7th Annual Meeting of the Americas Committee for Treatment and Research in Multiple Sclerosis

18th Congress of the European Committee for Treatment and Research in Multiple Sclerosis

Wednesday, September 18 through Saturday, September 21, 2002

Baltimore Marriott Waterfront Hotel
700 Aliceanna Street
Baltimore, Maryland, USA

Final Program, Abstract Listing and Meeting Information
Acknowledgement of Sponsors

The ACTRIMS-ECTRIMS 2002 Steering Committee acknowledges the generous support of our Gold Sponsors:

The Steering Committee also acknowledges the support of Wyeth Pharmaceuticals

This program is offered in collaboration with the National Multiple Sclerosis Society.
Welcome Letter

Dear Colleagues and Friends,

We cordially welcome you to Baltimore, Maryland, and ACTRIMS-ECTRIMS 2002, the second joint meeting of the Americas and European Committees for Research and Treatment in Multiple Sclerosis. This is an exciting time in MS research and clinical care. Decades of basic investigation have resulted in greatly improved understanding of MS, which is now being translated into proven therapies. However, we—and the patients we serve—cannot be satisfied with the current state of our knowledge and treatments. We hope that the information provided during this important meeting will further advance the goal of finally conquering MS and its resulting disability.

Our three key areas of research highlighted at the conference are:

- Inflammation, Demyelination, and Axonal Loss: Pathological and MRI Perspectives
- Neuroprotection
- Methodological Issues in Clinical Trials

In addition to the official program, we are pleased to offer two pre-conference symposia sponsored by groups long associated with the treatment of MS and five satellite symposia supported by our gold sponsors.

We extend our appreciation to our Program Committee, chaired by Drs. Suhayl Dhib-Jalbut and Paul O’Connor, for their tireless effort in guiding and shaping the content of this comprehensive meeting.

We hope that you will join us for each of the social activities, planned by our Local Arrangements Committee chaired by Drs. Chris Bever and Peter Calabresi, and that you will allow some time to explore the harbor city of Baltimore, with its array of shopping destinations, historical sites, museums and galleries.

We look forward to your active participation in this very interesting program. Thank you for joining us.

Dr. Kenneth Johnson
Chair, ACTRIMS

Dr. Alan Thompson
President, ECTRIMS
Committees

Steering Committee
Kenneth Johnson (USA)
Chair, ACTRIMS
Alan Thompson (UK)
President, ECTRIMS
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Peter Calabresi, Co-Chair
Lee Koski
Mary Rose
Peggy Allen

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Co-Chair
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Roland Martin (USA)
Chris Polman (The Netherlands)
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Alan Thompson (UK)

National MS Society Organizing Committee
Debra Entin, Chair
Leslie DiLeo
Abe Eastwood
Nancy Holland
Dinah Martinez
Diann Rohde
Bill Rosen
Kristin Summers
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Response.

Proven results for people with relapsing MS.

MS affects different people in different ways each and every day. Rebif® has been proven to provide patients with the following treatment benefits in the main measures of disease activity:

• Reduces MRI lesion activity
• Reduces frequency of relapse
• Delays progression of disability

*The exact relationship between MRI findings and the clinical status of patients is unknown.

Rebif is the market leader outside the US. Rebif is available in more than 70 countries.

Please see brief summary directly following MS LifeLines™ ad

References:
Results. Rebif.

Adverse reactions at 24 weeks were generally similar despite higher, more frequent, subcutaneous dosing with Rebif\textsuperscript{2}. Exceptions included injection-site disorders, hepatic function disorders, and leukopenia\textsuperscript{2,3}.

Rebif\textsuperscript{®} (interferon beta-1a) is indicated for the treatment of patients with relapsing multiple sclerosis to decrease the frequency of clinical exacerbations and delay the accumulation of physical disability. Rebif should be used with caution in patients with depression, pre-existing seizure disorders, and liver problems.

Avonex\textsuperscript{®} is a registered trademark of Biogen, Inc.
Convenience
Independence for Patients

- Rebif® is the first and only interferon available in ready-to-use, prefilled syringes
- Rebif is administered subcutaneously
- Rebiject™ injection device ensures proper injection technique
- Injection training with a licensed nurse
- Travel Kit including prepaid phonecard makes travel easier
- Patients are just one toll-free phone call away from professionals who can offer immediate assistance

For more information on the benefits of Rebif therapy, please visit www.rebif.com or call 1-877-44-Rebif (447-3243)
Prescribe Your Patients Rebif® and More.

MS LifeLines™

Response
- Dedicated team of trained Customer Support Specialists available Monday through Friday from 8 AM to 8 PM EST
- English and Spanish language support available

Results
- Reimbursement support
- Pharmacy coordination
- Injection training
- Ongoing nurse follow-up

Rebif®
- Complimentary Travel Kit
- Education support materials
- Rebiject™ injection device

Patients with MS are just one toll-free phone call away from professionals who can offer immediate assistance.

Patients can call today
Phone: 1-877-44-Rebif (1-877-447-3243)
Fax: 1-866-22-Rebif (1-866-227-3243)
or visit our websites at:
www.MSLifeLines.com
www.rebif.com

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BRIEF SUMMARY

Please see package insert for full prescribing information.

Indications and Usage
Rebif® (interferon beta-1a) is indicated for the treatment of patients with relapsing forms of multiple sclerosis to decrease the frequency of clinical exacerbations and delay the accumulation of physical disability. Efficacy of Rebif® in chronic progressive multiple sclerosis has not been established.

Clinical Studies
Two multicenter studies evaluated the safety and efficacy of Rebif® in patients with relapsing-remitting multiple sclerosis (see table). Study B demonstrated that Rebif® significantly reduced the number of relapses per patient compared to placebo at 2 years. Study 2 was a comparative trial comparing the recommended dosage of Rebif® and Avonex®. The results of this trial demonstrated that patients treated with Rebif® 44 mcg at 3 days/wk had a 24% reduction in the treatment period than patients treated with Avonex® 30 mcg im qds. Adverse reactions were generally similar between the two treatment groups. Exceptions included injection site disorders (60% of patients on Rebif® vs. 24% of patients on Avonex®), hepatic function disorders (14% on Rebif® vs. 7% on Avonex®), leukopenia (3% on Rebif® vs. 1% on Avonex®), which were observed with greater frequency in the Rebif® group compared to the Avonex® group.

Contraindications
Rebif® (interferon beta-1a) is contraindicated in patients with a history of hypersensitivity to natural or recombinant interferon, human albumin, murine IL-6, sodium azide, or Water for Injection USP.

WARNINGS
Rebif® (interferon beta-1a) should be used with caution in patients with diabetes, a condition that is common in people with multiple sclerosis. Depression, suicidal ideation, and suicide attempts have been reported to occur with increased frequency in patients receiving interferon products, including Rebif®. Patients should be advised to report immediately any symptoms of depression and suicidal ideation to their physician. If a patient develops depression, cessation of treatment with Rebif® should be considered.

A case of fulminant hepatic failure requiring liver transplantation in a patient who initiated Rebif® therapy while taking another potentially hepatotoxic medication has been reported from a non-US, post-marketing study. In patients with pre-existing liver disease, presently an ongoing study, has been reported as a rare complication of Rebif® use. Asymptomatic elevation of hepatic transaminases (particularly SGPT) is common with interferon therapy (see ADVERSE REACTIONS). Rebif® should be initiated with caution in patients with active liver disease, alcohol abuse, increased serum SGPT > 2.5 times ULN, or a history of significant liver disease. Dose reduction should be considered if SGPT rises above 5 times the upper limit of normal. The dose may be gradually re-escalated when enzyme levels have normalized.

Treatment with Rebif® should be stopped if jaundice or other clinical symptoms of liver dysfunction appear.

Anaphylaxis has been reported as a rare complication of Rebif® use. Other allergic reactions have included skin rash and urticaria, and have ranged from mild to severe without a clear relationship to dose or duration of exposure. Several allergic reactions, some severe, have occurred after prolonged use.

PRECAUTIONS
General:
Caution should be exercised when administering Rebif® to patients with pre-existing seizure disorder. Seizures have been associated with the use of beta interferons. A relationship between occurrence of seizures and the use of Rebif® has not been established. Leukopenia and or worsening thyroid abnormalities have developed in some patients treated with Rebif®. Regular monitoring for these conditions is recommended.

Information for Patients
All patients should be instructed to read the Rebif® Medication Guide supplied to them. Patients should be counseled on the importance of adhering to the recommended schedule of administration without medication breaks. Patients should be informed of the most common and the most severe adverse reactions associated with the use of Rebif®. Patients should be advised of the symptoms associated with these conditions, and to report them to their physician.

Female patients should be cautioned about the abortifacient potential of Rebif®.

Patients should be instructed in the use of aseptic technique when administering Rebif®. Appropriate instruction for self-injection or injection by another person should be provided, including careful review of the Rebif® Medication Guide. If a patient is to self-administer Rebif®, the physical and cognitive ability of that patient to self-administer and properly dispose of syringes should be assessed. The injection should be performed under the supervision of an appropriately qualified health care professional. Patients should be advised of the importance of rotating sites of injection with each dose, to minimize the likelihood of severe injection site reactions or necrosis.

Laboratory Tests:
In addition to those laboratory tests normally required for monitoring patients with multiple sclerosis, blood cell counts and liver function tests are recommended at regular intervals (1, 3, and 6 months) following treatment with Rebif® therapy and then periodically thereafter, in the absence of clinical symptoms. Thyroid function tests are recommended every 6 months in patients with a history of thyroid dysfunction or as clinically indicated. Patients with myelosuppression may require more intensive monitoring of complete blood cell counts, with differential and platelet counts.

Drug Interactions:
Drug interaction studies have not been conducted with Rebif®. Due to its potential to cause neutropenia and lymphopenia, proper monitoring of patients is required if Rebif® is given in combination with myelosuppressive agents.

Carcinogenesis, Mutagenesis, Impairment of Fertility:
No carcinogenicity data for Rebif® are available in animals or humans. Rebif® was not mutagenic when tested in the Ames bacterial test and in an in vitro chromosomal assay. In human lymphocytes it has not been shown to induce chromosomal aberrations or mitotic nasalisation. No studies have been conducted to evaluate the effects of Rebif® on fertility in humans. In studies in normally cycling female cynomolgus monkeys given daily sc injections of Rebif® for 6 months at doses of up to 9 times the maximum human dose, based on body surface area, no effects were observed on either menstrual cycling or serum estradiol levels. The validity of extrapolating dosages used in animal studies to human dosages is not clear. On the other hand, the results of Rebif® in monkeys had no demonstrable adverse effects on sperm count, motility, morphology, or function.

Pregnancy Category C: Rebif® treatment has been associated with significant increases in embryotoxic or abortifacient effects in cynomolgus monkeys administered doses approximately 2 times the cumulative weekly human dose (based on either body weight or surface area) either during the period of organogenesis (gestation day 21-60 or later in pregnancy). There were no fetal malformations or other evidence of teratogenicity noted in these studies. These effects are considered to be related to the abortifacient activity of Rebif® due to the absence of other type I interferons. There are no adequate and well-controlled studies of Rebif® in pregnant women. However, in Studies 1 and 2, there were 2 spontaneous abortions observed and 5 fetuses examined in animals where 27 women in the Rebif® group were pregnant and 13 became pregnant while taking Rebif®; she should be informed about the potential hazards to the fetus and discontinuation of Rebif® should be considered.

Nursing Mothers: It is not known whether Rebif® is excreted in human milk.

Pediatric Use: The safety and effectiveness of Rebif® in pediatric patients have not been established.

Gastric Ulcer: Clinical studies of Rebif® did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently than younger subjects.

ADVERSE REACTIONS
The most frequently reported serious adverse reactions with Rebif® were psychiatric disorders including depression and suicidal ideation or attempt (see WARNINGS). The incidence of depression of any severity in the Rebif®-treated group and placebo-treated group was approximately 25%. The most commonly reported adverse reactions were injection site disorders, influenza-like symptoms (headache, fatigue, fever, rigors, myalgia), abdominal pain, depression, elevation of liver enzymes and hematological abnormalities. The most frequently reported adverse reactions resulting in clinical intervention (e.g., discontinuation of Rebif®, adjustment in dosage, or the need for concomitant medication to treat an adverse reaction symptom) were injection site disorders, influenza-like symptoms, depression and elevation of liver enzymes (see WARNINGS). Injection site necrosis was rare.

The safety of Rebif® (22 mcg and 44 mcg vs. placebo) was studied in 560 patients with RMS who were treated for 24 months (Study 1). Table 1 enumerates adverse events and laboratory abnormalities that occurred in the placebo group that occurred at least 2% more in either Rebif®-treated group than observed in the placebo group.

Immunogenicity: As with all therapeutic proteins, there is a potential for immunogenicity. Serum NAB were detected in 45% (24%) of Rebif®-treated patients at the 44 mcg dose level at one or more times during Study 1. The clinical significance of the presence of NAB to Rebif® was not determined. However, in 1 patient who received 180 mcg of Rebif® over 4 weeks and continued to receive 44 mcg x 2 wk, no major adverse events or laboratory changes were noted. The incidence of antibodies to other products may be misleading.

DOSAGE AND ADMINISTRATION
The recommended dosage of Rebif® is 44 mcg injected sc at 3 days/wk. Rebif® should be administered, if possible, at the same time (preferably in the late afternoon or evening) on the same 3 days (e.g., Monday, Wednesday, and Friday) at least 48 hours apart each week. Generally, patients should be started at 8.8 mcg x 2 sc and increased to 22 mcg x 2 sc at 44 mcg x 2 wks (see Table 2). A Rebif® "Starter Pack" containing 22 mcg syringes is available for use in titrating Rebif®. Once daily 3 days/wk or 2 days/wk (see Table 2). For more information on how to start serum neutropenia or elevated liver function tests may necessitate dose reductions of 20-50% until toxicity is resolved. Rebif® is intended for use under the guidance and supervision of a physician. It is recommended that the following be done:...
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<thead>
<tr>
<th><strong>Wednesday September 18</strong></th>
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<td>Grand Ballroom Level</td>
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<td>2:00 pm–4:30 pm</td>
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<td>12:00 pm–1:00 pm</td>
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<td>Satellite Symposium III</td>
<td>Satellite Symposium III</td>
<td>Satellite Symposium V</td>
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<td>Social Event</td>
<td>Committee Reception</td>
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<td>B&amp;O Railroad Museum</td>
<td>(by invitation)</td>
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<tr>
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<td>in Baltimore</td>
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<td>7:30 pm–10:30 pm</td>
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<td>Baltimore Museum of Industry</td>
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**Exhibits and Poster Displays**
*Harborside and Grand Ballroom Levels*
- **Wednesday**: 1:00 pm–5:00 pm
- **Thursday and Friday**: 8:00 am–5:00 pm
Biogen and Elan – pioneers in adhesion molecule biotechnology
General Information

Attire
Since meeting room temperature and personal comfort levels vary, it is recommended that you bring a sweater or jacket to the conference activities. Attire for meetings and social events is business casual.

