

Cu/Zn-Superoxide Dismutase (*SOD1*). Modern genetic studies of ALS began with the identification of 11 missense mutations in *SOD1* in 13 distinct FALS families.[21] The discovery was the first to pinpoint a genetic cause for ALS, paving the way for *in vivo* models of FALS that enabled analysis of disease mechanisms[41,42] and large-scale ALS bioinformatics

Verzögerung des Krankheitsverlaufes- negative klinische Studien bei ALS

Zahlreiche neue Studien sind geplant

Heute alle randomisiert placebo-kontrolliert

Grösster Benefit wahrscheinlich für nachkommende Patienten

Warum waren alle bisherigen Studien negativ ?

- Sehr unterschiedliche Krankheitsverläufe
- Modelle können Therapieeffekte beim Menschen nicht vorhersagen
- Keine Messinstrumente (Biomarker) zur Messung des Krankheitsverlaufes


PROJECTS SUPPORTED BY JPND

JPND research

SOPHIA

Sampling and biomarker OPtImization and HarmonizAtion in ALS and other motor neuron diseases

Amyotrophic Lateral Sclerosis (ALS) is one of the most devastating diseases in neurology affecting some 50,000 individuals at any time in Europe, and causing around 10,000 deaths each year. The main clinical features are weakness and wasting of muscles, but dementia may also occur. ALS represents a good model for study of all neurodegenerative conditions, as it has a characteristic phenotype, rapid progression and the correlation between diagnosis during life and autopsy diagnosis is close to 100%. However, validated neurochemical biomarkers for monitoring disease activity, for generating earlier diagnoses and for defining prognosis are lacking. Active European collaborations are in place for harmonizing clinical datasets, neuroimaging and neuropathology protocols. A preliminary strategy for harmonization of biological and tissue samples has been established. Standardized protocols for clinical data and sample collection are now urgently required for optimization and harmonization of biomarker development.

The overall aim of this project is to provide a common European strategy for the prioritization and selection of candidate biomarker domains for optimization and harmonization. This will in turn provide a long-term platform by which existing collaborative structures that are relevant to neurodegenerative disease biomarkers (including academic initiatives, co-funding strategies, biobanks, industrial efforts, private-public alliances) are integrated within an inclusive web-based virtual biobank. Samples and clinical / imaging / neurophysiologic and neuro-pathological datasets provided by participating members can then be optimally utilized to enable state of the art collaborative analyses.

The established platform will also act as an important communication channel between this consortium and the rest of the ALS/Neurodegeneration field to ensure that the optimization efforts are in line with the whole ALS/ND field, to avoid duplication of work, and to ensure better acceptance and utilization of the project results by all stakeholders. Ultimately, the platform will be used to disseminate the results to the whole ALS/Neurodegeneration field, and will act as a permanent interactive European ALS biomarker platform for researchers to optimize/harmonize novel biomarkers using an established pan-European ALS methodology. The platform will also allow interaction with those of other cognate groups (e.g. the NEALS group in the USA) and with patient groups and other relevant stakeholders.

Total Funding: € 2.4 million (approx.)
Start Date: June 2012
Duration: 3 years
Coordinator: Leonard Van den Berg
T: +31 88 7557939
E: lberg@umcutrecht.nl

