17th State of the Art

Symposium of the Swiss Multiple Sclerosis Society  www.multiplesklerose.ch

Programme | Abstracts
Saturday, January 24th, 2015

KKL Lucerne, Culture and Convention Centre

Recent Research Developments: Key Tools to Unmask MS
Dear colleagues,

On behalf of the Swiss Multiple Sclerosis Society and its Scientific Advisory Board it is our pleasure to invite you to the 17th State of the Art Symposium.

«Recent research developments: key tools to unmask MS» is the 2015 general theme that proposes a large review of some still unknown key aspects of MS pathophysiology. During the plenary morning session, international experts will present the most recent promising advances in different fields. Martin Weber and Roland Liblau will respectively explore the role of B and T cells in MS autoimmunity. Britta Engelhardt will discuss the major role of brain barriers in the control of the attack of the central nervous system in MS. Declan Chard and Manuel Comabella will review the best radiological and biological tools susceptible to help to unmask MS burden. Finally, Milo Puhan will present a preview of the National MS Registry, a key project of the Swiss MS-Society.

The afternoon session, with two sets of three parallel interactive workshops, will bring to discussion topics relevant to the daily practice. Speakers will present practical situations.

Updated information about the Symposium can be found on the website www.ms-state-of-the-art.ch

In the name of the organisers and speakers, we sincerely hope that the programme meets your interest and that you will be able to attend and actively take part in the discussions.

We wish you an interesting symposium.

PD Dr. Myriam Schluep  Patricia Monin
President of the  Director of the
Scientific Advisory Board  Swiss MS-Society
GENERAL INFORMATION

Venue
KKL Lucerne, Culture and Convention Centre, Europaplatz 1, CH-6005 Lucerne
www.kkl-luzern.ch

Programme committee
Britta Engelhardt, Berne; Claudio Gobbi, Lugano; Patrice Lalive, Geneva; Tobias Derfuss, Basel;
Ludwig Kappos, Basel; Michael Linnebank, Zurich and Myriam Schluep, Lausanne (chair)

Organisation
Swiss MS-Society and its Scientific Advisory Board

Contact
Swiss MS-Society, Josefstrasse 129, CH-8031 Zurich
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Credits
The Swiss Neurological Society will award 5 credit points.
The Swiss Society of General Internal Medicine (SGIM/SGAM) will award 4.5 credit points.

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PROGRAMME

Chairpersons

PD Dr. Myriam Schluep, Lausanne
Prof. Roland Martin, Zurich
Prof. Ludwig Kappos, Basel

09.30-10.00  Welcome with Coffee and Gipfeli

10.00-10.10  Patricia Monin, Zurich
Welcome from the Swiss MS-Society

10.10-10.40  Prof. Martin Weber, Göttingen
The underestimated role of B cells in MS

10.40-11.10  Prof. Roland Liblau, Toulouse
T cells that recognise distinct self-antigens drive the progression of CNS autoimmunity

11.10-11.40  Prof. Britta Engelhardt, Berne
How brain barriers control immune cell entry into the CNS

11.40-12.10  Coffee Break

12.10-12.35  Dr. Declan Chard, London
Recent imaging developments to unmask MS burden

12.35-13.00  Dr. Manuel Comabella, Barcelona
Use of biomarkers to anticipate MS severity

13.00-13.15  Prof. Milo Puhan, Zurich
National MS Registry

13.15-14.15  Lunch

14.15-15.00  Interactive Workshops A, B, C

15.00-15.20  Coffee Break

15.20-16.05  Interactive Workshops D, E, F

16.05  Farewell Apero
## CONTACTS

### Programme Committee and Chairpersons

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### Speakers (Lectures)

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**Speakers (Workshops)**

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<td>Neurological Rehabilitation Unit</td>
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THE UNDERESTIMATED ROLE OF B CELLS IN MS

Prof. Martin Weber, Göttingen
University Medicine Göttingen, Clinic for Neurology

B cells were recently found to play important roles for the development and progression of central nervous system (CNS) autoimmune disease. While earlier investigations focused on the pathogenic contribution of plasma cells producing autoreactive antibodies, and differentiated B cells acting as potent antigen-presenting cells (APC) for the activation of pathogenic T cells, our recent data in experimental autoimmune encephalomyelitis (EAE) and MS have emphasized the importance of B cell-derived cytokines as drivers and regulators of disease activity (Lehmann-Horn et al., Ther Adv Neurol Disord. 2013).