Badges
Attendees will be required to wear their delegate badge at all times to access the exhibition area, the conference rooms and the posters area. Colored ribbons denote the following:

- Committee Members: WHITE
- Exhibitors: ORANGE
- Poster Presenters: LIGHT BLUE
- Press: PINK
- Speakers: BLUE
- Staff: RED
- Volunteers: GREEN

Baltimore
For information regarding airports, activities, ground transportation, etc., consult the Baltimore Area Convention and Visitors Association website at www.baltimore.org.

Child Care Information
A variety of child care options are available in Baltimore. You may wish to check with the concierge at your hotel upon arrival.

Contact Information
ACTRIMS-ECTRIMS 2002
c/o NMSS
733 Third Avenue 6th Floor
New York, NY 10017 USA
Phone: 212-476-0465
Fax: 212-661-9735
E-mail: ae2002@nmss.org
www.actrimsectrims2002.nmss.org

E-Mail Stations
Complimentary e-mail stations and printers will be available in the Harborside Level Foyer. Please limit your use to 15 minutes. This service is available to registered participants only. Thank you for your cooperation.

Language
English is the official language of the conference. No simultaneous translation is available.

Meals
The registration fee for conference participants includes continental breakfast, coffee breaks, lunches during the conference, and the evening social events organized by the Steering Committee.

Mobile Phones
The Steering Committee request that attendees turn cellular phones and pagers to vibrate upon entering all exhibit and social functions.

No Smoking
For the health and comfort of everyone, smoking is prohibited at any meeting function. This includes all scientific activities, exhibits and social functions.

Optional Social Tours
For half-day and whole-day excursions to sites in and around Baltimore, please consult the concierge at your hotel.

Photography
Flash picture taking is not allowed during the scientific activities or in the exhibit area.

Recording of Programs
Audio and videotaping are not allowed in the meeting rooms, exhibit area, or at social functions.

Special Needs
If you have a special need that requires an accommodation, please stop by the registration desk and speak with an organizing staff member.

Weather
September temperatures range from 65 F (18 C) to 80 F (27 C).
Floor Diagrams

Level 4

Level 3
## Transportation Schedule

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<td>7:00 am–2:00 pm</td>
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<tr>
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<td><strong>B&amp;O Party</strong></td>
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<td><strong>Committee Reception</strong></td>
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<td><strong>8:30 pm–11:00 pm</strong></td>
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### A
Continuous shuttle service between auxiliary hotels and Baltimore Marriott Waterfront Hotel

### B
Transportation from Baltimore Marriott Waterfront Hotel to the National Aquarium in Baltimore for those unable/reluctant to walk

### C
Transportation from the National Aquarium in Baltimore to auxiliary hotels and to Baltimore Marriott Waterfront Hotel

### D
Transportation from Baltimore Marriott Waterfront Hotel to B&O Railroad Museum

### E
Transportation from B&O Railroad Museum to auxiliary hotels and to Baltimore Marriott Waterfront Hotel

### F
Round-trip water taxi service from Baltimore Marriott Waterfront Hotel to the Baltimore Museum of Industry
She works hard
So does her treatment

AVONEX® in relapsing forms of MS

- **Strong against both disability and relapses**
  - reduces the progression to sustained disability by 37% and lowers relapse rates ¹

- **Strong against inflammation**
  - 91% reduction in T2 lesion volume and 89% reduction in gadolinium-enhanced lesions²,³

- **Strong against atrophy**
  - decreased brain atrophy by 55% in year 2 of a clinical trial⁴

- **Strong with patients**
  - AVONEX® is the #1 prescribed MS therapy and delivers 95% patient satisfaction⁵,⁶

- **The difference is in the delivery**
  - IM administration keeps effective amounts of AVONEX® in the body longer than the SC route⁷

The most common side effects associated with AVONEX® treatment are flu-like symptoms, muscle ache (myalgia), fever, and chills. Other common side effects seen, but not statistically different from placebo, were headache (AVONEX®: 67%, placebo: 57%), pain (AVONEX®: 24%, placebo: 20%), and asthenia (AVONEX®: 21%, placebo: 13%).

AVONEX® should be used with caution in patients with depression and in patients with seizure disorders. AVONEX® should not be used by pregnant women. Patients with cardiac disease should be closely monitored. Routine periodic blood chemistry and hematology tests are recommended during treatment with AVONEX®.

The exact relationship between MRI findings and the clinical status of patients is unknown. Changes in lesion area often do not correlate with changes in disability progression.
AVONEX® (Interferon beta-1a)

For more detailed information, consult full prescribing information.

A brief summary follows.

INDICATIONS AND USAGE
AVONEX® (Interferon beta-1a) is indicated for the treatment of relapsing forms of multiple sclerosis to slow the accumulation of physical disability and decrease the frequency of clinical exacerbations. Safety and efficacy in patients with progressive multiple sclerosis have not been evaluated.

CONTRAINDICATIONS
AVONEX® (Interferon beta-1a) is contraindicated in patients with a history of hypersensitivity to natural or recombinant interferon beta, human albumin, or any other component of the formulation.

WARNINGS
AVONEX® (Interferon beta-1a) should be used with caution in patients with depression. Depression and suicide have been reported to occur in patients receiving interferon compounds. Depression and suicidal ideation are known to occur at an increased frequency in the multiple sclerosis population. A relationship between occurrence of depression and/or suicidal ideation and the use of AVONEX® has not been established. An equal incidence of depression was seen in the placebo-treated and AVONEX®-treated patients in the placebo-controlled multiple sclerosis study. Patients treated with AVONEX® should be advised to report immediately any symptoms of depression and/or suicidal ideation to their prescribing physicians. If a patient develops depression, cessation of AVONEX® therapy should be considered.

PRECAUTIONS
General
Caution should be exercised when administering AVONEX® (Interferon beta-1a) to patients with pre-existing seizure disorder. In the placebo-controlled study, 4 patients receiving AVONEX® experienced seizures, while no seizures occurred in the placebo group. Three of these 4 patients had no prior history of seizure. It is not known whether these seizures were related to the effects of multiple sclerosis alone, to AVONEX®, or to a combination of both. For patients with no prior history of seizure who develop seizures during therapy with AVONEX®, an etiologic basis should be established and appropriate anti-convulsant therapy instituted prior to considering resumption of AVONEX® treatment. The effect of AVONEX® administration on the medical management of patients with seizure disorders is unknown.

Patients with cardiac disease, such as angina, congestive heart failure, or arrhythmia, should be closely monitored for worsening of their clinical condition during initiation and continued treatment with AVONEX®. While AVONEX® does not have any known direct-acting cardiac toxicity, during the post-marketing period infrequent cases of congestive heart failure, cardiomyopathy, and cardiomyopathy with congestive heart failure have been observed in patients without known predisposition to these events or other known etiologies. In rare cases, these events have been temporally related to the administration of AVONEX®. In rare cases, these events have recurred upon rechallenge in patients with known predisposition.

Information to Patients
Patients should be informed of the most common adverse events associated with AVONEX® administration, including symptoms associated with flu syndrome (see Adverse Reactions section). Symptoms of flu syndrome are most prominent at the initiation of AVONEX® administration, including symptoms associated with flu syndrome (see Adverse Reactions section). Symptoms of flu syndrome are most prominent at the initiation of therapy and decrease in frequency with continued treatment. In the placebo-controlled study, patients were instructed to take 650 mg acetaminophen immediately prior to injection and for an additional 24 hours after each injection to mitigate acute symptoms associated with AVONEX® administration. Patients should be cautioned to report depression or suicidal ideation (see Warnings). Patients should be advised about the abortifacient potential of interferon beta (see Pregnancy – Teratogenic Effects).

When a physician determines that AVONEX® can be used outside of the physician’s office, directions who will be administering AVONEX® should be given in the instruction in reconstitution and injection, including the review of the injection procedures (see full prescribing information). If a patient is self-administering, the physical ability of that patient to self-inject intramuscularly should be assessed. The first injection should be performed under the supervision of a qualified health care professional. A puncture-resistant container for disposal of needles and syringes should be used. Patients should be instructed in the technique and importance of proper syringe and needle disposal and be cautioned against reuse of these items.

Laboratory Tests
In addition to those laboratory tests normally required for monitoring patients with multiple sclerosis, complete blood and differential white blood cell counts, platelet counts, and blood chemistry, including liver function tests, are recommended during AVONEX® (Interferon beta-1a) therapy. During the placebo-controlled study, these tests were performed at least every 6 months. There were no significant differences between the placebo and AVONEX® groups in the incidence of liver enzyme elevation, leukopenia, or thrombocytopenia. However, these are known to be dose-related laboratory abnormalities associated with the use of interferons. Patients with myelosuppression may require responsible monitoring of complete blood cell counts, with differential and platelet counts.

Drug Interactions
No formal drug interaction studies have been conducted with AVONEX® (Interferon beta-1a). In the placebo-controlled study, corticosteroids or ACTH were administered for treatment of exacerbations in some patients concurrently receiving AVONEX®. In addition, some patients receiving AVONEX® were also treated with anti-depressant therapy and/or other neuro-receptive therapy. No unexpected adverse effects were associated with these concomitant therapies.

Other Interferences
Other interferons have been noted to reduce cytochrome P-450 oxidase-mediated drug metabolism. Formal hepatic drug metabolism studies with AVONEX® in humans have not been conducted. Hepatic microsomes isolated from AVONEX®-treated rhesus monkeys showed no influence of AVONEX® on hepatic P-450 enzyme metabolism activities.

As with all interferon products, proper monitoring of patients is required if AVONEX® is given in combination with myelosuppressive agents.

Table. Adverse Events and Selected Laboratory Abnormalities in the Placebo-Controlled Study

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Placebo (N = 143)</th>
<th>AVONEX® (N = 158)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body as a Whole</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td>27%</td>
<td>9%</td>
</tr>
<tr>
<td>Focal symptoms (otherwise unspecified)*</td>
<td>40%</td>
<td>61%</td>
</tr>
<tr>
<td>Pain</td>
<td>20%</td>
<td>24%</td>
</tr>
<tr>
<td>Asthenia</td>
<td>13%</td>
<td>21%</td>
</tr>
<tr>
<td>Chills*</td>
<td>7%</td>
<td>21%</td>
</tr>
<tr>
<td>Fever</td>
<td>6%</td>
<td>11%</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>6%</td>
<td>9%</td>
</tr>
<tr>
<td>Chest pain</td>
<td>4%</td>
<td>6%</td>
</tr>
<tr>
<td>Injection site reaction</td>
<td>4%</td>
<td>3%</td>
</tr>
<tr>
<td>Malaise</td>
<td>3%</td>
<td>4%</td>
</tr>
<tr>
<td>Injection site inflammation</td>
<td>0%</td>
<td>3%</td>
</tr>
<tr>
<td>Hypersensitivity reaction</td>
<td>0%</td>
<td>3%</td>
</tr>
<tr>
<td>Ovarian cyst</td>
<td>0%</td>
<td>3%</td>
</tr>
</tbody>
</table>
**Table. Adverse Events and Selected Laboratory Abnormalities in the Placebo-Controlled Study (continued)**

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Placebo (N = 143)</th>
<th>AVONEX® (N = 158)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiovascular System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Syncope</td>
<td>2%</td>
<td>4%</td>
</tr>
<tr>
<td>Vasodilation</td>
<td>1%</td>
<td>4%</td>
</tr>
<tr>
<td><strong>Digestive System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>23%</td>
<td>33%</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>10%</td>
<td>16%</td>
</tr>
<tr>
<td>Dyspepsia</td>
<td>7%</td>
<td>11%</td>
</tr>
<tr>
<td><strong>Hemic and Lymphatic System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aemia*</td>
<td>3%</td>
<td>8%</td>
</tr>
<tr>
<td>Eosinophils ≥ 10%</td>
<td>4%</td>
<td>5%</td>
</tr>
<tr>
<td>HCT (%) ≤ 32 (females) or ≤ 37 (males)</td>
<td>1%</td>
<td>3%</td>
</tr>
<tr>
<td><strong>Musculoskeletal System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Metabolic and Nutritional Disorders</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SGOT ≥ 3 x ULN</td>
<td>1%</td>
<td>3%</td>
</tr>
<tr>
<td><strong>Nervous System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle ache*</td>
<td>15%</td>
<td>34%</td>
</tr>
<tr>
<td>Arthritis</td>
<td>5%</td>
<td>9%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Respiratory System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>28%</td>
<td>31%</td>
</tr>
<tr>
<td>Sinusitis</td>
<td>17%</td>
<td>18%</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>3%</td>
<td>6%</td>
</tr>
<tr>
<td><strong>Skin and Appendages</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urticaria</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>Alopecia</td>
<td>1%</td>
<td>4%</td>
</tr>
<tr>
<td></td>
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<td></td>
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<tr>
<td><strong>Special Senses</strong></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Urinary Tract</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary incontinence</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>Other genitourinary disorder</td>
<td>3%</td>
<td>6%</td>
</tr>
</tbody>
</table>

*Significantly associated with AVONEX® treatment (p ≤ 0.05).

AVONEX® (interferon beta-1a) has also been evaluated in 290 patients with illnesses other than multiple sclerosis. The majority of these patients were enrolled in studies to evaluate AVONEX® therapy of chronic viral hepatitis B and C, in which the doses studied ranged from 15 mcg to 75 mcg, given SC, 3 times a week, for up to 6 months. The incidence of adverse events in these studies was generally seen at a frequency similar to that seen in the placebo-controlled multiple sclerosis study. In these non-multiple sclerosis studies, inflammation at the site of the SC injection was seen in 52% of treated patients. In contrast, injection site inflammation was seen in 3% of multiple sclerosis patients receiving 30 mcg AVONEX® by IM injection. Subcutaneous injections were also associated with the following local reactions: injection site necrosis, injection site atrophy, injection site edema and injection site hemorrhage. None of the above was observed in the multiple sclerosis patients participating in the placebo-controlled study.

Other events observed during premarket and postmarket evaluation of AVONEX®, administered either SC or IM, are listed in the paragraph that follows. Because most of the events were observed in open and uncontrolled studies, or in marketed use, the role of AVONEX® (interferon beta-1a) in their causation cannot be reliably determined.

Body as a Whole: abcess, ascites, cellulitis, facial edema, hernia, injection site fibrosis, injection site hypersensitivity, injection site pain, lipoma, neoplasm, photosensitivity, reaction, rashes, seizures, sinus headache, toothache.

Cardiovascular System: arrhythmia, arthralgia, cardiomyopathy, congestive heart failure, heart arrest, hemorrhage, hypotension, palpitation, pericardial effusion, peripheral ischemia, peripheral vascular disease, postural hypotension, pulmonary embolus, spider angioma, tachycardia, telangiectasia, vascular disorder.

Digestive System: blood in stool, colitis, constipation, diarrhea, diverticulitis, dry mouth, gallbladder disorder, gastritis, gastrointestinal hemorrhage, telangiectasia, vascular disorder.

Endocrine System: diabetes mellitus, thyroid disorder.

Hemic and Lymphatic System: abdominal pain, anemia, anxiety, Bell's palsy, clausimnesis, confusion, depersonalization, drug dependence, emotional lability, facial paralysis, hyperactivity, hyperkinesis, hypoglycemia, hypothyroidism.