SOPHIA

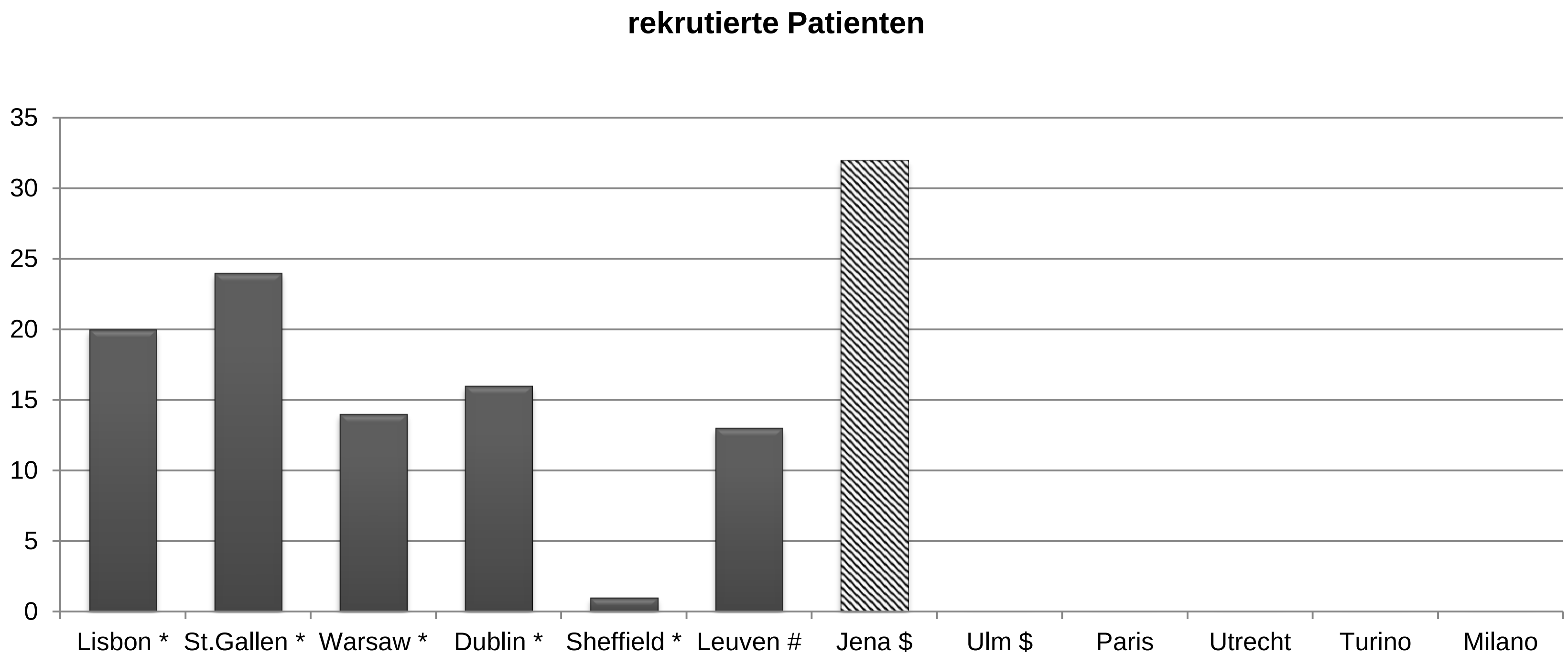
Project Partners:

COORDINATOR LEONARD VAN DEN BERG

Leonard Van den Berg, University Medical Center Utrecht, Netherlands
Wim Robberecht, Vlaams Instituut voor Biotechnologie vzw, Belgium
François Salachas, Assistance Publique-Hopitaux de Paris, France
Markus Otto, University of Ulm, Germany
Julian Grosskreutz, University Hospital Jena, Germany
Orla Hardiman, Trinity College Dublin, Ireland
Adriano Chio, University of Turin, Italy
Vincenzo Silani, Istituto Auxologico Italiano, Italy
Magdalena Kuzma-Kozakiewicz, Medical University of Warsaw, Poland
Mamede De Carvalho, Instituto de Medicina Molecular, Portugal
Julia Costa, Instituto de Tecnologia Química e Biológica, Portugal
Jesus S. Mora Pardina, Servicio Madrileño de Salud, Spain
Markus Weber, Kantonsspital St.Gallen, Switzerland
Yesim Parman, Istanbul University, Turkey
Pamela Shaw, University of Sheffield, United Kingdom
Martin Turner, Oxford University Nuffield Department of Clinical Neurosciences, UK

Bestimmung der Zahl motorischer Nerven (MUNIX)