In regard to this dichotomy in B cell function, we were able to demonstrate that primarily the activation status of B cells is crucial for their respective function. While antigen-activated B cells contribute as potent APC, naive tend to down-regulate pro-inflammatory function of myeloid APC, presumably by provision of regulatory IL-10 (Weber et al., Ann Neurol, 2010). Regarding the relative importance of B cells within other APC, our group could further show that B cell APC function is a prerequisite for development of CNS autoimmune disease, when larger proteins need to be recognized and presented (Molnarfi et al., J Exp Med, 2013). From this work, two translational projects derived. Firstly, we were able to demonstrate for the first time that unselective, anti-CD20 B cell depletion collaterally abolishes pre-existing regulatory B cell function in patients with MS and NMO (Lehmann-Horn et al., J Neuroinflamm, 2011). Secondly, we could demonstrate as a proof of principle that intrathecal application of anti-CD20 could be possibly more selective and efficient in targeting pathogenic B cell function in CNS autoimmune disease.
Zusammenfassung Fachinformation Betaferon.

Betaferon® – Mit Familie in die Zukunft.
The central nervous system (CNS) is confronted by a double challenge regarding its interactions with the immune system. On the one hand it should allow the immune system to fight invading pathogens and on the other it should prevent inflammatory damage given its vital functions and poorly regenerative capacity. A series of mechanisms, collectively referred to as «immune privilege», ensure that immune reactions are kept minimal and are rapidly controlled within the CNS.

However, accumulating evidence shows that T cells readily penetrate the brain and spinal cord parenchyma in numerous inflammatory, infectious or degenerative neurological diseases. The consequence for CNS resident cells, and more specifically for neurons, of their encounter with activated T cells is a question that we have addressed recently using experimental rodent models. I will present our efforts to understand how cytotoxic CD8 T cells and helper CD4 T cells can target neuronal antigens and thereby contribute to CNS tissue damage.

Intriguingly, some autoreactive T cells recognise several autoantigens but the functional significance of such «cross-reactivity» is not fully understood. We have identified, in mice, autoreactive CD4 T cells recognising both MOG and NF-M and have investigated their pathogenic contribution using animals deficient for one or the other self-antigens.

Shedding light on the mechanisms by which T cells promote CNS tissue damage may allow the design of more refined therapeutic strategies for immune-mediated neurological diseases, among which multiple sclerosis is the most frequent.
Eine neue Therapie in der 1. line-Behandlung der RRMS

* vs. Placebo gemäss DEFINE. Dosierung BID, 2 x 120 mg 1 Woche, dann 2 x 240 mg.

The central nervous system (CNS) is an immunologically privileged site to which access of circulating immune cells is tightly controlled by the endothelial blood-brain barrier (BBB) localised in CNS microvessels and the epithelial blood-cerebrospinal fluid barrier (BCSFB) within the choroid plexus. Due to the specialised structure of the CNS barriers, immune cell entry into the CNS parenchyma involves two differently regulated steps: migration of immune cells across the BBB or BCSFB into the cerebrospinal fluid (CSF) drained spaces of the CNS, followed by progression across the glia limitans into the CNS parenchyma. With a focus on multiple sclerosis and its animal models I will describe the distinct molecular mechanisms required for the migration of different immune cell subsets across the different CNS barriers during immunosurveillance and MS. I will include discussion of the therapeutic efficacy and its associated risks by therapeutic targeting of immune cell entry into the CNS in MS.
Fighting complex diseases with groundbreaking therapies.