Musculoskeletal System: arthralgia, arthritis, cardiomyopathy, congestive heart failure, heart arrest, hemorrhage, hypotension, palpitation, pericardial effusion, peripheral ischemia, peripheral vascular disease, postural hypotension, pulmonary embolus, spider angioma, tachycardia, telangiectasia, vascular disorder.

Nervous System: abnormal gait, amnesia, anxiety, Bell's palsy, clausimnesis, confusion, depersonalization, drug dependence, emotional lability, facial paralysis, hyperactivity, hyperkinesis, hypoglycemia, hypothyroidism.

Metabolic and Nutritional Disorders: abnormal healing, dehydration, hypoglycemia, hypomagnesemia, hypokalemia.

Musculoskeletal Disorders: arthralgia, arthritis, cardiomyopathy, congestive heart failure, heart arrest, hemorrhage, hypotension, palpitation, pericardial effusion, peripheral ischemia, peripheral vascular disease, postural hypotension, pulmonary embolus, spider angioma, tachycardia, teleangiectasia, vascular disorder.

Respiratory System: abnormal gait, amnesia, anxiety, Bell's palsy, clausimnesis, confusion, depersonalization, drug dependence, emotional lability, facial paralysis, hyperactivity, hyperkinesis, hypoglycemia, hypothyroidism.

Skin and Appendages: basal cell carcinoma, blisters, cold clammy skin, contact dermatitis, erythema, fasciculation, genital pruritus, herpes, pruritus, rash, seborrhea, skin ulcer, skin discoloration.

Special Senses: abnormal vision, conjunctivitis, earache, eye pain, labyrinthitis, vitreous floaters.

Urinary Tract: breast fibroadenosis, breast mass, dysuria, epididymitis, fibrocystic change of the breast, fibroids, gynecomastia, hematuria, kidney calculi, kidney pain, leukorrea, menopause, nocturia, pelvic inflammatory disease, penile disorder, Peyronie's Disease, polyuria, postmenopausal hemorrhage, prostatic disorder, pyelonephritis, testis disorder, urinary pain, urinary urgency, urinary retention, urinary incontinence, vaginal hemorrhage.

Serum Neutralizing Activity

Throughout the placebo-controlled multiple sclerosis study, serum samples from patients were monitored for the development of interferon beta-1a neutralizing activity. During the study, 24% of AVONEX®-treated patients were found to have serum neutralizing activity at one or more time points tested. Fifteen percent of AVONEX®-treated patients tested positive for neutralizing activity at a level at which no placebo patient tested positive. The significance of the appearance of serum neutralizing activity is unknown.

**DRUG ABUSE AND DEPENDENCE**

There is no evidence that abuse or dependence occurs with AVONEX® (interferon beta-1a) therapy. However, the risk of dependence has not been systematically evaluated.

**DOSEAGE AND ADMINISTRATION**

The recommended dosage of AVONEX® (interferon beta-1a) for the treatment of relapsing forms of multiple sclerosis is 30 mcg injected intramuscularly once a week. AVONEX® is intended for use under the guidance and supervision of a physician. Patients may self-inject only if their physician determines that it is appropriate and with medical follow-up, as necessary, after proper training in intramuscular injection technique.

**References:**

<table>
<thead>
<tr>
<th>Event</th>
<th>Date/Time</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACTRIMS Steering Committee</td>
<td>Friday, Sept. 20, Noon–1 pm</td>
<td>Board Room, Level 3</td>
</tr>
<tr>
<td>ECTRIMS Council</td>
<td>Friday, Sept. 20, Noon–1 pm</td>
<td>Kent AB, Level 4</td>
</tr>
<tr>
<td>EDMUS Users Group</td>
<td>Thursday, Sept. 19, 4–6 pm</td>
<td>Essex C, Level 4</td>
</tr>
<tr>
<td>MS Journal Editorial Board</td>
<td>Friday, Sept. 20, 7:30 – 9:30 am</td>
<td>Iron, Level 4</td>
</tr>
<tr>
<td>NMSS Pediatric Study Group</td>
<td>Wednesday, Sept. 18, 10:00 am–Noon</td>
<td>Board Room, Level 3</td>
</tr>
<tr>
<td>NMSS Rescue Therapy</td>
<td>Wednesday, Sept. 18, Noon–4 pm</td>
<td>Heron, Level 4</td>
</tr>
<tr>
<td>NMSS Directors of Affiliated</td>
<td>Friday, Sept. 20, 7 – 8:30 am</td>
<td>Atlantic, Level 3</td>
</tr>
<tr>
<td>Clinical Facilities</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Wednesday, September 18

12:00 pm–1:30 pm PRE-CONFERENCE SYMPOSIA

MS FORUM
Controversies Across Continents
Co-chairs:  D Bates (Newcastle, UK)  
G Ebers (Oxford, UK)

Grand Ballroom I–V

12:00  WELCOME and INTRODUCTION  D Bates

12:10  SCENARIO I: DIAGNOSIS  M Clanet (Toulouse, France) and  
B Arnason (Chicago, USA)

12:25  ANN GUIDELINES AND THE EUROPEAN PERSPECTIVE  
D Goodin (San Francisco, USA) and X Montalban (Barcelona, Spain)

12:40  SCENARIO 2: FAILING SLIGHTLY, NO DISEASE PROGRESSION  
L Kappos (Basel, Switzerland) and G Rice (London, Canada)

1:00  SCENARIO 3: RAPID PROGRESSION OF DISEASE  R Hohlfeld  
(Munich, Germany) and B Arnason (Chicago, USA)

1:20  CONCLUDING REMARKS  D Bates

Sponsored by Center for BioMedical Communications, Inc.

Supported through an unrestricted educational grant from  
Schering AG, Germany / Berlex

EUROPEAN CHARCOT FOUNDATION

Does IVIG Have an Effect on Brain Atrophy?  
A Second Look at ESIMS Results

Moderator:  OR Hommes (Nijmegen, Netherlands)

Grand Ballroom VI

12:00  INTRODUCTION  OR Hommes

12:05  ESIMS CLINICAL RESULTS IN PERSPECTIVE OF PREVIOUS  
TRIALS  PS Sørensen (Copenhagen, Denmark)

12:25  ESIMS MRI RESULTS (BPE, MT-MRI)  C Enzinger (Graz, Austria)

12:45  MPRAGE BRAIN ATROPHY MEASUREMENTS IN ESIMS  
PATIENTS  C Constantinescu (Leicester, UK)

1:05  IS BRAIN ATROPHY A NEW PARAMETER TO USE IN CLINICAL  
TRIALS?  M Freedman (Ottawa, Canada)

1:25  DISCUSSION

Supported by a grant from Bayer Corporation
Wednesday, September 18

YOUNG SCIENTIFIC INVESTIGATORS SESSION

Co-chairs: D Brassat (San Francisco, USA)
A Petzold (London, UK)
S Dhib-Jalbut (Baltimore, USA)

Grand Ballroom I–V

2:00 WELCOME and ANNOUNCEMENT, 2002 YOUNG NEUROLOGISTS AND TRAINEES (YNT)–SCHERING FELLOWSHIP AWARD
S Hickmann (London, UK)

2:10 HLA-DRB5*0101 AND -DRB1*1501 EXPRESSION IN THE MULTIPLE SCLEROSIS-ASSOCIATED HLA-DR15DW2 HAPLOTYPE
E Prat, WW Kwok, N Kruse, R Pujol-Borrell, MP Bettinotti, HF McFarland, R Martin (Bethesda, USA)

2:25 QUALITATIVE AND QUANTITATIVE ANALYSIS OF THE BLOOD TCR β CHAIN TRANSCRIPTOME AT DIFFERENT TIME POINTS OF MULTIPLE SCLEROSIS COURSE
DA Loplaud, S Wiertlewski, M Guillet, C Ruiz, B Melchior, G Edan, P Damier, J Soulillou (Nantes, France)

2:40 EXPRESSION OF METABOTROPIC GLUTAMATE RECEPTORS IN MULTIPLE SCLEROSIS BRAIN: UPRÉGULATION IN AXONS AND REACTIVE ASTROCYTES
JJ Geurts, W Kamphorst, P van der Valk, EM Aronica (Amsterdam, Netherlands)

2:55 GROUP CONNECTIVITY MAPS OF OPTIC RADIATIONS AFTER ISOLATED OPTIC NEURITIS
O Ciccarelli, SJ Hickman, AT Toosy, GJ Parker, CA Wheeler-Kingshott, GJ Barker, DH Miller, AJ Thompson (London, UK)

3:10 FUNCTIONAL DIVERSITY OF ANTIBODIES AGAINST MYELIN/OLIGODENDROCYTE GLYCOPROTEIN IN EXPERIMENTAL AUTOIMMUNE DEMYELINATION
H von Büdingen, SL Hauser, A Fuhrmann, CB Nobavi, CF Genain (San Francisco, USA)

3:25 MRI EVIDENCE OF MORE EXTENSIVE TISSUE DAMAGE IN MS PATIENTS WITH THE e4 ALLELE OF APOLIPOPROTEIN E: HIGHER PROPORTION OF LESIONS EVOLVING TO BLACK HOLES DURING TWO-YEAR FOLLOW-UP
C Enzing, S Ropele, S Strasser-Fuchs, P Kapeller, T Seifert, B Poitron, H Schmidt, R Schmidt, F Fazekas (Graz, Austria)

3:40 EVIDENCE FOR AXONAL PATHOLOGY AND ADAPTIVE CORTICAL REORGANIZATION IN PATIENTS AT PRESENTATION WITH CLINICALLY ISOLATED SYNDROMES SUGGESTIVE OF MULTIPLE SCLEROSIS
MA Rocco, D Mezzapesa, A Falini, A Ghezzi, V Martinelli, M Rodegher, G Scotti, G Comi, M Filippi (Milan, Italy)

3:55 A 36-MONTH LONGITUDINAL STUDY ON THE EVALUATION OF THE EFFECT OF INTERFERON BETA IN THE DURATION OF BLACK HOLES IN MULTIPLE SCLEROSIS
F Bagnato, N Jeffries, J Ohayon, R Stone, N Richert, C Bash, HF McFarland, JA Frank (Bethesda, USA)

4:10 DOES FUNCTIONAL MRI ALLOW INFERENCES ABOUT COGNITIVE TRAINING EFFICACY IN MULTIPLE SCLEROSIS?
I Penner, L Kappos, M Rausch, K Opwis, E Radu (Basel, Switzerland)

4:25 COMBINATION THERAPY OF MS PATIENTS WITH INCOMPLETE RESPONSE TO INTERFERON-BETA WITH HUMANIZED ANTIBODY AGAINST THE INTERLEUKIN-2 RECEPTOR ALPHA CHAIN
B Bielekova, S Reichert-Scrimer, J Wuerfel, J Ohayon, J McCartin, N Richert, J Frank, T Waldmann, H McFarland, R Martin (Cambridge, UK)

SATELLITE SYMPOSIUM I

Selective Adhesion Molecule Inhibition: A Potential Future Treatment for Multiple Sclerosis
Chair: C Confavreux (Lyon, France)

Grand Ballroom VI–X

2:00 WELCOME and OBJECTIVES
C Confavreux

5:05 CELLULAR ADHESION PATHWAYS: POTENTIAL TARGETS FOR FUTURE MULTIPLE SCLEROSIS THERAPIES
P Calabresi (Baltimore, USA)

5:25 EMERGING CLINICAL DATA FOR SELECTIVE ADHESION MOLECULE INHIBITION IN THE TREATMENT OF MULTIPLE SCLEROSIS
D Miller (London, UK)

5:40 CLOSING REMARKS
C Confavreux

5:50 PANEL DISCUSSION

Sponsored by the Division of Continuing Medical Education, Discovery International

Supported through an education grant from Biogen, Inc. and Elan

7:00 pm–9:00 pm
ACTRIMS-ECTRIMS 2002 WELCOME RECEPTION
Grand Ballroom
Thursday, September 19

8:15 am–9:30 am  OPENING SESSION
Grand Ballroom I–X

8:15 am  WELCOME  
K Johnson (Baltimore, USA) and A Thompson (London, UK)
Opening Remarks  
Mike Dugan, General, USAF Ret.
President and CEO, National Multiple Sclerosis Society, New York, USA
Victor Rivera, President, LACTRIMS

8:45 am  KEYNOTE ADDRESS
I Inflammation, Demyelination and Axonal Loss: Unraveling the Relationships
SK Ludwin★ (Kingston, Canada)

9:30 am  BREAK

10:00 am–12:00 pm  PARALLEL SESSIONS

SESSION I
Inflammation, Demyelination and Axonal Loss: Insights from Pathology
Co-chairs: C Polman (Amsterdam, Netherlands)  
E Radue (Basel, Switzerland)

Grand Ballroom I–V

10:00 12  MECHANISMS OF AXONAL LOSS  
BD Trapp★, C Bjartmar, J Peterson, A Chang, R Rudick (Cleveland, USA)

10:25 13  RELATIONSHIP BETWEEN INFLAMMATION AND AXONAL LOSS  
W Brueck★ (Berlin, Germany)

10:50 14  DIFFERENTIAL GENE EXPRESSION ANALYSIS OF MULTIPLE SCLEROSIS TISSUE: COMPARISON OF ACTIVE AND INACTIVE LESIONS  
MP Mycko, R Patoa, U Boschert, CS Raine, KW Selma★ (Lodz, Poland)

11:00 15  MICROARRAY ANALYSIS OF NORMAL APPEARING WHITE MATTER (NAWM) AND LESIONS IN SECONDARY PROGRESSIVE MS VERIFIES MS AS A GENERALIZED CNS DISEASE  
RL Lindberg, CJ De Groot, U Cerva, R Ravid, F Hoffmann, L Kappos, D Leppert (Basel, Switzerland)

11:10 16  MULTIPLE SCLEROSIS: EXPANDED CSF B CELLS ARE ALSO PRESENT IN THE BRAIN TISSUE  
N Goebels, H Weber, M Hofbauer, H Wekerle, R Hotilfied (Munich, Germany)

11:20 17  HIGH VULNERABILITY OF HUMAN NEURONS TO T CELL CYTOTOXICITY: A NEW MODEL TO EXPLAIN NEURODEGENERATION IN MULTIPLE SCLEROSIS  
F Giuliani, V Yong (Calgary, Canada)

11:30 18  LEUKEMIA INHIBITORY FACTOR LIMITS IMMUNE-MEDIATED DMYELINATION BY ENHANCING OLIGODENDROCYTE SURVIVAL  
H Butzkueven, J Zhang, M Soili-Hanninen, F Bartlett, T Kilpatrick (Parkville, Australia)

11:40 19  CILIARY NEUROTROPHIC FACTOR ENHANCES MYELIN FORMATION: A NOVEL ROLE FOR CNTF AND CNTF-RELATED MOLECULES  
S Bruno, N Frederic, A Marie Stephane, Z Bernard, L Catherine (Paris, France)

11:50  CONCLUSIONS

SESSION II
Impact of Relapses on Disability; Natural History and Clinical Trials Data
Co-chairs: L Kappos (Basel, Switzerland)  
H Panitch (Burlington, USA)

Grand Ballroom VI–X

10:00 20  THE ROLE OF EXACERBATIONS IN PERSISTENT IMPAIRMENT IN MS  
F Lublin★, G Cutter, M Baier (New York, USA)