MUNIX Qualifikationsprozess

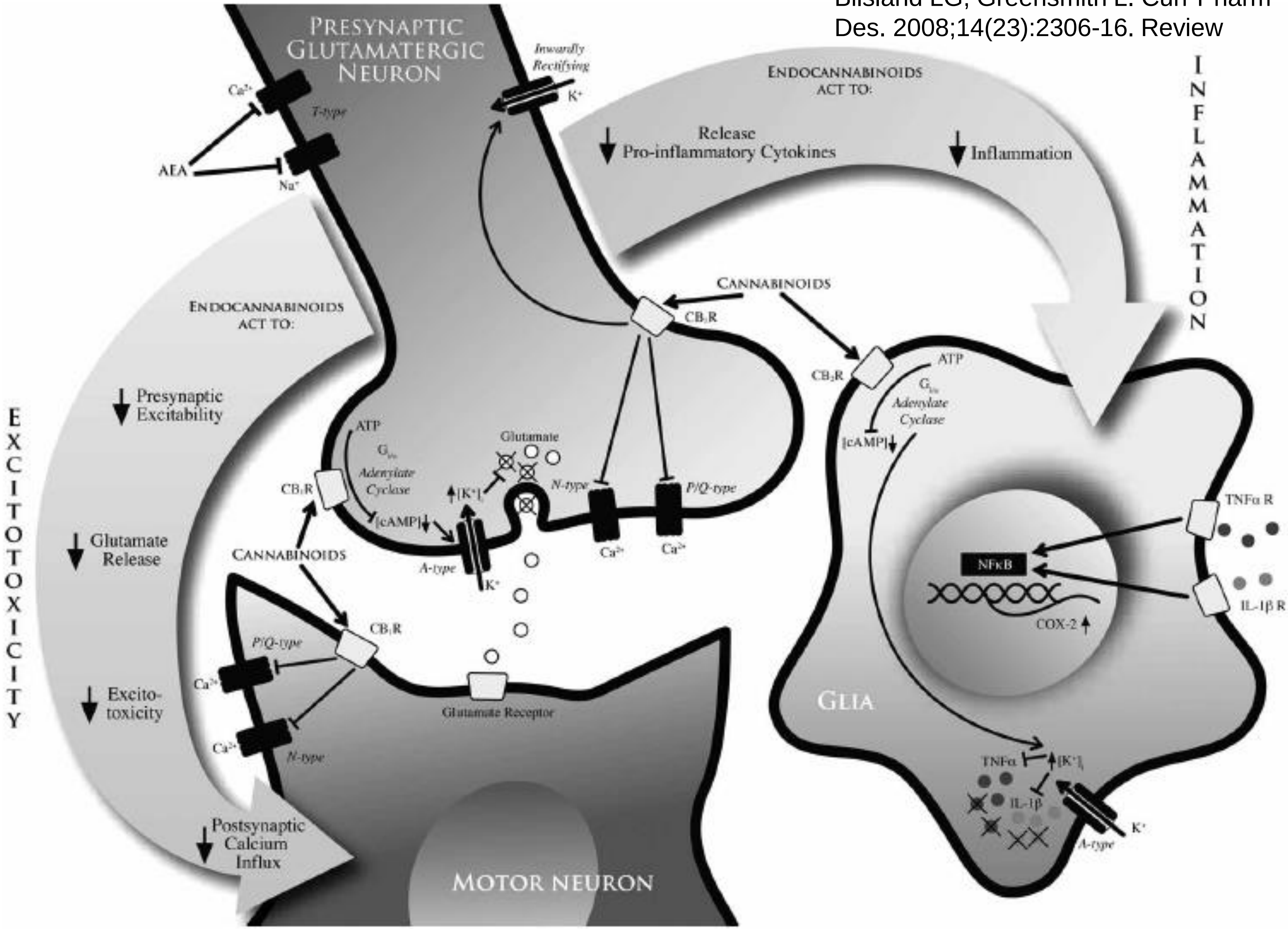


*** = test-retest certified centre**
 # = Certified with provisions and monitored
$ = test retest certification failed

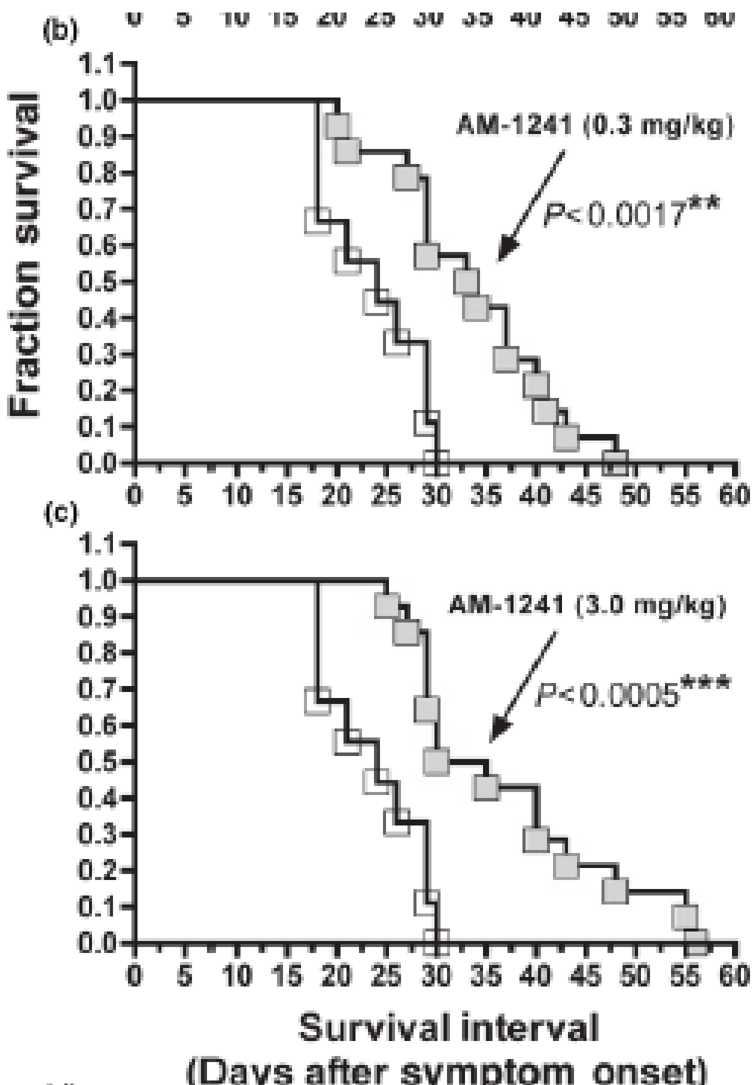