Your partner in rare diseases and multiple sclerosis

Visit www.genzyme.ch
While white matter lesions are the most obvious pathological manifestation of multiple sclerosis (MS), and white matter lesion load the magnetic resonance imaging (MRI) measure that is most often used in clinical practice and trials, they only partly explain clinical outcomes. This has led to a search for pathology elsewhere that explains this discrepancy, and it has become apparent that white matter lesions actually represent the minority of the overall disease burden in people with MS. For example, white matter that appears to be lesion free when looked at using conventional MRI scans is abnormal when it is assessed using techniques such as magnetisation transfer imaging. Grey matter is also not spared the effects of MS, and in people with long-standing progressive disease the extent of demyelinating lesions may be significantly greater in grey matter than in white matter. Grey matter lesions are difficult to see on conventional MRI, but are detectable in much greater numbers using new MRI sequences such as double inversion recovery or phase sensitive inversion recovery. Histopathological studies have also shown that non-lesional grey matter is abnormal, and advanced MRI methods are now being used to assess this.
Referenzen
5. PSUR No. 26 for Interferon beta-1a/Rebif®, 2012;28.

Gekürzte Fachinformation Rebif®/Rebif® multidose/ Rebif® RebiDose Interferonum beta-1a ADNr. E: Patienten mit einem ersten klinischen, auf Multiple Sklerose (MS) hinweisenden neurologischen Ereignis bei Ausschluss anderer Diagnosen und günstigem Risiko für das Auftreten einer schubförmigen MS. Schubförmige MS: AD Nr. 44 µg dreimal pro Woche subkutan.
KI: Behandlungsbeginn während der Schwangerschaft, Überempfindlichkeit gegen einen Inhaltsstoff, schwerwiegende Depressionen und/oder Suizidgedanken.
V: Depressive Störungen, Kramphafte, nicht adaquate therapierte Epilepsie, Angina pectoris, kongestive Herzinsuffizienz, Arrhythmie, schwere Hypersensibilitätsreaktionen, Hautulzera, neuropsychische Störungen, akute Myokardischemie, Anorexia nervosa, Regelmäßige Kontrolle der Leberwerte, grosses, resp. ungewöhnliches ungewöhnliches Blutbild, ggf. Schilddrüsenfunktionstest. IA: Immeda-}

Merck (Schweiz) AG, Merck Serono, Chamerstrasse 174, CH-6300 Zug, Tel. +41 41 729 22 22, www.merckserono.com
Multiple sclerosis (MS) is a chronic inflammatory disease of the central nervous system which is characterized by a high degree of heterogeneity in different disease aspects, for instance, clinical manifestations, disease course, radiological findings, histopathological characteristics of brain lesions, and response to treatment. In this scenario, there is a necessity in MS of biomarkers that reliably capture these different aspects of disease heterogeneity and may help in the MS diagnosis and disease stratification, prediction of disease course, or identification of new therapies beneficial for the disease. This talk will focus on prognostic cerebrospinal fluid molecular biomarkers that may help to predict disease severity in patients presenting with a clinically isolated syndrome or CIS.
Verträglichkeit
1x täglich oral, gut verträglich1–3

Erfahrung
Über 100'000 MS-Patienten behandelt4

Wirksamkeit
52% zusätzliche Schubreduktion gegenüber Interferon Beta-1a (i.m.)

* GILENYA® ist zur Behandlung von Patienten mit schubförmig remittierender erweiterter Multipier Sklerose (MS) zur Reduzierung der Schubhäufigkeit und zur Verzögerung des Fortschreitens der Behinderung indiziert.

Referenzen:


[Image of the GILENYA® package insert with additional text and diagrams not shown]
NATIONAL MS REGISTRY

Prof. Milo Puhan, Zurich
University of Zurich, Epidemiology, Biostatistics & Prevention Institute

The Swiss MS-Society has committed itself to funding a Swiss Multiple Sclerosis Registry (SMSR). The main goal of the SMSR is to conduct surveillance of MS, and to promote MS research that reflects the perspective of patients in Switzerland. More specifically, the goals are to estimate the number of patients with MS in Switzerland and the burden of disease for affected persons, their relatives, and society as a whole. The registry should also aid Swiss MS research by establishing a large database of well-documented MS patients and by enabling international collaborations.