10:20 21  RELAPSES ARE NOT AN IMPORTANT CAUSE OF DISABILITY  
C Conferux★ (Lyon, France)

10:40  PANEL DISCUSSION  
Moderator: K Kappos

11:00 22  ONSET OF CLINICAL BENEFIT OF GLATIRAMER (COPAXONE®) ACETATE IN PATIENTS WITH RELAPSING REMITTING MULTIPLE SCLEROSIS (RRMS)  
KP Johnson, BR Brooks, CC Ford, A Goodman, JB Guarneri, RP Lisak, LW Myers, HS Panitch, AA Pruitt, N Kachuck, JS Wolinsky, and the Copolymer 1 MS Study Group (Baltimore, USA)

11:15 23  EFFECT OF EARLY INTERFERON TREATMENT ON CONVERSION TO DEFINITE MULTIPLE SCLEROSIS: THE ETOMS STUDY—4-YEAR RESULTS  
G Comi, M Filippi, F Barkhof, L Durelli, G Edan, O Fernandez, H Hartung, P Seelstreyer, F Soelberg Sorensen, O Hommes (Turin, Italy)

11:30 24  NEUTRALIZING ANTIBODIES AGAINST INTERFERON (IFN)-BETA REDUCE THE CLINICAL EFFECT IN RELAPSING-REMITTING MULTIPLE SCLEROSIS  
P Sorensen, N Koch-Henriksen, C Ross, KM Clemmensen, M Svenson, K Bendor, K Jensen, O Kristensen, T Petersen, E Steneger, (Copenhagen, Denmark)

11:45 25  THE SYLVIA LAWRY CENTRE FOR MULTIPLE SCLEROSIS RESEARCH (SLCMSR): BACKGROUND AND PROGRESS REPORT  
JH Noseworthy and SLCMSR Staff, Scientific Oversight Committee and Working Groups (Rochester, USA)

★ Invited speaker
Thursday, September 19

12:00 pm–2:00 pm  LUNCH and POSTER SESSION I  
Harborside Level

2:00 pm–4:00 pm  PARALLEL SESSIONS

SESSION III  
Inflammation, Demyelination and Axonal Loss: Insights from Imaging  
Co-chairs:  J Simon (Denver, USA)  
N Richert (Bethesda, USA)  

Grand Ballroom I–V

2:00 26  RELATIONSHIP BETWEEN CONTRAST ENHANCING LESIONS AND AXONAL LOSS  
JA Frank★ (Bethesda, USA)

2:20 27  IN VIVO MONITORING OF AXONS AND MYELIN IN MULTIPLE SCLEROSIS  
Z Caramanos, DL Arnold★ (Montreal, Canada)

2:40 28  CAN WE IMAGE REMYELINATION?  
F Barkhof★ (Amsterdam, Netherlands)

3:00 29  WHAT IS NORMAL-APPEARING WHITE MATTER?  
R Grossman★ (San Francisco, USA)

3:20 30  BRAIN VOLUME CHANGES IN PATIENTS AT PRESENTATION WITH SUSPECTED MULTIPLE SCLEROSIS: RESULTS FROM THE ETOMS STUDY  
G Comi, M Inglese, N De Stefano, S Smith, F Barkhof, L Durelli, G Edan, O Fernandez, HP Hartung, PM Matthews, P Seeldrayers, PS Sorensen, V Martinelli, OR Hommes, M Filippi (Milan, Italy)

3:30 31  PREDICTIVE VALUE OF INFRAVENTSORIAL LESIONS IN PATIENTS WITH CLINICALLY ISOLATED SYNDROMES FOR LONG TERM DISABILITY  
A Minneboo, F Barkhof, CH Polman, BM Uitdehaag, D Knol, JA Casteljns (Amsterdam, Netherlands)

3:40 32  A 48-MONTH LONGITUDINAL STUDY ON THE RELATIONSHIP BETWEEN THE DURATION OF THE ENHANCEMENT IN AN ACTIVE LESION AND THE DURATION OF A BLACK HOLE IN MULTIPLE SCLEROSIS  
E Bagnato, N Jeffries, J Ohayon, R Stone, JA Frank, HF McFarland (Bethesda, USA)

3:45 P133  THE EFFECT OF INTERFERON B-1B ON QUANTITIES DERIVED FROM MT MRI IN SECONDARY PROGRESSIVE MS  
M Inglese, J vanWaesberghe, M Rowaris, K Beckmann, F Barkhof, D Hahn, L Kappos, D Miller, C Polman, C Pozzilli, A Thompson, T Youns, K Wagner, G Comi, M Filippi (Milan, Italy)

3:50 P40  ISOLATED SPINAL DEMYELINATING EVENTS WITH NORMAL BRAIN MRI: PROGRESSION TO MS, CLINICAL AND MRI FOLLOW UP  
R Milo, T Katz, J Corot-Simon (Ashkelon, Israel)

3:55 CONCLUSIONS

SESSION IV  
The Blood-Brain-Barrier as a Target for Treatment  
Co-chairs:  S Dhib-Jalbut (Baltimore, USA)  
J Oger (Vancouver, Canada)

Grand Ballroom VI–X

2:00 33  ADHESION MOLECULES AND THEIR ROLE IN PATHOGENESIS  
JP Antel★, K Biemacki, R Seguin, A Prat (Montreal, Canada)

2:20 34  CHEMOKINES AND CHEMOKINE RECEPTORS: WHAT’S THE ATTACHMENT  
RM Ransohoff★ (Cleveland, USA)

2:40 35  MATRIX METALLOPROTEINASES IN MS  
VV Yang★ (Calgary, Canada)

3:00 36  CLINICAL TRIALS OF AGENTS TARGETING THE BLOOD BRAIN BARRIER: SUCCESSES AND FAILURES  
D Miller★ (London, UK)

3:20 P143  RANTES AND CHEMOKINE RECEPTOR 5 POLYMORPHISMS: SUSCEPTIBILITY TO AND OUTCOME IN MULTIPLE SCLEROSIS  
JM Partridge, A Fryer, W Ollier, M Boggild, R Strange, C Hawkins (Stoke-on-Trent, UK)

3:25 P145  MMP-9 MICROSYLULATE POLYMOPHISM INCREASES THE RISK OF MULTIPLE SCLEROSIS  
F Bagnato, T Katz, J Corot-Simon (Ashkelon, Israel)

3:30 P114  IL-12 DEPENDENT/IFN GAMMA INDEPENDENT EXPRESSION OF CCR5 BY MYELIN-REACTIVE CD4+ T CELLS CORRELATES WITH ENCEPHALITOREGALITY  
L Bagaeva, LP Williams, BM Segal (Rochester, USA)

3:35 P91  LONGITUDINAL ANALYSIS OF CSF EXPANDED CD8+ CLONOTYPES IN THE PERIPHERAL BLOOD OF MULTIPLE SCLEROSIS PATIENTS  
S Cepok, D Zhou, F Vogel, N Sommer, B Hemmer (Marburg, Germany)

3:40 P98  MOLECULAR TRACKING OF MYELIN BASIC PROTEIN-SPECIFIC T CELL EXPANSION IN MULTIPLE SCLEROSIS  
PM Munro, K Wandinger, B Bielekova, HF McFarland, R Martin (Bethesda, USA)

3:45 P149  IMMUNE REGULATORY EFFECTS OF GLATIRAMER ACETATE (GA) ON HUMAN MONOCYTES: BYSTANDER SUPPRESSION REVISTED?  
H Kim, M Duddy, A Bar-Or (Montreal, Canada)

3:50 CONCLUSIONS

★ Invited speaker
### Thursday, September 19

#### SATELLITE SYMPOSIA

<table>
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<tr>
<th>Time</th>
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| 4:30 pm–5:30 pm | **SATELLITE SYMPOSIUM II**  
Exploring the Boundaries of Multiple Sclerosis Treatment | Chair: D Goodin (San Francisco, USA)  
Grand Ballroom I–V |
| 6:00 pm–7:00 pm | **SATELLITE SYMPOSIUM III**  
Defining Factors That Impact Efficacy in the Treatment of Relapsing Remitting Multiple Sclerosis | Program Chair: HP Hartung (Dusseldorf, Germany)  
Panel Discussion Chair: H McFarland (Washington, DC, USA)  
Grand Ballroom VI–X |

**4:30**  
**WELCOME**  
D Goodin

**4:35**  
**EXPLORING TREATMENT OPTIONS FOR PRIMARY PROGRESSIVE MULTIPLE SCLEROSIS**  
X Montalban (Barcelona, Spain)

**4:50**  
**LONG TERM EXPERIENCE WITH MULTIPLE SCLEROSIS THERAPIES**  
G Rice (London, Canada)

**5:05**  
**BEYOND THE STANDARD DOSE OF BETA INTERFERON IN MULTIPLE SCLEROSIS**  
HP Hartung (Dusseldorf, Germany)

**5:20**  
**DISCUSSION AND CLOSING REMARKS**  
D Goodin

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**Sponsored by Bio-Medical Communications, Inc.**

**Supported by an unrestricted educational grant from Schering AG, Germany / Berlex**

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**6:00**  
**WELCOMING REMARKS/INTRODUCTIONS**  
HP Hartung

**6:05**  
**SELECTING HIGH-RISK PATIENTS FOR EARLY TREATMENT**  
F Munschauer (Buffalo, USA)

**6:15**  
**DO DOSE AND DOSE FREQUENCY IMPACT EFFICACY? A REVIEW OF THE EUROPEAN DOSE COMPARISON STUDY AND SUPPORTING DATA**  
X Montalban (Barcelona, Spain)

**6:25**  
**LONG-TERM EFFICACY OF INTERFERON BETA—WHY NABS MATTER**  
PS Sorensen (Copenhagen, Denmark)

**6:40**  
**PANEL DISCUSSION/Q&A**  
H McFarland

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**Sponsored by the Health Science Center for Continuing Medical Education**

**Supported by an unrestricted educational grant from Biogen**

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<tr>
<td>7:30 pm–10:30 pm</td>
<td><strong>RECEPTION and DINNER BUFFET</strong></td>
<td>National Aquarium in Baltimore</td>
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23
## Friday, September 20

### 8:30 am–9:30 am OPENING SESSION
Grand Ballroom I–X

**8:30 am**  
**WELCOME**  
*S Dhib-Jalbut* (Baltimore, USA)  
**PRESENTATION:** ACTRIMS LIFE ACHIEVEMENT AWARD  
*Kenneth P. Johnson,* Honoree

**8:45 am**  
**KEYNOTE ADDRESS**  
37 Neural Stem Cells to Rebuild the Diseased Brain: How Realistic Is This Approach?  
*E Snyder* ★ (Boston, USA)

**9:30 am**  
**BREAK**

### 10:00 am–12:00 pm PARALLEL SESSIONS

#### SESSION V  
**Neuroprotection**
Co-chairs:  
*A Cross* (St Louis, USA)  
*R Lisak* (Detroit, USA)

Grand Ballroom I–V

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<th>Time</th>
<th>Title</th>
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<tr>
<td>10:00</td>
<td>MOLECULAR BASIS OF LIMITED REMYELINATION IN MULTIPLE SCLEROSIS</td>
<td><em>CS Raine</em>★, G John, CF Brosnan* (Bronx, USA)</td>
<td>Grand Ballroom I–V</td>
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<tr>
<td>10:25</td>
<td>COMPLEMENT: DUAL ROLE IN INJURY AND PROTECTION</td>
<td><em>ML Shin</em>★, H Rus* (Baltimore, USA)</td>
<td>Grand Ballroom I–V</td>
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<tr>
<td>10:50</td>
<td>IS NEUROPROTECTION A REALISTIC OPTION IN MS?</td>
<td><em>R Hohlfeld</em>★ (Munich, Germany)</td>
<td>Grand Ballroom I–V</td>
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#### SESSION VI  
**Hot topics in Neuroimmunology**
Co-chairs:  
*M Racke* (Dallas, USA)  
*J Richert* (Washington, DC, USA)

Grand Ballroom VI–X

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<tr>
<td>10:00</td>
<td>CYTOKINE REGULATION IN MULTIPLE SCLEROS</td>
<td><em>H Weiner</em>★, S J Khoury* (Boston, USA)</td>
<td>Grand Ballroom VI–X</td>
</tr>
<tr>
<td>10:25</td>
<td>ARE SPECIFIC IMMUNOTHERAPIES AN OPTION FOR MS?</td>
<td><em>R Martin</em>★ (Bethesda, USA)</td>
<td>Grand Ballroom VI–X</td>
</tr>
<tr>
<td>10:50</td>
<td>TCR PEPTIDE THERAPY IN AUTOIMMUNE DISEASE</td>
<td><em>AA Vandenbark</em>★ (Portland, USA)</td>
<td>Grand Ballroom VI–X</td>
</tr>
<tr>
<td>11:15</td>
<td>LARGE SCALE TRANSCRIPTIONAL AND PROTEOMIC ANALYSIS OF MS TISSUE YIELDS NEW TARGETS FOR THERAPY</td>
<td><em>L Steinman</em>★ (Stanford, USA)</td>
<td>Grand Ballroom VI–X</td>
</tr>
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Friday, September 20

12:00 pm–2:00 pm  LUNCH and POSTER SESSION II
Harborside Level

2:00 pm–4:00 pm  PARALLEL SESSIONS

**SESSION VII**
Methodological Issues in Clinical Trials

Co-chairs:  
HP Hartung (Dusseldorf, Germany)  
R Rudick (Cleveland, USA)

Grand Ballroom I–V

2:00  50  THE NEW DIAGNOSTIC CRITERIA AND THEIR IMPLICATIONS FOR CLINICAL TRIALS  
JS Wolinsky★ (Houston, USA)

2:25  51  APPLICATION OF MACDONALD CRITERIA TO CLINICALLY ISOLATED SYNDROMES SUGGESTIVE OF MULTIPLE SCLEROSIS  
M Tintore, A Rovira, J Rio, C Nos, E Grieve, J Sastre-Garriga, I Pericot, E Sanchez,  
M Comabella, X Montalban (Barcelona, Spain)

2:35  52  METHODOLOGICAL ISSUES IN SHORT-TERM CLINICAL TRIALS  
JH Noseworthy★ (Rochester, USA)

3:00  53  THE ROLE OF MRI AS A SURROGATE MARKER IN MS  
H McFarland★ (Bethesda, USA)

2:25  54  A STANDARDIZED MRI SCAN IN THE DIAGNOSIS AND FOLLOW-UP OF MS PATIENTS  
Patty★, DK Li, A Traboulsi, J Simon, J Frank (Vancouver, Canada)

3:50  P227  COURSE AND PROGNOSIS IN EARLY ONSET MULTIPLE SCLEROSIS  
R Simone, D Carrara, C Tortorella, M Liguori, V Lepore, F Pelligrini, A Bellacosa, A Ceccarelli, I Pavone, F Girolamo, P Livrea (Bari, Italy)

3:55  P308  PLACEBO-CONTROLLED DOUBLE-BLIND DOSE RANGING STUDY OF FAMPRIFINE-SR IN MULTIPLE SCLEROSIS  
AD Goodman, A Blight, J. Cohen, AH Cross, M Katz, MA Rizzo, T Vollmer (Rochester, USA)