Blick in die Zukunft


MARIJUANA
IS
MEDICINE

Bilsland LG, Greensmith L. Curr Pharm Des. 2008;14(23):2306-16. Review

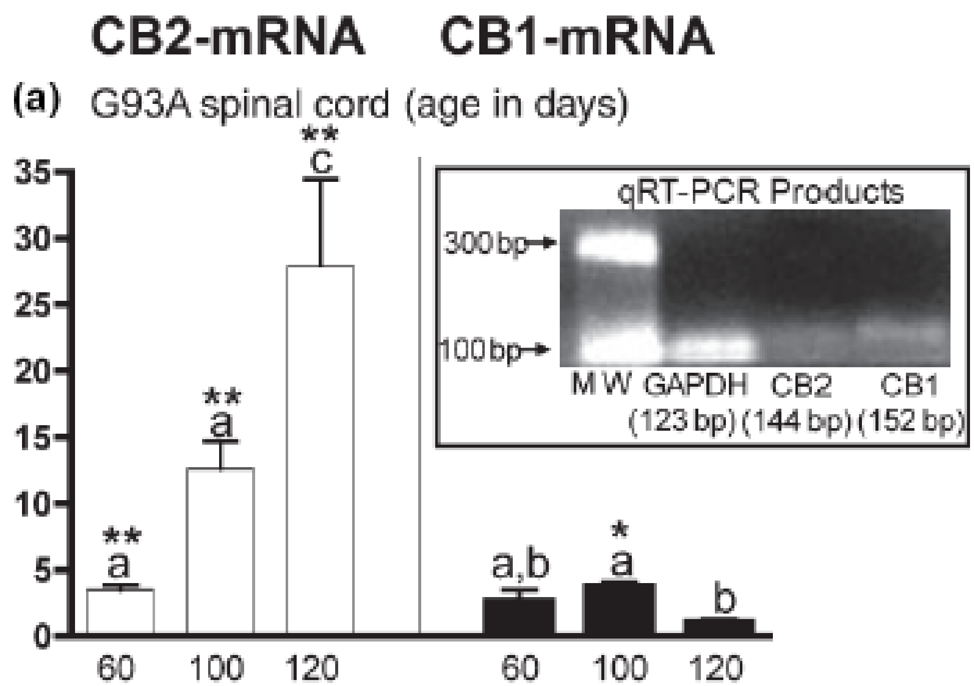


J Neurochem. 2007 April ; 101(1): 87. doi:10.1111/j.1471-4159.2006.04346.x.