In order to achieve these very different goals we will implement a flexible, layer-based design. The different layers are distinguished by the level of commitment by participants and the type and amount of data collected (i.e. from basic information about interested stakeholders in layer 1 to detailed phenotyping in layer 4). We will, in collaboration with social media and IT specialists, implement an IT infrastructure that will include a web platform through where interested stakeholders (patients, relatives, health care professionals, researchers, etc.) can get information about the registry and MS in general and interact.

The SMSR shall be operated in a spirit of cooperation and mutual trust. Regional, national and international collaborations will be central to the SMSR. As a large-scale project the SMSR will have a clearly defined governance structure for making transparent and agreed decisions. A steering committee will oversee the activities and make strategic decisions. To avoid competing interests, the core (data collection) of the SMSR will be financed by the Swiss MS-Society. Additional funding for specific sub-studies can and will be sought from other parties under the condition that those funds are unrestricted.

The expected benefits of the SMSR are manyfold. Through their active participation, patients and their proxies will experience better networks, more and better information on disease-related topics and advice regarding physical and mental health. Through translational research from the lab to the patients we expect that health care services and access to existing therapies and, as a consequence, prognosis and quality of life of patients will improve.
AKTIV LEBEN. 
GESTERN. HEUTE. MORGEN.

COPAXONE® Z: 
Glatirameracetat. 
I: Behandlung von Patienten mit CIS sowie zur Reduktion der Schubfrequenz und zur Verlangsamung des Fortschreitens von Behinderungsgrad, Intensität und Schwere der Krankheit bei remittierenden Formen der MS mit einem Score von ≤5 auf der EDSS. 
D: Injektion s.c. an wechselnden Injektionsstellen, 20 mg täglich. 
K: Hypersensibilität gegenüber Inhaltsstoffen, Schwangerschaft. 
A: Überempfindlichkeitsreaktionen, Nierenfunktion niereninsuffizienter Patienten regelmässig überprüfen. 
UAW: Schwindel, Depression, Angst, erhöhte Muskeltonus, Polyurie, Polydipsie, Dysphorie, Nausea, Obstipation, Diarrhoe, Rash, Schwitzen. 
ATC: L03AX13 P. COPAXONE® 28 Fertigspritzen (20mg/ml) [B]. 
INTERACTIVE WORKSHOPS

Clinical case management
These six interactive workshops are focused on the discussion of practical cases, with emphasis on aspects relevant to the daily management of MS patients.

14.15-15.00 Interactive Workshops A, B, C

Workshop A
Prof. Renaud Du Pasquier, Lausanne and Prof. Tobias Derfuss, Basel
Complications of current and emerging MS therapies

Workshop B
Prof. Till Sprenger, Basel and PD Dr. Cristina Granziera, Lausanne
MRI to monitor patients with brain and spinal cord white matter changes

Workshop C
Prof. Patrice Lalive, Geneva and PD Dr. Jens Kuhle, Basel
Can cerebrospinal fluid and blood biomarkers add to the management of MS?

15.00-15.20 Coffee Break

15.20-16.05 Interactive Workshops D, E, F

Workshop D
Dr. Claudio Gobbi, Lugano and Dr. Christian Kamm, Berne
New treatment options: pro and contra

Workshop E
Dr. Sven Schippling, Zurich and Prof. Peter Fuhr, Basel
OCT and electrophysiological techniques as tools to monitor MS: update

Workshop F
PD Dr. Michael Linnebank, Zurich and Dr. Claude Vaney, Montana
Increasing MS patients’ mobility: update

16.05 Farewell Apero
WORKSHOP A

COMPLICATIONS OF CURRENT AND EMERGING MS THERAPIES

Prof. Renaud du Pasquier, Lausanne
Lausanne University Hospital, Service of Neurology

Prof. Tobias Derfuss, Basel
University Hospital Basel, Departments of Neurology and Biomedicine

Recently, a number of new treatments have been registered for the treatment of relapsing-remitting MS and some more are in the late stage of clinical development. These treatments obviously open new possibilities but also present novel challenges.