3:20  56  INSIGHTS INTO THE GENETICS OF MS FROM THE CANADIAN COLLABORATIVE PROJECT  
GC Ebers★, D Sodovnick, N Risch (Vancouver, Canada)

2:40  57  THE ROLE OF THE HLA REGION IN MULTIPLE SCLEROSIS  
S Hauser★, LF Barcellos, MA Pericak-Vance, JL Haines, RR Lincoln, S Schmidt,  
A Swedlin, JR Oksenberg (Durham, USA)

3:00  58  HORMONAL INFLUENCES IN MS  
RVoskuhl★ (Los Angeles, USA)

3:20  59  OVARIAN HORMONES DIFFERENTIALLY EFFECT NEURON DEATH MEDIATED BY TNFα VIA EXPRESSION OF ANTI-APOPTOTIC PROTEINS AND ACTIVATION OF JNK1 PRO-APOPTOTIC SIGNAL CASCADE  
C Kossi, S Hila, T Popescue, G Hoffman (Baltimore, USA)

3:30  60  A NEW GENE OVEREXPRESSED IN MULTIPLE SCLEROSIS AND RHEUMATOID ARTHRITIS  
D Greene, R Crusio, L Chen, C Rose,  
D Connelly, M Grekova, JR Richert (Washington, USA)

3:40  P325  ASSOCIATION OF APOLIPOPROTEIN E AND MYELOPEROXIDASE GENOTYPES WITH THE CLINICAL COURSE OF FAMILIAL AND SPORADIC MULTIPLE SCLEROSIS  
B Zakrzeswska-Pniewska, A Podlecka, M Styczynska, R Samacka, B Peplonska, M Barcikowska,  
H Kwiecinski (Warsaw, Poland)

3:45  P314  TUMOR NECROSIS FACTOR RECEPTOR II POLYMORPHISM IN PATIENTS WITH MULTIPLE SCLEROSIS  
R Ehling, C Gassner,  
F Fazekas, H Kollegger, W Kristoferitsch, M Reindl, T Berger (Innsbruck, Austria)

3:50  P297  A SYNTHETIC ANDROSTERONE DERIVATIVE WITHOUT GENDER-RELATED SIDE EFFECTS INHIBITS EAE. CANDIDATE FOR CLINICAL TRIALS IN MS?  
H Offner, A Zamora, A Matejek, D Auci, E Morgan,  
C Reading (Portland, USA)

3:55  CONCLUSIONS

**SESSION VIII**
Genetics and Hormonal Influence

Co-chairs:  
M Freedman (Ottawa, Canada)  
D Hafler (Boston, USA)

Grand Ballroom VI–X

2:00  55  GENETIC ANALYSIS OF MULTIPLE SCLEROSIS IN EUROPEANS (GAMES)  
A Compston★, S Sawcer (Cambridge, UK)

3:20  59  OVARIAN HORMONES DIFFERENTIALLY EFFECT NEURON DEATH MEDIATED BY TNFα VIA EXPRESSION OF ANTI-APOPTOTIC PROTEINS AND ACTIVATION OF JNK1 PRO-APOPTOTIC SIGNAL CASCADE  
C Kossi, S Hila, T Popescue, G Hoffman (Baltimore, USA)

3:30  60  A NEW GENE OVEREXPRESSED IN MULTIPLE SCLEROSIS AND RHEUMATOID ARTHRITIS  
D Greene, R Crusio, L Chen, C Rose,  
D Connelly, M Grekova, JR Richert (Washington, USA)

3:40  P325  ASSOCIATION OF APOLIPOPROTEIN E AND MYELOPEROXIDASE GENOTYPES WITH THE CLINICAL COURSE OF FAMILIAL AND SPORADIC MULTIPLE SCLEROSIS  
B Zakrzeswska-Pniewska, A Podlecka, M Styczynska, R Samacka, B Peplonska, M Barcikowska,  
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H Offner, A Zamora, A Matejek, D Auci, E Morgan,  
C Reading (Portland, USA)

3:55  CONCLUSIONS

★ Invited speaker
Friday, September 20

SATELLITE SYMPOSIA

4:30 pm–5:30 pm
SATELLITE SYMPOSIUM IV
Milestones in Immunomodulatory Therapy: Decisions in the Treatment of Multiple Sclerosis
Co-chairs:  G Comi (Milan, Italy)
            J Wolinsky (Houston, USA)
Grand Ballroom I–V

4:30 IMMUNE-MEDIATED INJURY AND NEUROPROTECTION IN MS
W Yong (Calgary, Canada)

4:50 LESSONS FROM MRI: EVIDENCE OF EARLY AND PROGRESSIVE
CNS INJURY  D Arnold (Montreal, Canada)

5:10 GLATIRAMER ACETATE IN MS: A NEW LOOK AT THE CLINICAL
EFFECTS IN THE LIGHT OF MECHANISMS OF ACTION  R Lisak (Detroit, USA)

Sponsored by Postgraduate Institute for Medicine

Supported by an unrestricted educational grant from
Teva Pharmaceuticals, LTD, Teva Neuroscience, and Aventis

6:00 A CRITICAL ANALYSIS OF DISEASE-MODIFYING DRUGS IN
CLINICAL STUDIES: IMPLICATIONS AND TREATMENT GUIDELINES
D Goodin (San Francisco, USA)

6:15 THE EVIDENCE FOR EFFICACY OF DISEASE-MODIFYING
DRUGS: IMPLICATIONS FOR THE CLINICIAN  M Freedman (Ottawa,
Canada)

6:35 THE PATIENT PERSPECTIVE: LIVING WITH MULTIPLE SCLEROSIS
AFTER STARTING A DISEASE-MODIFYING DRUG

6:50 Q&A AND CLOSING REMARKS

Sponsored by Serono

6:00 pm–7:00 pm
SATELLITE SYMPOSIUM V
The Modern Management of Multiple Sclerosis: An Evidence-Based Approach
Chair:  D Bates (Newcastle, UK)
Grand Ballroom VI–X

7:30 pm–10:00 pm  RECEPTION and DINNER BUFFET
B&O Railroad Museum
8:30 am–10:30 am PARALLEL SESSIONS

SESSION IX
Long Term Management Issues in Multiple Sclerosis
Co-chairs: C Bever (Baltimore, USA) J Cohen (Cleveland, USA)
Grand Ballroom I–V

SESSION X
Late Breaking News
Co-chairs: P O’Connor (Toronto, Canada) A Thompson (London, UK)
Grand Ballroom VI–X

8:30 WELCOME C Bever
8:45 LB1 NEUROPSYCHOLOGICAL ASPECTS OF MULTIPLE SCLEROSIS A Feinstein★ (Toronto, Canada)
9:05 LB2 PATHOPHYSIOLOGY OF MS FATIGUE G Comi★, L Leocani, P Rossi, B Colombo (Milan, Italy)
9:25 LB3 STEREOTACTIC SURGERY E Montgomery★ (Cleveland, USA)
9:45 LB4 CHILDHOOD ONSET MULTIPLE SCLEROSIS (THE KID-MUS STUDY): NATURAL HISTORY AND PROGNOSTIC FACTORS IN THE LYON COHORT C Renoux Y Mikaeloff, S Ykussic, L Gignoux, F Durand-Dubief, I Achiti, C Confavreux (Lyon, France)
10:05 LB6 GADOLINIUM ENHANCING LESIONS AS A SURROGATE MARKER OF INTERFERON RESPONSE RA Rudick, G Cutter, M Baier, D Dougherty, B Weinstock-Guttman, M Mass, E Fisher, DM Miller, A Sandrock, J Simon (Cleveland, USA)
10:15 LB7 MITOXANTRONE (NOVANTRONE) FOR TREATMENT OF RECURRENT NEUROMYELITIS OPTICA B Weinstock-Guttman, J Feichter, R Bakshi, C Brownscheidle, N Lincoff (Buffalo, USA)
10:25 CONCLUSIONS

8:30 WELCOME P O’Connor
8:45 LB1 VALIDATION OF DIAGNOSTIC MRI CRITERIA FOR MS AND RESPONSE TO TREATMENT WITH INTERFERON-BETA-1A E Barkhof, M Rocca, G Francis, J van Woeberghoe, B Uitdehaag, O Hommes, H Hartung, L Durelli, G Edan, O Fernandez, P Seeldrayers, P Sorenson, S Margrie, G Comi, M Filippi (Milan, Italy)
9:00 LB2 ANTI-MOG ANTIBODIES PREDICT EARLY CONVERSION TO CLINICALLY DEFINITE MS IN PATIENTS WITH A FIRST DEMYELINATING EVENT T Berger, P Rubner, F Schautzer, R Egg, H Ulmer, I Mayringer, E Dilitz, F Deisenhammer, M Reindl (Innsbruck, Austria)
9:15 LB3 NEUROREHABILITATION IN MULTIPLE SCLEROSIS CONTRIBUTES TO FUNCTIONAL RECOVERY ACCOMPANIED BY CHANGES OF BRAIN ACTIVITY ON FMRI—PRELIMINARY RESULTS K Raso, J Krasensky, J Havrdova, J Obenberger, M Zalisova, Z Seidl (Prague, Czech Republic)
9:30 LB4 TIGHT JUNCTION ABNORMALITY IN MS AFFECTS ALL CALIBRES OF VESSEL AND CORRELATES WITH LESION ACTIVITY J Kirk, J Plumb, M Mirokhur, S McQuaid (Belfast, UK)
9:45 LB5 SINGLE CENTRE DBPC, RANDOMISED TRIAL OF INTERFERON-β1B IN PRIMARY PROGRESSIVE AND TRANSITIONAL PROGRESSIVE MULTIPLE SCLEROSIS: AN EXPLORATORY PHASE II STUDY X Montalban, L Brieva, M Tintore, C Borras, J Ria, C Nos, X Aymerich, J Alonso, R Horno, M Vicente, A Rovira (Barcelona, Spain)
10:00 LB6 SUCCESSFUL TREATMENT WITH IFN-β1B IN RR MS PATIENTS IS ASSOCIATED WITH AN INCREASE IN THE NUMBER OF IL-10 PRODUCING (REGULATORY) CD4+ T CELLS A van Baal-Dezaire, M Smits, B Uitdehaag, C Polman, L Nagelkerken (Leiden, Netherlands)
10:15 LB7 MULTIPLE SCLEROSIS DOCUMENTATION SYSTEM—MSDS 2.0 M Eulitz, T Kugel, PA Muraro, M Pette (Dresden, Germany)

10:30 am COFFEE BREAK

11:00 am–12:00 pm CLOSING SESSION
Grand Ballroom I–X

11:00 am ECTRIMS LECTURE 68 Quo Vadis? Agenda for European MS Research OR Hommes★ (Nijmegen, Netherlands)
11:45 am PRESENTATION: 2ND ANNUAL ECTRIMS AWARD OR Hommes, Honoree PROGRAM AWARDS and CLOSING REMARKS

12:00 pm–1:00 pm CLOSING LUNCHEON
Harborside Ballroom

7:00 pm–10:00 pm COMMITTEE RECEPTION
Baltimore Museum of Industry (by invitation)
COPAXONE® is indicated for the reduction of relapses in relapsing-remitting multiple sclerosis.

Please see brief summary of prescribing information on next page.

COPAXONE® is a registered trademark of Teva Pharmaceutical Industries Ltd.
The only MS therapy with

- A presumed mechanism of action that distinguishes it from interferons\textsuperscript{1,2}
- No evidence of neutralizing antibodies\textsuperscript{3}
- No recommended monitoring of liver function or complete blood count\textsuperscript{4}
- Pregnancy Category B rating\textsuperscript{4}

Significant relapse rate reduction

- Long-term efficacy demonstrated over 2 years\textsuperscript{5,6}
- Efficacy confirmed in 4 additional studies\textsuperscript{7-10}

Reduction in Gd-enhancing lesions

- 35% reduction in median cumulative number of lesions vs placebo\textsuperscript{11}

Safety and tolerability you can count on

- No increase in incidence of flu-like symptoms, depression, or fatigue when compared to placebo\textsuperscript{4}

- Most common adverse effects in controlled trials were injection site reactions, vasodilatation, chest pain, asthenia, infection, pain, nausea, arthralgia, anxiety, and hypertonia

- About 10% of patients experienced an immediate postinjection reaction (flushing, chest pain, palpitations, anxiety, dyspnea, throat constriction, and urticaria). The symptoms were transient and self-limited, and did not require specific treatment

- Transient chest pain was noted in 26% of COPAXONE\textsuperscript{®} patients (vs 10% placebo); no long-term sequelae

NEW!

COPAXONE\textsuperscript{®}
(glatiramer acetate injection)

Benefits you can measure over time
Impairment of Fertility

Patients should be cautioned against the reuse of needles or syringes and instructed in safe disposal methods. Injection (see PRECAUTIONS: Information for Patients and the COPAXONE® INJECTION PATIENT INFORMATION Leaflet). The first injection should be performed under the supervision of an appropriately qualified health care professional. Patient education and understanding of use of aspetic self-injection techniques and procedures should be periodically reevaluated. Patients should be cautioned against the reuse of needles or syringes and instructed in safe disposal procedures. They should use a puncture-resistant container for disposal of used needles and syringes. Patients should be instructed on the safe disposal of full containers according to local laws.

Awareness of Adverse Reactions: Physicians are advised to counsel patients about adverse reactions associated with the use of COPAXONE® Injection (see ADVERSE REACTIONS section). In addition, patients should be advised to read the COPAXONE® INJECTION PATIENT INFORMATION Leaflet and resolve any questions regarding it prior to beginning COPAXONE® Injection therapy.

Laboratory Tests

Data collected during premarketing development do not suggest the need for routine laboratory monitoring. Treatment of your physician if you are pregnant, if you are planning to have a child, or if you become pregnant while taking this medication. Inform your physician if you are nursing.

2. Inform your physician if you are nursing.

3. Do not change the dose or dosing schedule without consulting your physician.

4. Do not stop taking the drug without consulting your physician. Patients should be instructed in the use of asptic techniques when administering COPAXONE® Injection. Appropriate instructions for the self-injection of COPAXONE® Injection should be given, including a careful review of the COPAXONE® INJECTION PATIENT INFORMATION Leaflet. The first injection should be performed under the supervision of an appropriately qualified health care professional. Patient education and understanding of use of aspetic self-injection techniques and procedures should be periodically reevaluated. Patients should be cautioned against the reuse of needles or syringes and instructed in safe disposal procedures. They should use a puncture-resistant container for disposal of used needles and syringes. Patients should be instructed on the safe disposal of full containers according to local laws.

Caricogenesis, Mutagenesis, Impairment of Fertility

Carcinogenesis

In a two-year carcinogenicity study, mice were administered up to 60 mg/kg/day glatiramer acetate by subcutaneous injection (up to 15 times the human therapeutic dose on a mg/m² basis). No increase in systemic neoplasms was observed. In males of the high dose group (60 mg/kg/day), but not in females, there was an increased incidence of fibrousomas at the injection sites. These sarcomas were associated with skin damage precipitated by repetitive injections of an irritant over a limited skin area. In patients with MS, patients who received COPAXONE® and patients with placebo (Table 3). These trials include the first two controlled trials in RR MS patients and a controlled trial in patients with Chronic Progressive MS. Adverse reactions were usually mild in intensity. The prescriber should be aware that these figures cannot be used to predict the frequency of adverse experiences in the course of usual medical practice where patient characteristics and other factors may differ from those prevailing during clinical studies. Similarly, the cited frequencies cannot be directly compared with figures obtained from other clinical investigations involving different treatments, uses, or investigators. An inspection of these frequencies, however, does provide the prescriber with one basis on which to estimate the relative contribution of drug and nondrug factors to the adverse reaction incidences in the population studied.