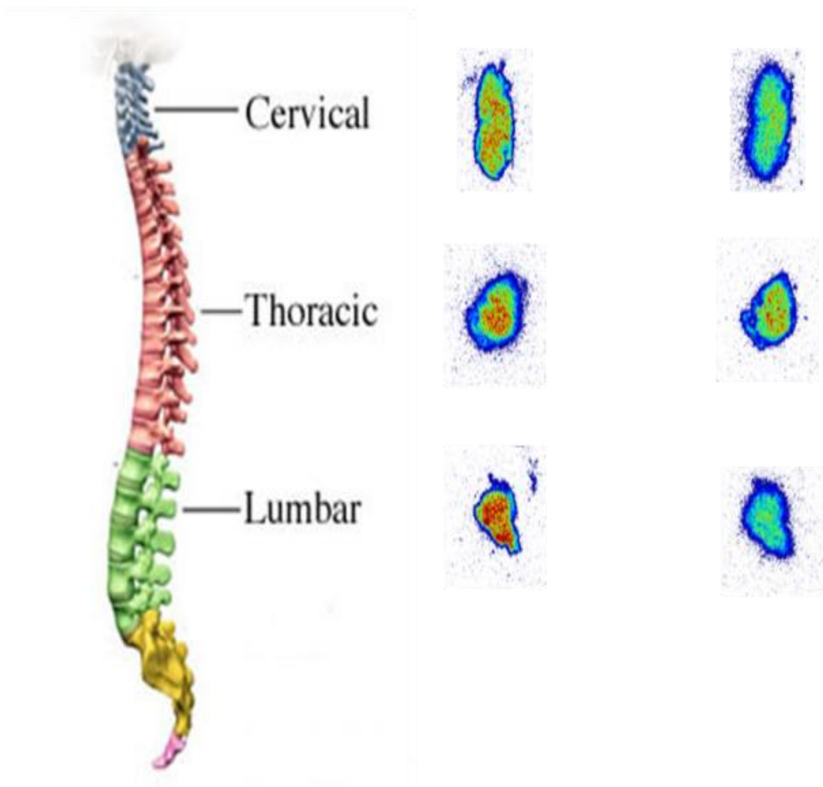


The CB2 cannabinoid agonist AM-1241 prolongs survival in a transgenic mouse model of amyotrophic lateral sclerosis when initiated at symptom onset

Jennifer L. Shoemaker, **Kathryn A. Seely**, **Ronald L. Reed**, **John P. Crow**, and **Paul L. Prather**
Department of Pharmacology and Toxicology, College of Medicine, University of Arkansas for Medical Sciences, Little Rock, Arkansas, USA



In vitro autoradiography



- Figure 2 In vitro autoradiography with slices from spinal cord of an ALS patient. A: Incubation with 0.2 nM [^{11}C]KD2; B: Blocking of [^{11}C]KD2 binding with 1 µM GW405833, a specific CB2R agonist.
- Courtesy of Mu, Linjing, PhD

Forschungsprojekt in Zusammenarbeit mit ETH

Journal of Neurochemistry JNC

JOURNAL OF NEUROCHEMISTRY | 2013 doi: 10.1111/jnc.12354

ORIGINAL ARTICLE

Radiolabeling and *in vitro* /*in vivo* evaluation of N-(1-adamantyl)-8-methoxy-4-oxo-1-phenyl-1,4-dihydroquinoline-3-carboxamide as a PET probe for imaging cannabinoid type 2 receptor

Linjing Mu,* Daniel Bieri,† Roger Slavik,† Konstantin Drandarov,* Adrienne Müller,† Stjepko Čermak,†[1] Markus Weber,‡ Roger Schibli,*† Stefanie D. Krämer† and Simon M. Ametamey†

**Department of Nuclear Medicine, Center for Radiopharmaceutical Sciences of ETH-PSI-USZ, University Hospital Zürich, Zürich, Switzerland*

†Department of Chemistry and Applied Biosciences, Center for Radiopharmaceutical Sciences of ETH-PSI-USZ, Institute of Pharmaceutical Sciences, ETH Zurich, Zürich, Switzerland

‡Neuromuscular Diseases Unit/ALS Clinic, Kantonsspital St. Gallen, St. Gallen, Switzerland

doi:10.1093/brain/awp239 Brain 2009: 132; 3152–3164 | 3152

BRAIN
A JOURNAL OF NEUROLOGY

Microglial CB_2 cannabinoid receptors are neuroprotective in Huntington's disease excitotoxicity

Javier Palazuelos,[1,2] Tania Aguado,[1,2] M. Ruth Pazos,[1,3] Boris Julien,[1,2] Carolina Carrasco,[1,2] Eva Resel,[1,2] Onintza Sagredo,[1,3] Cristina Benito,[1,4] Julián Romero,[1,4] Iñigo Azcoitia,[5] Javier Fernández-Ruiz,[1,3] Manuel Guzmán[1,2] and Ismael Galve-Roperh[1,2]