This workshop will focus on the side effect profile of the new oral therapies Gilenya®, Aubagio®, and Tecfidera® and also discuss the emerging treatments with alemtuzumab and daclizumab that have completed phase III clinical development. The most prominent problem of all these therapies, to varying degrees - are infectious complications.

However, every drug has its own risk profile and therefore requires specific precautions. We will provide information on screening exams before start of treatment and safety monitoring during treatment. We will also touch the topic of switching and sequencing treatments that may also have an influence on the treatment emerging side effects.
WORKSHOP B

MRI TO MONITOR PATIENTS WITH BRAIN AND SPINAL CORD WHITE MATTER CHANGES

Prof. Till Sprenger, Basel
University Hospital Basel, Clinic and Polyclinic for Neurology

PD Dr. Cristina Granziera, Lausanne
Lausanne University Hospital, Department of Clinical Neurosciences

MRI is a key paraclinical tool for the diagnosis and differential diagnosis of demyelinating disorders. It is also becoming more and more important for monitoring the disease course of MS and related disorders as MRI is very sensitive to subclinical disease activity.

However, there is no general agreement on how and when to follow up MS patients with MRI. Several MRI metrics such as lesional Gadolinium enhancement, new or enhancing T2 lesions, lesion volume, black holes, brain atrophy, or subtle tissue changes as detected with MTR or DTI could potentially inform treatment decisions. As of yet, however, a consensus remains to be formed as to what levels of MRI activity should lead to treatment change or at least a consideration of change. Furthermore, some of the MRI metrics are difficult to standardise and implement in clinical practice.

In this workshop, we will discuss conventional and advanced neuroimaging metrics that could guide treatment decisions.
WORKSHOP C

CAN CEREBROSPINAL FLUID AND BLOOD BIOMARKERS ADD TO THE MANAGEMENT OF MS?

Prof. Patrice Lalive, Geneva
Geneva University Hospital, Service of Neurology

PD Dr. Jens Kuhle, Basel
University Hospital Basel, Department of Neurology

Despite the fact that diagnosis of multiple sclerosis (MS) is based on clinical suspicion and typical magnetic resonance imaging findings, cerebrospinal fluid (CSF) analysis remains important in order to support the diagnosis and exclude alternative diseases. This is especially important since our understanding of the defining disease pathogenesis remains incomplete and no specific markers are currently available to confirm the disease. According to the recent diagnostic criteria, MS requires establishing that «there is no better explanation for the clinical picture».

Using optimised, standardised methodology, preferentially protein separation by isoelectric focusing followed by immunoblotting, more than 95% of patients with MS have CSF oligoclonal bands (OCB) of IgG class not detectable in serum, thereby providing powerful evidence for the diagnosis of MS. Although the clinical picture as well as findings from MRI are essential for an MS diagnosis, this should be re-evaluated in CSF OCB-negative patients, keeping in mind the many disease entities imitating MS.

We will review the stepwise approach to CSF analysis and present some patient cases illustrating the relevance of CSF analysis. The assessment classically includes blood tests to exclude infections and systemic autoimmune diseases. Here, we propose to make a short, non-exhaustive review of viral and immunological tests that can be performed in the assessment of a patient with suspicion of MS. In addition we will discuss the utility of new biological tests, including vitamin D, EBV serology, oligoclonal IgM bands, new antibodies like KIR 4.1, chitinase 3 like 1 and neurofilaments that may help to monitor therapeutic decision in MS in future.
WORKSHOP D

NEW TREATMENT OPTIONS: PRO AND CONTRA

Dr. Claudio Gobbi, Lugano
Regional Hospital of Lugano, Neurocenter of Southern Switzerland

Dr. Christian Kamm, Berne
Berne University Hospital, Department of Neurology

Considering the growing number of therapeutic agents in multiple sclerosis with different modes of action, application, efficacy, tolerability and safety profiles, the future challenge will be to choosing the most efficacious, safe and tolerable drug for the individual patient.