Controlled Trials in Patients with Multiple Sclerosis: Incidence of Glatiramer Acetate Adverse Reactions and More Frequent than Placebo

<table>
<thead>
<tr>
<th>Preferred Term</th>
<th>N</th>
<th>%</th>
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</table>
Other events which occurred in at least 2% of glatiramer acetate patients but were present at equal or greater rates in the placebo group included:

- **Respiratory System**: Bronchitis, rhinitis, and sinusitis.
- **Skin and Appendages**: Erythema, herpes simplex, herpes zoster, psoriasis, and acne.
- **Gastrointestinal System**: Abdominal pain, diarrhea, and constipation.
- **Nervous System**: Dizziness, headache, and dizziness.
- **Musculoskeletal System**: Arthritis, myalgia, and myopathy.
- **Other**: Urinary tract infection, abdominal pain, and injection site reactions.

Postmarketing experience has shown an adverse event profile similar to that presented above. Reports of adverse reactions occurring under treatment with COPAXONE® (glatiramer acetate for injection) not mentioned above that have been received since market introduction and that may have or not have causal relationship to the drug include the following:

- **Cardiovascular**: Thrombosis, peripheral vascular disease, and paroxysmal atrial fibrillation.
- **Central Nervous System**: Psychosis, delirium, and anxiety.
- **Gastrointestinal**: Gastrointestinal obstruction, ileus, and abdominal pain.
- **Respiratory**: Hyperventilation, hyperventilation, and wheezing.
- **Musculoskeletal**: Arthritis, muscle atrophy, bone pain, bursitis, and tendinopathy.

**References:**
5. *COPAXONE® prescribing information. Teva Neuroscience, Inc.
7. *COPAXONE® prescribing information. Teva Neuroscience, Inc.

**Preferred Term (continued)**

<table>
<thead>
<tr>
<th>Preferred Term</th>
<th>Glatiramer Acetate (N = 201)</th>
<th>Placebo (N = 206)</th>
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<tr>
<td><strong>Postmarketing Experience</strong></td>
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**COPAXONE® prescribing information. Teva Neuroscience, Inc.
Cop2190BP Rev 5 10/2002

**References:**
5. *COPAXONE® prescribing information. Teva Neuroscience, Inc.
7. *COPAXONE® prescribing information. Teva Neuroscience, Inc.

3-467-1338
I20573
Rev 8 10/2002

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3-467-1338
I20573
Rev 8 10/2002
The Steering Committee acknowledges the exhibitors’ participation in ACTRIMS-ECTRIMS 2002. Stop by and visit exhibitors during the following hours:

- Wednesday, September 18 from 1 pm–5 pm
- Thursday, September 19 from 8 am–5 pm
- Friday, September 20 from 8 am–5 pm

**GOLD SPONSOR EXHIBITORS**

**Biogen**  **Booth 12**
Biogen, Inc., winner of the US National Medal of Technology, is the world’s oldest independent biotechnology company and a recognized leader in the field of multiple sclerosis research. Biogen produces and markets AVONEX® (Interferon beta-1a), the leading treatment for relapsing forms of multiple sclerosis.

**Biogen/Elan**  **Booth 13**
Biogen, Inc. and Elan Corporation have established a worldwide, collaboration to develop, manufacture, and commercialize Antegren® (natalizumab), the first Selective Adhesion Molecule (SAM) Inhibitor. Natalizumab, a humanized monoclonal antibody binds to the cell surface receptors known as alpha-4-beta-1 (VLA-4) and alpha-4-beta-7 integrins, which are believed to play an important role in the trafficking of mononuclear cells, such as lymphocytes, into sites of inflammation. Natalizumab is currently in Phase III clinical trials for MS and Crohn’s Disease and further trials could determine its potential in the treatment of a range of autoimmune diseases.
Schering AG Germany / Berlex  Booth 11

Betaseron® was the first therapy approved in the United States to treat relapsing-remitting multiple sclerosis. Berlex has filed a supplemental (sBLA) Biologics License Application to expand the indication of Betaseron® to include secondary progressive MS. A new room-temperature formulation of Betaseron® is now available. Betaseron® is the first and only therapy available as a room-temperature formulation (25°C/77°F) for relapsing-remitting MS, providing a convenient option for MS patients in the United States.

Serono  Booth 1

Rebif (interferon beta-1a) was approved for sale in the United States on March 7, 2002, for the treatment of relapsing forms of multiple sclerosis based upon the results of two large multi-center studies. Rebif provides significant treatment benefits for people with relapsing forms of MS by decreasing the frequency of relapses and delaying the accumulation of physical disability. Efficacy in chronic progressive MS has not been established. Rebif is available in ready-to-use, pre-filled syringes and can be administered using Rebiject, an autoinjector developed exclusively for use with Rebif. For more information on Rebif visit www.Rebif.com or call MS LifeLines at 877-44-REBIF.

Teva Neuroscience  Booth 5

Teva Neuroscience invites you to visit our booth to discuss Copaxone (glatiramer acetate for injection). We will also be discussing MSWatch, the first fully integrated, interactive disease management Web application for people with Multiple Sclerosis. MSWatch is available free of charge to all MS patients and their health care providers.

ASSOCIATION AND COMMERCIAL EXHIBITORS

Arnold Publishers  Booth 10

Multiple Sclerosis is published by Arnold Publishers and is now in its 9th year. It focuses on the etiology and pathogenesis of demyelinating and inflammatory diseases of the central nervous system and on the application of such studies to scientifically based therapy. Multiple Sclerosis is a vital journal for research in the following areas: clinical neurology; myelin chemistry; neuroimaging; pathobiology of the blood/brain barrier; glial pathobiology/myelin repair; pathology; epidemiology; therapeutics; genetics; immunology; and virology. Editor-in-Chief, Donald H Silberberg, Department of Neurology, University of Pennsylvania School of Medicine, USA. For more information, visit www.multiplesclerosisjournal.com.

Cephalon  Booth 2

Cephalon, Inc., headquartered in West Chester, PA., is an international biopharmaceutical company dedicated to the discovery, development and marketing of products to treat neurological disorders, sleep disorders, cancer and pain. The Company currently markets three products in the United States: PROVIGIL® (modafinil) Tablets [C-IV] for the treatment of excessive daytime sleepiness associated with narcolepsy, which is being developed for other potential uses; GABITRIL® (tiagabine hydrochloride) for the adjunctive treatment of partial seizures associated with epilepsy; and ACTIQ® (oral transmucosal fentanyl citrate) for the treatment of breakthrough cancer pain.

Consortium of Multiple Sclerosis Centers  Booth 7

The Consortium of Multiple Sclerosis Centers (CMSC) is the largest organization of MS healthcare professionals in the world. Our membership includes MS Centers, Clinics, VA members, and individual healthcare providers. The CMSC has established standards of care in MS that are being adopted worldwide. CMSC/NARCOMS, the North American Research Committee on MS, facilitates multi-center studies and clinical trials through its web site and registry of 23,000 patients. Our journal is The International Journal of MS Care. The Foundation of the CMSC provides funds to train healthcare professionals in research and care in MS.
Serono is pleased to bring you the Living with MS Art and Music Festival.

This therapeutic happening is an exhibition of American and international art created by patients, neurologists, and nurses in the MS community.

**Evening festivities include:**

- Gallery viewing and reception
- Buffet dinner served on Waterside Boulevard
- Jazz concert under the Pavilion on Pier Six

Please join us at 7:00 PM on Pier Six immediately following the Satellite Symposium on Friday, September 20, 2002.

*Please visit us at booth #1 for more information.*
**EDMUS** Booth 4
The European Database for Multiple Sclerosis (EDMUS) is a standardised computerised databasing system which has been conceived within consecutive European Concerted Actions on Multiple Sclerosis (MS) since 1990. EDMUS is a working tool available for clinical and research purposes. It is the result of joint reflections of clinicians and researchers from the whole European Union, all involved in MS. A specific Steering Committee with at least one delegate from each country of the European Union has been set up for this purpose. Today the EDMUS system is established in more than 190 centers over 26 countries. It is used for the clinical follow-up of patients, independent research projects and collaborative multicenter studies where a “common language” is mandatory. A new version of the software has been developed in order to come up to all users’ expectations.

**European Charcot Foundation** Booth 8
The European Charcot Foundation started in 1990 as the legal carrier of a Concerted Action in Multiple Sclerosis (MS) Research funded by the European Communities. From 1994 on the European Charcot Foundation continued as a non-profit Foundation, supported by private organisations, National MS Societies and Industries. Its working base in Europe now consists of more than 550 institutions and 1700 investigators. With their dedication the Foundation wants to realize a European dimension in MS Research and capitalize on the great resources of European co-operation and co-ordination to overcome this debilitating disease.

**Multiple Sclerosis International Federation** Booth 6
The Multiple Sclerosis International Federation (MSIF) was established in 1967 and links the work of its 42 national Member Societies worldwide. It is committed to working with these Societies and with the international research community to eliminate MS and its devastating effects. The MSIF also speaks out on a global level for those affected by MS.

**National Multiple Sclerosis Society** Booth 9
Through our Professional Resource Center, the NMSS provides timely and expert information to physicians and other healthcare professionals involved in the care of people with MS and their families. Contact us for information and consultation about the disease and its management; library and literature search services; information about insurance and long-term care; continuing education opportunities; and consultation on the development of National MS Society-affiliated clinical facilities. For further information, contact the PRC by phone at 1-866-MS-TREAT, by e-mail at MD_info@nmss.org, or visit us online at www.nationalmssociety.PRC/asp.

**The Sylvia Lawry Centre for Multiple Sclerosis Research (SLCMSR)** Booth 6
The SLCMSR was founded in February 2001 at the Technical University of Munich under the direction of Prof. Albrecht Neiß and sponsored by the Multiple Sclerosis International Federation (MSIF). Using the combination of computer science, mathematics and medicine, it aims to make future development of MS therapies faster and less costly. This will be achieved by identifying clinical and MRI markers, which will be more reliably predictive of the future course of MS than those so far identified, using mathematical models based on an unequalled collection of placebo data from clinical trials and natural history data. It is an excellent example of what can be done when industry and academics work together.

**WebMD** Booth 3
WebMD Corporation provides a comprehensive suite of information, transaction and technology solutions that help consumers, physicians, providers and other participants navigate the complexity of the healthcare system. Our products and services promote informed decision-making, increased efficiency and, ultimately, higher quality patient care at a lower cost. WebMD Corporation consists of three divisions, each a leader in their respective field: WebMD Envoy, WebMD Medical Manager and WebMD Health.
NEW ROOM-TEMPERATURE FORMULATION REFRIGERATION-FREE BETASERON® (Interferon beta-1b)

For ambulatory patients with relapsing-remitting multiple sclerosis (MS) to treat clinical exacerbations...

Carol—Betaseron user since 1994.

Serious side effects include depression, suicide, suicidal ideation, and injection-site necrosis (skin breakdown, drainage of fluid, and tissue destruction), which has been reported in 5% of patients in a controlled MS trial. The necrotic lesions are typically 3 cm or less in diameter, but larger areas have been reported, and they may occur at single or multiple sites. Patients should be advised of the importance of rotating areas of injection with each dose, and of consulting with their physician if they experience any of the above signs or symptoms. (See WARNINGS, PRECAUTIONS, and ADVERSE REACTIONS sections of the full prescribing information.)

Common side effects of Betaseron therapy include flu-like symptoms, shortness of breath, menstrual disorders, and injection-site reactions; redness, pain, swelling, and blue-black discoloration have been reported.

Please see full prescribing information on adjacent page.

Betaseron is a registered trademark and the Design and MS Pathways are service marks of Berlex Laboratories, Inc.
Deliver NEW Convenience that Helps Improve Compliance —
NEW Refrigeration-Free Betaseron is the only therapy that can be stored and transported at room temperature — and as always, with a neutral pH of approximately 7.4

Deliver the Full Effect With Frequent Dosing — taken every other day subcutaneously, Betaseron (250 mcg) is specifically designed to maintain a constant effect on biological response markers

Deliver Manageability — common side effects are usually easily managed and often improve over time

Deliver the Full Support of MS Pathways® — helps you assist your patients in managing their therapy through a 24/7 toll-free hot line that features MS-specialized registered nurses
**Betaseron® Interferon beta-1b**

**DESCRIPTION**
Betaseron® interferon beta-1b is a purified, sterile, lyophilized/protein product produced by recombinant DNA techniques and formulated for subcutaneous use. Interferon beta-1b is manufactured by bacterial fermentation of a strain of E. coli that bears a genetically engineered plasmid containing the gene for human interferon beta. The native gene was obtained from human fibroblasts and is earth in a strain of E. coli that is a high-producing strain that has 160 times more interferon beta-1b than an unmodified E. coli. The approximate molecular weight of interferon beta-1b is 20,000 daltons. It does not contain the carbohydrate side chain found in the natural material.

**Clinical Trials:**
Controlled clinical investigations of 2 years duration. The study enrolled MS patients, aged 18 to 50, who were ambulatory (Kurtzke expanded disability status scale [EDSS] of 0–5.5). Betaseron was compared to placebo. Important inclusion criteria were 1) recent clinical deteriorations of 20 to 60% in Expanded Disability Status Scale (EDSS) scores and 2) CRF form, 1200 mg of Betaseron in 2 vials to be self-injected subcutaneously over 2 days. Based on the results of the 240 randomized patients was evaluated.

Patients who met the following criteria were included in the study:
- Recent clinical deterioration of 20 to 60% in Expanded Disability Status Scale (EDSS) scores
- CRF form, 1200 mg of Betaseron in 2 vials to be self-injected subcutaneously over 2 days. Based on the results of the 240 randomized patients was evaluated.

**Pharmacokinetics**
Betaseron® is a family of naturally occurring proteins, which have molecular weights ranging from 15,000 to 21,000 daltons. Three interferons are known to be found in humans: interferons-α, interferons-β, and interferons-γ. Interferon beta-1b is a highly purified protein that has 165 amino acids and an approximate molecular weight of 20,000 daltons. It does not contain the carbohydrate side chain found in the natural material.

**Safety and Tolerability**
Betaseron® is contraindicated in patients with a history of hypersensitivity to natural or recombinant interferon beta, Albumin Human USP, or any component of the formulation.