BJP British Journal of Pharmacology

DOI:10.1111/j.1476-5381.2011.01278.x
www.brjpharmacol.org

Themed Issue: Cannabinoids in Biology and Medicine, Part I

RESEARCH PAPER

Symptom-relieving and neuroprotective effects of the phytocannabinoid Δ^9-THCV in animal models of Parkinson's disease

C García[1,2,3], C Palomo-Garo[1,2,3], M García-Arencibia[1,2], JA Ramos[1,2,3], RG Pertwee[4] and J Fernández-Ruiz[1,2,3]

[1]Departamento de Bioquímica y Biología Molecular, Instituto Universitario de Investigación en Neuroquímica, Facultad de Medicina, Universidad Complutense, Madrid, Spain, [2]Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain, [3]Instituto Ramón y Cajal de Investigación Sanitaria (IRYCIS), Madrid, Spain, and [4]School of Medical Sciences, Institute of Medical Sciences, University of Aberdeen, Foresterhill, Aberdeen, UK

Correspondence
Javier Fernández-Ruiz, Department of Biochemistry and Molecular Biology, Faculty of Medicine, Complutense University, 28040-Madrid, Spain. E-mail: jjfr@med.ucm.es

Keywords
Δ^9-THCV; cannabinoid receptors; Parkinson's disease; 6-hydroxydopamine-lesioned rats; LPS-lesioned mice; neuroprotection

Received
17 November 2010
Revised
21 December 2010
Accepted
18 January 2011

Journal of Alzheimer's Disease 35 (2013) 847–858
DOI 10.3233/JAD-130137
IOS Press

CB_2 Cannabinoid Receptor Agonist Ameliorates Alzheimer-Like Phenotype in AβPP/PS1 Mice

Ester Aso[a,*], Salvador Juvés[a,b], Rafael Maldonado[c] and Isidro Ferrer[a,b]

[a]*Institut de Neuropatologia, Servei d'Anatomia Patològica, IDIBELL-Hospital Universitari de Bellvitge, Universitat de Barcelona, L'Hospitalet de Llobregat, Spain*

[b]*CIBERNED, Centro de Investigación Biomédica en Red de Enfermedades Neurodegenerativas, Instituto Carlos III, Spain*

[c]*Laboratori de Neurofarmacologia, Departament de Ciències Experimentals i de la Salut, Universitat Pompeu Fabra, Barcelona, Spain*

American Journal of Hospice & Palliative Medicine®
000(00) 1-10

DOI: 10.1177/1049909110369531
http://ajhpm.sagepub.com
SAGE

Cannabis and Amyotrophic Lateral Sclerosis: Hypothetical and Practical Applications, and a Call for Clinical Trials

Gregory T. Carter, MD, MS[1], Mary E. Abood, PhD[2], Sunil K. Aggarwal, PhD[3], and Michael D. Weiss, MD[1,4,5]

Abstract

Significant advances have increased our understanding of the molecular mechanisms of amyotrophic lateral sclerosis (ALS), yet this has not translated into any greatly effective therapies. It appears that a number of abnormal physiological processes occur simultaneously in this devastating disease. Ideally, a multidrug regimen, including glutamate antagonists, antioxidants, a centrally acting anti-inflammatory agent, microglial cell modulators (including tumor necrosis factor alpha [TNF-α] inhibitors), an antiapoptotic agent, 1 or more neurotrophic growth factors, and a mitochondrial function-enhancing agent would be required to comprehensively address the known pathophysiology of ALS. Remarkably, cannabis appears to have activity in all of those areas. Preclinical data indicate that cannabis has powerful antioxidative, anti-inflammatory, and neuroprotective effects. In the G93A-SOD1 ALS mouse, this has translated to prolonged neuronal cell survival, delayed onset, and slower progression of the disease. Cannabis also has properties applicable to symptom management of ALS, including analgesia, muscle relaxation, bronchodilation, saliva reduction, appetite stimulation, and sleep induction. With respect to the treatment of ALS, from both a disease modifying and symptom management viewpoint, clinical trials with cannabis are the next logical step. Based on the currently available scientific data, it is reasonable to think that cannabis might significantly slow the progression of ALS, potentially extending life expectancy and substantially reducing the overall burden of the disease.

Keywords

cannabis, endocannabinoids, amyotrophic lateral sclerosis, clinical trials, motor neuron disease

FRAGEN ?