This workshop aims at discussing the current treatment options focusing on the «pros» and «cons» of «old» vs. «new» drugs and at proposing a treatment algorithm. This will finally be illustrated and discussed with the aid of practical case reports.
WORKSHOP E

OCT AND ELECTROPHYSIOLOGICAL TECHNIQUES AS TOOLS TO MONITOR MS: UPDATE

Dr. Sven Schippling, Zurich
University Hospital Zurich, Department of Neurology

Prof. Peter Fuhr, Basel
University Hospital Basel, Department of Neurology

Until about 20 years ago, the main role of electrophysiology in the assessment of MS was diagnostic. With the broad accessibility of MRI, evoked potentials have largely lost their diagnostic importance. However, research into and clinical application of disease modifying treatment have led to an increased interest in prognosis and long term monitoring, and in this domain functional assessment including characterization of neurophysiological capacity is important.

A reliable and valid way to characterize some important aspects of neurophysiological capacity consists in the recording of evoked potentials (EP). Since MS may afflict any central nervous connection, the sensitivity of EP recording increases with the number of modalities and the length of the tested tracts. Therefore, multimodal (visual, somato-sensory, motor, and possibly other) EP are used to assess electrophysiologically a patient. Multimodal EP yield numerical data and have been demonstrated to correlate with clinical findings in cross-sectional and longitudinal comparison. Moreover, probably in part due to the sensitivity of EP to subclinical lesions, they also correlate with prognosis over two and up to 20 years.

While validity of multimodal EP to characterize groups of patients has been shown repeatedly, their applicability for counselling individual patients is still a goal of current research. Nevertheless, they can be helpful to define prognostic groups of patients in MS and measure their course, and therefore, may contribute to acceleration of clinical trials.
**WORKSHOP F**

**INCREASING MS PATIENTS’ MOBILITY: UPDATE**

PD Dr. Michael Linnebank, Zurich
University Hospital Zurich, Department of Neurology

Update: Detailed effects of prolonged-release fampridine on disability of patients with multiple sclerosis (FAMPKIN and FAMPKIN-EXT)

**Background and Objective:** Recent Phase III clinical trials demonstrated the beneficial effects of fampridine (4-aminopyridine) on gait velocity in a subset of patients with MS. Fampridine blocks voltage-gated potassium channels leading to enhanced signal conduction in demyelinated nerve fibers. The FAMPKIN core study (http://clinicaltrials.gov) aimed to characterise the effects of prolonged-release (PR) fampridine on different modalities of ambulatory function including muscle strength, stability and coordination. The extension study (Fampkin-EXT) followed the core study and aimed to assess the long-term effects and tolerability of fampridine in some of the patients who completed the core study (n=53 patients).

**Methods:** FAMPKIN designed as a phase II, double-blind, randomized and placebo-controlled cross-over study assessed gait function during two double-blind fampridine or placebo treatment phases (each 6 weeks). Walking was investigated using different clinical tests, questionnaires and detailed kinematic-kinetic gait analysis. 55 patients with relapsing-remitting, primary- and secondary-progressive MS completed the study (34 women, 21 men; age 48.4 +/- 9.7 years; median EDSS = 4.5). 53 patients previously enrolled in the FAMPKIN study were included in the extension study (33 women, 20 men; age: 49.7 +/- 9.2 years; EDSS 5.3 +/- 1.2) and treated with prolonged-release fampridine (10mg twice daily; open-label).