**WARNINGS**
- Allergic Reaction: A 4.5% incidence of serious or life-threatening allergic reactions occurred in the Betaseron study. Patients should be instructed in injection techniques to assure the safe self-administration of Betaseron. (See PRECAUTIONS, Laboratory Tests, Patients should be instructed in injection techniques to assure the safe self-administration of Betaseron. (See PRECAUTIONS, Laboratory Tests,
- Fatigue: Fatigue is common with interferon treatment. Patients should be instructed in injection techniques to assure the safe self-administration of Betaseron. (See PRECAUTIONS, Laboratory Tests,
- Hepatic Transaminase: Hepatic transaminase levels (SGOT/SGPT) may rise above upper limits of normal, but severe liver injury has not been reported. (See PRECAUTIONS, Laboratory Tests,
- Renal Function: Renal function may be impaired in patients with MS. (See PRECAUTIONS, Laboratory Tests,
- Renal Function: Renal function may be impaired in patients with MS. (See PRECAUTIONS, Laboratory Tests,

**CLINICAL PHARMACOLOGY**
Interferon beta-1b is manufactured by bacterial fermentation of a strain of E. coli. The mechanisms by which Betaseron exerts its actions in multiple sclerosis patients are not well understood, but the most likely cause of the disease-modifying properties of interferon beta-1b is mediated through its interaction with specific cell receptors that bear a genetically engineered plasmid containing the gene for human interferon beta. The native gene was obtained from human fibroblasts and is earth in a strain of E. coli that is a high-producing strain that has 160 times more interferon beta-1b than an unmodified E. coli. The approximate molecular weight of interferon beta-1b is 20,000 daltons. It does not contain the carbohydrate side chain found in the natural material.

**PRECAUTIONS**
**Injection**
Injection Site Reactions: Reaction at injection sites were recorded in 372 study patients during a 3-year period. All the patients received Betaseron® in the 0.05 mg and 0.1 mg subcutaneous groups. Intra-lesional necrosis has occurred. (See PRECAUTIONS, Laboratory Tests, Intra-lesional necrosis has occurred. (See PRECAUTIONS, Laboratory Tests, Intra-lesional necrosis has occurred. (See PRECAUTIONS, Laboratory Tests, Intra-lesional necrosis has occurred. (See PRECAUTIONS, Laboratory Tests,

**CONTRAINdications**
Betaseron® is contraindicated in patients with a history of hypersensitivity to natural or recombinant interferon beta, Albumin Human USP, or any component of the formulation.

**ADVERSE REACTIONS**
Flu-like symptoms are common following initiation of therapy with Betaseron®. In the controlled MS clinical trial, acetaminophen was permitted and was effective in reducing these symptoms. In a study of 120 patients, 94% of patients reported flu-like symptoms. In a study of 120 patients, 94% of patients reported flu-like symptoms. In a study of 120 patients, 94% of patients reported flu-like symptoms. In a study of 120 patients, 94% of patients reported flu-like symptoms. In a study of 120 patients, 94% of patients reported flu-like symptoms.
Betaseron® (interferon beta-1b) is not ergotamine when associated for gynecology in the Ames bacterial test in the presence of absence of neutrophils.

**Impairment of fertility:** Studies in mice revealed at doses up to 0.05 mg/kg (21 times the recommended human dose based on body surface area comparison) a normal fertility. Female fertility in rats and an apparent adverse effect on the menstrual cycle and on associated hormonal profiles (in peakergonne and estradiol) when administered over 3 consecutive menstrual cycles. The only adverse effect observed in human does not follow. Effects of Betaseron on normal cycling human females are not known. There are no data on the use of Betaseron in pregnant women.\*\*

**Teratogenic effects:** Pregnancy (Category C). Betaseron was not teratogenic at doses up to 0.05 mg/kg (21 times the recommended human dose based on body surface area comparison) and 0.5 mg/kg (43 times the recommended human dose based on body surface area comparison). The extrapolability of animal doses to humans is not known. Lower doses were not studied in mice. Spontaneous abortions during treatment were not reported in the Betaseron group on gestation day 20 to 70 did not cause teratogenic effects, however, it is not known if teratogenic effects exist in humans. There are no adequate and well-controlled studies in pregnant women. The patient should be apprised of the potential to cause harm to the fetus and should be recommended that this pregnancy be terminated. Nursing mothers: it is not known whether Betaseron is excreted in human milk. Since many drugs are excreted in human milk and the potential for serious adverse reactions in nursing infants from Betaseron, a decision should be made as to whether to discontinue nursing or discontinue the drug, taking into account the importance of the drug to the mother.

**Pediatric use:** Safety and efficacy in children under 18 years of age have not been established.

**ADVERSE REACTIONS**

Experience with Betaseron in patients with MS is limited to a total of 147 patients at the recommended dose of 0.25 mg three times a day over 12 months and 300 patients for up to 30 months in the randomized clinical trial. Common adverse reactions that were significantly associated (p<0.05) with the 0.25 mg Betaseron-treated group. Only inflammation, pain, injection site reactions, and injection site reactions were reported as severe events (see WARNINGS AND PRECAUTIONS). The incidence of injection site reactions is 24% at 1.25 mg and 6% at 0.25 mg Betaseron

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**Cardiovascular System**

| Malignant | 1% | 1% | 1% |
| Malignancy | 1% | 1% | 1% |
| Malignancies | 1% | 1% | 1% |
| Hypertension* | 1% | 1% | 1% |
| Tachycardia | 1% | 1% | 1% |
| Peripheral vascular disorder | 1% | 1% | 1% |
| Mitral valve prolapse | 1% | 1% | 1% |

**Digestive System**

| Diarrhea | 2% | 4% | 1% |
| Constipation | 2% | 4% | 1% |
| Vomiting | 1% | 1% | 1% |
| Gastrointestinal disorder | 1% | 1% | 1% |

**Endocrine System**

| Gaster | 2% | 2% | 1% |

**Heme and Lymphatic System**

| Lymphopenia (less than 1000/mm³) | 0% | 15% | 0% |
| ANC < 1500/mm³ | 0% | 13% | 0% |
| WBC < 3000/mm³ | 0% | 14% | 0% |

**Metaplasia**

| Neutrophilic | 20% | 20% | 1% |
| Monocytes | 20% | 20% | 1% |
| Lymphocytes | 20% | 20% | 1% |

**Musculoskeletal System**

| Myalgia | 24% | 44% | 13% |
| Malignant | 15% | 10% | 1% |

**Nervous System**

| Disorder | 24% | 36% | 1% |
| Hyperactive | 12% | 15% | 1% |
| Anxiety | 12% | 15% | 1% |
| Somnolence | 12% | 15% | 1% |
| Confusion | 12% | 15% | 1% |
| Sleep disorder | 12% | 15% | 1% |
| Dizziness | 12% | 15% | 1% |

**Respiratory System**

| Rhinitis | 20% | 30% | 1% |
| Laryngitis | 20% | 30% | 1% |

**Skin and Appendages**

| Sweating* | 11% | 23% | 1% |
| Acne | 11% | 23% | 1% |

**SPECIAL SENSORY**

| Olfactory | 10% | 11% | 1% |
| Tactile | 10% | 11% | 1% |

**Urogenital System**

| Urinary infection | 11% | 18% | 1% |
| Urinary incontinence | 11% | 18% | 1% |
| Urinary frequency | 11% | 18% | 1% |

**Vascular System**

| Vascular disorder | 11% | 18% | 1% |
| Malignant | 11% | 18% | 1% |
| Myocardial infarction | 11% | 18% | 1% |

**Other**

| Gastrointestinal disorder | 2% | 2% | 1% |

**Drug Abuse and Dependence**

No evidence or experience suggests that abuse or dependence occurs with Betaseron® (interferon beta-1b); however, the risk of depen- dence can not be systematically evaluated.

**Drug Interactions**

The recommended dose of Betaseron for the treatment of ambulatory relapsing remitting MS is 0.25 mg injected subcutaneously every other day. Limited data regarding the activity of these agents are presented below (see CLINICAL PHARMACOLOGICAL, Clinical trial).

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- Lengyel P. Annu Rev Biochem 1982; 51: 251
- Lengyel P. Annu Rev Biochem 1982; 51: 251
The Program Committee congratulates authors whose abstracts were selected for poster display at ACTRIMS-ECTRIMS 2002. Posters are on display from 8 am–5 pm. Presenting authors will stand by their posters from Noon–2 pm.

Further information regarding poster room assignments will be available onsite.

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**LB1**

**VALIDATION OF DIAGNOSTIC MRI CRITERIA FOR MS AND RESPONSE TO TREATMENT WITH INTERFERON-BETA-1A**


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- European Charcot Foundation, Nijmegen, Netherlands;
- Karl-Franzens Universität, Graz, Austria;
- University of Turin, Turin, Italy;
- Université de Rennes, Rennes, France;
- Hospital Carlos Haya, Malaga, Spain;
- C.H.U. de Charleroi, and Hôpital Erasme, Brussels, Belgium;
- Rigshospitalet, Copenhagen, Denmark;
- Quintiles Pty Limited, Sydney, Australia

**Background:** In the recently adopted diagnostic criteria for MS by McDonald, the modified criteria of Barkhof have been adopted.

**Objectives:** To prospectively test the validity of the modified Barkhof criteria and their predictive value for IFN-beta-1a treatment response in the ETOMS study

**Methods:** The ETOMS study was a randomised, double-blind, placebo-controlled study of IFN-beta-1a (Im.) once weekly in 309 patients with a first episode consistent with demyelinating disease. Baseline MRI was assessed for the presence of gadolinium-enhancement (or 9 T2 lesions), juxtacortical, infratentorial, and 3 periventricular lesions. Conversion to CDMS was used as the outcome parameter.

**Results:** Conversion to CDMS occurred in 41% of patients with gadolinium-enhancement on MRI. The frequency of 9 T2 lesions versus 11% of those without (p=0.017); similar comparisons were 44% vs. 31% for infratentorial (p=0.026), 40% vs. 35% for juxtacortical (p=0.413), and 41% vs. 17% for more than 3 periventricular lesions (p=0.034). For the cumulative number of modified Barkhof criteria, the rate of conversion to CDMS was 25% for 1 abnormal criterion, rising to 47% with 4 abnormal criteria. For a cut-off of 3 positive criteria, the hazard ratio for time to CDMS was 2.3 (95% CI 1.1-7.4, p=0.016). While the effect of treatment seemed most evident in patients with 4 abnormal criteria, statistically significant treatment by variable interaction could not be detected. However, the number of patients needed to treat decreases from 50 with 2 or less criteria to 5.6 with 4 positive criteria.

**Conclusions:** This study confirms the validity of the modified Barkhof criteria for conversion to CDMS, and suggests that treatment with IFN-beta-1a is more cost-effective in patients with more abnormal criteria.

Disclosure: Most authors were consultants to Serono

**Funding:** Supported by the European Charcot Foundation and Serono

**LB2**

**ANTI-MOG ANTIBODIES PREDICT EARLY CONVERSION TO CLINICALLY DEFINITE MS IN PATIENTS WITH A FIRST DEMYELINATING EVENT.**


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- Neurology, County Hospital, Villach, Austria;
- Biostatistics, University of Innsbruck, Innsbruck, Austria

**Background:** 90% of MS patients present at onset with a clinically isolated syndrome (CIS). Although up to 80% of these patients will convert to clinically definite MS (CDMS), the further MS disease course is unpredictable at onset for individual patients.

**Objectives:** New neuropathological findings, e.g. antibody-mediated demyelination, and the concept of epitope spreading in the early disease phase, prompted us to investigate whether the presence of serum anti-MOG and anti-MBP antibodies (abs) in patients with CIS predicts the further disease course.

**Methods:** 103 consecutive patients with a CIS, confirmed by MRI and positive oligoclonal bands in CSF, were included and followed for at least 12 months. Anti-MOG and anti-MBP abs were measured as previously described (Reindl et al, 1999).

**Results:** 73 females and 30 males (mean age at disease onset: 32.0 years; mean disease duration: 50.9 months; range 12–96 months). 22 patients (21%) had serum abs against MOG and MBP. 42 (41%) were seropositive for anti-MOG abs only, and 39 (38%) were seronegative. Relapses occurred in only 9 (23%) seronegative patients, but in 95% of patients with abs against MOG and MBP. Seronegative patients had their first relapse after a mean relapse free interval of 45.1 months (range 25–83 months). In contrast, patients with initial seropositivity for anti-MOG and anti-MBP abs developed their first relapse after only 7.5 months (range 1–18 months, P=0.001). Quantitation of MRI showed higher mean numbers of T2 and Gd-enhancing T1 lesions in patients with anti-MOG and anti-MBP abs compared to seronegative patients. However, the number of MRI lesions varied in individual patients, irrespective of their antibody status, from 2 to 9 T2 lesions and 0 to 4 Gd-enhancing T1 lesions.

**Conclusions:** Analysis of antibodies against MOG and MBP in patients with CIS represent a rapid, expensive and precise method to identify patients with either a high or low risk for early conversion to CDMS. This may have implications for counseling and management in patients with a first demyelinating event suggestive of MS.

Disclosure: T Berger has nothing to disclose.

**Funding:** This work was supported by a grant of the Austrian Federal Ministry of Science (Nr. GZ 70.059/2-P/4/99).

**LB3**

**NEUROREHABILITATION IN MULTIPLE SCLEROSIS CONtributes to FUNCTIONAL RECOVERY ACCOMPANIED BY CHANGES OF BRAIN ACTIVITY ON fMRI—PRELIMINARY RESULTS.**

Rasova K, Krasensky J, Havrdova E, Oberhuber J, Zalisova M, Seidl Z

Department of Neurology, Charles University

**Background:** Although MS is an inflammatory demyelinating disease, which can lead to the axonal injury and loss, neurorehabilitation may contribute to functional recovery accompanied by the changes in brain activity.

**Objectives:** To show changes in brain activity on fMRI and their correlation with functional recovery.

**Methods:** 18 outpatients with MS were evaluated before and after individualized neurorehabilitation treatment (two sessions per week, 30 weeks) for impairment (EDSS), disability (Bi), handicap (ESS), quality of life (MSQoL) and amplitude of signal in the primary sensorimotor cortex (ASPSMC) using serial fMRI during the performance repetitive index-thumb opposition.

**Results:** There were 6 men and 12 women, EDSS was 4.19, age 41.11 yrs and illness duration 11.5 yrs. 8 patients had relapsing-remitting, 4 primary progressive and 6 secondary progressive MS. The therapy led to functional recovery and positively influenced the impairment (EDSS from 4.19 to 3.63; p<0.1), the disability (Bi from 94.16 to 98.05; p<0.03), the handicap (ESS from 7.30 to 4.25; p<0.05) and quality of life (MSQoL from 152.6 to 161.13; trend shown). Functional recovery was accompanied by changes in ASPSMC. There was a trend towards decreased ASPSMC after therapy (ASPSMC for right hand from 7.82 to 7.20%, for left hand from 8.61 to 7.71%). We found two different responses to therapy: in two thirds of patients the ASPSMC decreased, while in one third of patients it increased. We have found no relationship between functional recovery and changes in the brain activity. It was shown that when ASPSMC in left hand changed, it changed in right hand as well during the performance of paradigm (correlation coefficient 0.56). The therapy was not aimed at improving function of the hands, but control of the whole body. Nevertheless, the function of the hands and ASPSMC during the performance of the paradigm changed. It seems that neurorehabilitation influences the function of the whole brain.

**Conclusions:** There is very little scientific basis for the therapy that is designed to help damaged brain circuits recover. These preliminary results show that neurorehabilitation in MS contributes to functional recovery and can be accompanied by changes of brain activity.

Disclosure: K Rasova has nothing to disclose.