**Results:** In the FAMPKIN core study, patients were classified into responder and non-responder according to the criteria used by Goodman et al. (Lancet, 2009). 31% (n=17) of all participants were fampridine-responder, 5% (n=3) were placebo-responder, and 9% (n=5) met the responder criteria in both phases. During fampridine treatment, gait velocity (timed-25-foot walk) was increased by 5.8% in the total population (n=55), 12.3% in the pure fampridine-responder subgroup (n=17), and by 3.3% in non-responders (n=29) compared to the placebo treatment. Walking endurance (6-minute walk test) was improved by 8.5% in the total population, 13.5% in responders, and 6.1% in non-responders. Stationary and dynamic measures of stability did not reveal significant changes induced by fampridine. A portable accelerometer device (actimeter) measured physical activity during 14 days in each double-blind treatment period. Physical activity during daily life was significantly increased during the fampridine treatment phase compared to the placebo phase in the pure fampridine-responder subgroup, but not the other subgroups.
Gait profiles consisting of multiple kinematic and kinetic parameters demonstrated heterogeneous, fampridine-induced gait modifications. The differential changes in the gait pattern among patients most probably reflect individual improvements of the gait pattern depending on the specific deficits of each patient.

The results of the first extension year confirm the data of the core study: patients got slower (timed-25-foot walk: -15%) when fampridine was stopped for 14 days after continuous treatment for 11.5 months. Walking endurance (6-minute walk test) was reduced by 9% after the 14 days washout phase. These changes in walking function were self-perceived by the patients, as demonstrated by the 12-item MS walking scale (12MSWS).

**Conclusion:** The present studies demonstrate beneficial effects of fampridine on different aspects of locomotion and indicates the clinical relevance of this treatment for a proportion of patients with MS. Fampridine is a well tolerated symptomatic treatment for MS patients and can exert its beneficial effects over long periods of application.
WORKSHOP F

INCREASING MS PATIENTS’ MOBILITY: UPDATE

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Assessing quickly and graphically the Mobility in MS and other neurological Disease with the new Iphone App Sagas 10

Objectives: Assuming that this tool could also be used for other neurological diseases where walking and hand function is impaired, we set out to examine the validity and the responsiveness of SaGAS 10 in neurological patients attending a rehabilitation facility. Furthermore, we evaluated whether the 25 feet walking has a high correlation with the 2-minute walking test in patients with slow walking speed.

Methods: 646 consecutive patients with different neurological diseases (MS 296, stroke 152, Parkinson 21, neuromuscular disorders 42, trauma 42, others 93) were assessed at the beginning and at the end of their rehabilitation stay using the FIM (Functional Independence Measure), the RMI (Rivermead Mobility Index, the 2-minute timed walking distance at maximum speed (2MWD) and the 3 measures composing SaGAS 10 (the 25 feet timed walk at fast speed with a flying start (T25FW) and the nine-hole peg test (9-HPT) for each hand separately). Construct validity was assessed with correlations between FIM, RMI and the SaGAS 10, where correlations above 0.7 were hypothesized. Responsiveness was assessed by a receiver operating characteristic curves (ROCs) analyses comparing changes in SaGAS 10 with minimal clinically important changes in the RMI. An area under the curve value (AUC) of at least 0.7 was considered as appropriate.

Results: The correlation of the SaGAS 10 with the Rivermead Mobility Index is above 0.7 in all of the neurological diagnostic groups; the highest correlation coefficient was found in patients with stroke: 0.76. The correlation of the SaGAS 10 with the FIM was over 0.7 for stroke and MS. The responsiveness was acceptable with AUCs of 0.72 for stroke and values over 0.7 for all groups, with the exception of MS. The effect-sizes were moderate to high, especially for stroke with Cohen's d values of 0.48. The correlation between the 25 feet test and the 2 minutes walking test was 0.63 for those walking slower than 0.96 m/s; and 0.64 for those walking faster than 0.96 m/s.

Conclusions: These results indicate that SaGAS 10 is valid and sensitive to changes over time and that it could be a useful measure not only for patients with MS, but also for patients with other neurological diseases. Our results indicate that for slow walkers the 25 feet walking test might be a good alternative for the 2-minutes walking test.
WE THANK YOU FOR YOUR PARTICIPATION AND WISH YOU A SAFE JOURNEY HOME.

SEE YOU NEXT YEAR AT THE 18TH STATE OF THE ART SYMPOSIUM.

BEST REGARDS
SWISS MULTIPLE SCLEROSIS SOCIETY