**LB4**

**TIGHT JUNCTION ABNORMALITY IN MS AFFECTS ALL CALIBRES OF VESSEL AND CORRELATES WITH LESION ACTIVITY.**

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- Neuropathology, Royal Victoria Hospital, Belfast, Northern Ireland, United Kingdom

**Background:** Increased blood-brain barrier (BBB) permeability observed in MS has been linked to pathological change in the tight junctions (TJ) and vesicular transport of vascular endothelium.

**Objectives:** This study quantifies the pathological changes in TJs which we have recently reported in MS, including their uneven distribution and the relation between abnormal TJ and BBB leakage.

**Methods:** Frozen sections from plaque and normal appearing white matter (NAWM) in 14 post-mortem (PM) cases of MS were studied together with white matter from 6 neurological and 5 normal controls. Using single and double immunofluorescence and confocal microscopy the TJ-associated proteins zonula occludens-1 (ZO-1) and occludin were examined across lesion types and tissue categories, and in relation to fibrogenic leakage. Confocal image datasets were analysed for 2198 MS and 1062 control vessels.

**Results:** Significant differences in the extent of TJ abnormalities (i.e. beading, interruption, absence or redistribution of fluorescence signal, separation or opening of junctions) were detected between the different lesional types in MS and between MS and control white matter. They were frequent in oil-red O (ORO) ‘active’ plaques, affecting 42.5% of vessels, but less frequent in ORO ‘inactive’ plaques (22.8%) or NAWM (13.1%) and in both normal (3.9%) and neurological controls (9.3%). A similar pattern was found irrespective of the size of vessels examined. In both NAWM and inactive lesions, dual-labeling showed that those with the most TJ abnormality had the greatest fibrogenic leakage. This was most apparent in active lesions where 41% of vessels showed severe leakage.

**Conclusions:** TJ abnormality affects vessels of all sizes, suggesting a diffusible chemical (cytokine) cause. It occurs in lesional and non-lesional white matter, being most severe where there is evidence of active demyelination. Disruption of TJs, affecting both paracellular and transcellular pathways probably contributes to the BBB leakage detected in this study. The finding of TJ abnormality and BBB leakage in ‘inactive’ lesions points to a failure of effective and complete TJ repair or to the continuation of a pathological process. In NAWM it suggests either pre-lesional change or white matter damage secondary to remote lesions.

Disclosure: J Kirk has nothing to disclose.

**Funding:** JP is supported by The Irish Brain Research Foundation. Pilot studies were supported by the MS Society of GB & NI.
LB5
SINGLE CENTRE, DBPC, RANDOMISED TRIAL OF INTERFERONβ-1B IN PRIMARY PROGRESSIVE AND TRANSITIONAL PROGRESSIVE MULTIPLE SCLEROSIS: AN EXPLORATORY PHASE II STUDY.


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Background: Beneficial effects of interferonβ-1b have been shown only for patients in the relapsing phase of MS as its role in the treatment of SPMS patients still remains controversial. The single phase II randomized controlled trial on PPSMS using IFNβ-1a (M) shows no significant treatment effect on EDSS though some effect on T2 lesion load.

Objectives: To investigate safety and efficacy hints of interferonβ-1b given to patients with primary and transitional progressive multiple sclerosis (PPPSMS and TPPMS).

Methods: 73 patients (49 PPSMS, 24 TPPMS) with EDSS scores of 3.0 to 7.0, were randomized to receive 8 million IU of IFNβ-1b or placebo every other day, subcutaneously for 2 years. Safety parameters including the Ashworth spasticity, Krupp fatigue and Depression Inventory scales and blood tests were performed three monthly. Clinical outcomes (EDSS and MS Functional Composite—MSFC) were also performed three monthly and the Sickness Impact Profile six monthly. MRI measures (T2 and T1-weighted brain lesion load, brain parenchymal fraction, active lesions, spinal cord atrophy, HTR and spectroscopy) and neuropsychological assessment (BRINB) were done annually.

Results: Adverse events significantly associated with IFNβ-1b included injection-site reaction, flu-like symptoms and lymphopenia. One patient on placebo died of pulmonary infection. In all, 96% of the patients reached study end and 93% completed the treatment period.

Conclusions: IFNβ-1b is safe in treating patients with PPSMS and TPPMS. Our study seems to be the first indicating a beneficial effect of IFNβ-1b in these patients.

Disclosure: X Montalban has nothing to disclose.

Funding: Trial supported by Schering España SA (of Schering AG, Germany)

LB6
SUCCESSFUL TREATMENT WITH IFN-β1B IN RR MS PATIENTS IS ASSOCIATED WITH AN INCREASE IN THE NUMBER OF IL-10 PRODUCING (REGULATORY) CD4+ T CELLS.

van Boxel-Dezaire A, Smits M, Uitdehaag B, Polman C, Nagelkerken L

<Division of Immunological and Infectious Diseases, TNO Prevention and Health, Leiden, Netherlands; Department of Neurology, Vrije Universiteit Medical Center, Amsterdam, Netherlands>

Background: Although IFN-β is now widely used for treatment of MS, its mode of action still remains unclear; recent studies do not support a shift in the Th1/Th2 balance. In vitro studies show that type I interferons induce the differentiation of Regulatory-1 (Tr1) cells and the survival of CD4+CD25+ T cells, which are both recently described suppressor T cells able to produce IL-10.

Objectives: The aim of the present study was to investigate whether IFN-β1b therapy induces IL-10 in a general fashion or in a specific (regulatory) T cell subset only. In addition, it was evaluated whether differential effects on IL-10 production by peripheral blood mononuclears, CD4+ or CD8+ T cells could be correlated with clinical efficacy of IFN-β1b treatment.

Methods: Based on EDSS-progression and the number of relapses and steroid interventions in the 2 years before initiation of IFN-β1b treatment compared with those in the 2 years after initiation of treatment, 24 RR MS patients were classified as responders (15) and non-responders (9). Using intracellular cytokine staining techniques, the effect of IFN-β1b after 0, 3 and 6 months of treatment was studied on the number of IL-10 producing CD4+ T cells, CD4+ (CD25+) T cells and monocytes.

Results: Numbers of IL-10 producing CD4+ T cells were significantly decreased prior to treatment. Remarkably, after 3 and 6 months of treatment a significant increase in the number of such T cells could be found in the clinical responders only. In contrast, treatment decreased numbers of IL-10 producing monocytes in both responders and non-responders and did not affect numbers of CD8+ T cells that produced IL-10. In a subgroup of the responders (7 out of 15), the effect of IFN-β1b treatment was also studied on CD4+CD25+ T cells. Notably, a significant increase in the number of IL-10 producing CD4+CD25+ T cells could be observed after 6 months of treatment.

Conclusions: Enhancement of the number of CD4+CD25+ T cells that produce IL-10 may be an important mechanism in the therapeutic effect of IFN-β1b in RR MS.

Disclosure: A van Boxel-Dezaire has nothing to disclose.

Funding: Supported by the Dutch Foundation for the support of MS Research, grant 94-175MS; The Multiple Sclerosis Center for Research and Care, Amsterdam, The Netherlands

LB7
MULTIPLE SCLEROSIS DOCUMENTATION SYSTEM—MSDS 2.0
Eulitz M, Kugel T, Muraro PA, Pette M

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Background: The long and variable course of MS, as well as the individual and unpredictable response to currently available immune-modifying treatments require a detailed and standardized patient record. Furthermore, the systematic collection of data on large cohorts of consecutive patients may help to correlate clinical information with basic research. Although the development of database programs to monitor MS patients started almost thirty years ago, no single application has gained widespread use so far.

Objectives: To develop a modular electronic patient record supporting both the daily care of patients and the collection of specific information on MS.

Methods: MSDS is a SQL (structured query language) database. A multilingual (German, English, Italian and Spanish in preparation), graphical user interface allows easy data input and output. Client-server architecture optimizes MSDS for use in local area networks.

Results: Since its introduction in 12/2001, MSDS has been distributed to 25 hospitals (16 universities). Through MSDS users recorded and managed visiting dates, patient history (complaints, relapses), physical examination findings, and results of blood and CSF chemistry, evoked potentials and MRI. MS diagnosis can be specified according to criteria by Poser (applied automatically), by McDonald and by Lublin/Reingold. Clinical scores calculated by MSDS comprise the EDSS, the MSFC, and the Scirgus NRS. In general, data input is standardized (pull-down menus). Whenever required, specific details can be added as free text. Data consistency is controlled by internal check mechanisms. Multiple reports describe the individual disease course, as well as the local patient population. In addition to these built-in features, individual queries can be designed to retrieve specific information. Correspondence to practitioners is supported by MSDS. Additional features include a pedigree generator, a bio-sample database and a study-protocol editor. To support the building of a national MS registry, the present version of MSDS automatically keeps a consensus minimal data set of each patient up-to-date. Current users have found MSDS user-friendly and well suited to accurately describe the clinical aspects of MS.

Conclusions: MSDS may provide an improved platform for clinical documentation of MS and facilitate international standardization.

Disclosure: M Pette has nothing to disclose.

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   Phone: 410-385-3000
   Fax: 410-895-1900

2. Courtyard by Marriott Inner Harbor
   1000 Alice Anne Street
   Baltimore, MD  21202
   Phone: 443-923-4000
   Fax: 443-923-9970

3. Baltimore Marriott Inner Harbor
   110 South Eutaw Street
   Baltimore, Maryland  21201
   Phone: 410-962-0202
   Fax: 410-625-7892

4. Radisson Plaza Hotel Baltimore Inner Harbor
   20 West Baltimore Street
   Baltimore, MD  21201
   Hotel Phone: 410-539-8400
   Hotel Fax: 410-332-4229

5. Renaissance Harborside Hotel
   202 East Pratt Street
   Baltimore, MD  21202
   Phone: 410-547-1200
   Fax: 410-539-5780

6. Sheraton Inner Harbor Hotel
   300 South Charles Street
   Baltimore, MD  21201
   Phone: 410-962-0300
   Fax: 410-962-8211

7. Wyndham Baltimore Inner Harbor
   101 West Fayette Street
   Baltimore, MD  21201
   Phone: 410-752-1100
   Fax: 410-752-0832
Welcome Reception at the Baltimore Marriott Waterfront Hotel
700 Aliceanna Street, Phone: 410-385-3000
Wednesday, September 18th
7:00 pm–9:00 pm
On opening day, the Steering Committee invites all conference delegates to an informal “Welcome Reception” at the Baltimore Marriott Waterfront Hotel. Meet new friends, renew acquaintances, and congratulate the fine young investigators who presented their research that afternoon.

National Aquarium of Baltimore
Pier 3 at 501 E. Pratt Street, Phone: 410-576-3800
Reception and Buffet Dinner
Thursday, September 19th
7:30 pm–10:30 pm
Begin your evening with a leisurely stroll through the aquarium exhibits, enjoying a glass of wine and selections of hors d’oeuvres along the way. Dazzling tropical fish, a giant Pacific octopus, electric eels, graceful stingrays, playful puffins, two-toed sloths, red-bellied piranhas, poison dart frogs and a giant anaconda are among the more than 10,000 marine and freshwater animals waiting to say hello. The self-guided tour ends with a candlelight dinner buffet.

B&O Railroad Museum
901 W. Pratt Street at Poppleton Street, Phone: 410-752-2490
Reception and Buffet Dinner
Friday, September 20th
7:30 pm–10:00 pm
Affiliated with the Smithsonian Institution, the B&O Railroad Museum is dedicated to the preservation and interpretation of American railroading—especially the historic Baltimore and Ohio (B&O) line. You’ll enter the museum through Mt. Clare Station, built in 1851, and proceed to the 1884 Baldwin Roundhouse—a 22-sided room with a 136-foot high ceiling! Dinner and dancing are the order of the evening with the opportunity to stroll through some of the locomotive and artifacts exhibits.

Committee Reception and Dinner (by invitation)
Baltimore Museum of Industry
1415 Key Highway at Lawrence Street
Phone: 410-727-4808
Saturday, September 21st
7:00 pm–10:00 pm

Complimentary round-trip transportation will be available for all social events.
Next Year’s Meetings

ACTRIMS 2003
October 19
San Francisco
8th Annual Meeting of the Americas Committee for Treatment and Research in Multiple Sclerosis

ECTRIMS 2003
September 17–20
Milan
19th Congress of the European Committee for Treatment and Research in Multiple Sclerosis

The National Multiple Sclerosis Society proudly introduces the

Professional Resource Center

The National MS Society has long been a resource for health professionals providing care to people with multiple sclerosis. The new Professional Resource Center, which houses the most comprehensive library of MS information in the world, offers multidisciplinary expertise on MS disease process and management, opportunities for clinical affiliation, and a range of educational programs and materials.

Physicians are invited to consult via email with MS specialist colleagues who serve on our Medical Advisory Board:

MD_info@nmss.org

Allied health professionals are invited to consult via email with MS specialist colleagues:

HealthProf_info@nmss.org

Questions are also welcome at our toll-free number:

1-866-MS-TREAT (678-7328)

Visit our website: nationalmssociety.org/PRC.asp
### Travel Information

#### Reservation Assistance
Targa Tours can offer discounts with American Airlines and US Airways, and will also search for the lowest available fare on ANY airline serving Baltimore. A US$20 service fee applies.

Call toll-free: 1-800-756-7957 (From outside the USA or within the Chicago metro area, call 312-541-0780)
Fax: 312-541-0783
E-mail: targatours@aol.com
On-line: targatours.com

#### To the Airport

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<th>Service</th>
<th>Phone Number</th>
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<tbody>
<tr>
<td>The Airport Shuttle, Inc.</td>
<td>410-381-2772</td>
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<tr>
<td>Baltimore Airport Shuttle</td>
<td>410-821-5387</td>
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<tr>
<td>Butler Transportation</td>
<td>410-732-5098</td>
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<tr>
<td>BWI Airport Shuttle</td>
<td>800-258-3826</td>
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#### Taxi and Limousine Services

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<td>Yellow Transportation</td>
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<td>American Limousines, Inc</td>
<td>410-522-0400</td>
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<tr>
<td>Carey Limousine/Baltimore</td>
<td>410-880-0999</td>
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<tr>
<td>Prime Time Sedan and Limo Service</td>
<td>443-562-0067</td>
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#### Trains

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<tr>
<td>MARC Commuter trains (Baltimore/Washington)</td>
<td>410-539-5000</td>
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<tr>
<td>AMTRAK</td>
<td>800-872-7245</td>
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#### Visitor Services

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<tr>
<td>Baltimore Tickets</td>
<td>888-225-8849</td>
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#### Emergency Phone Numbers

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<td>Police</td>
<td>911</td>
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<tr>
<td>Fire</td>
<td>911</td>
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<tr>
<td>Ambulance</td>
<td>911</td>
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7th Annual Meeting of the Americas Committee for Treatment and Research in Multiple Sclerosis

18th Congress of the European Committee for Treatment and Research in Multiple Sclerosis

September 18–21, 2002
Baltimore Marriott Waterfront Hotel
700 Aliceanna Street
Baltimore, Maryland, USA

Tel: 212-476-0465
Fax: 212-661-8735
E-mail: ae2002@nmss.org
www.actrimsectrims2002.nmss.org

Final Program, Abstract Listing and Meeting Information

This program is offered in collaboration with the National Multiple Sclerosis Society