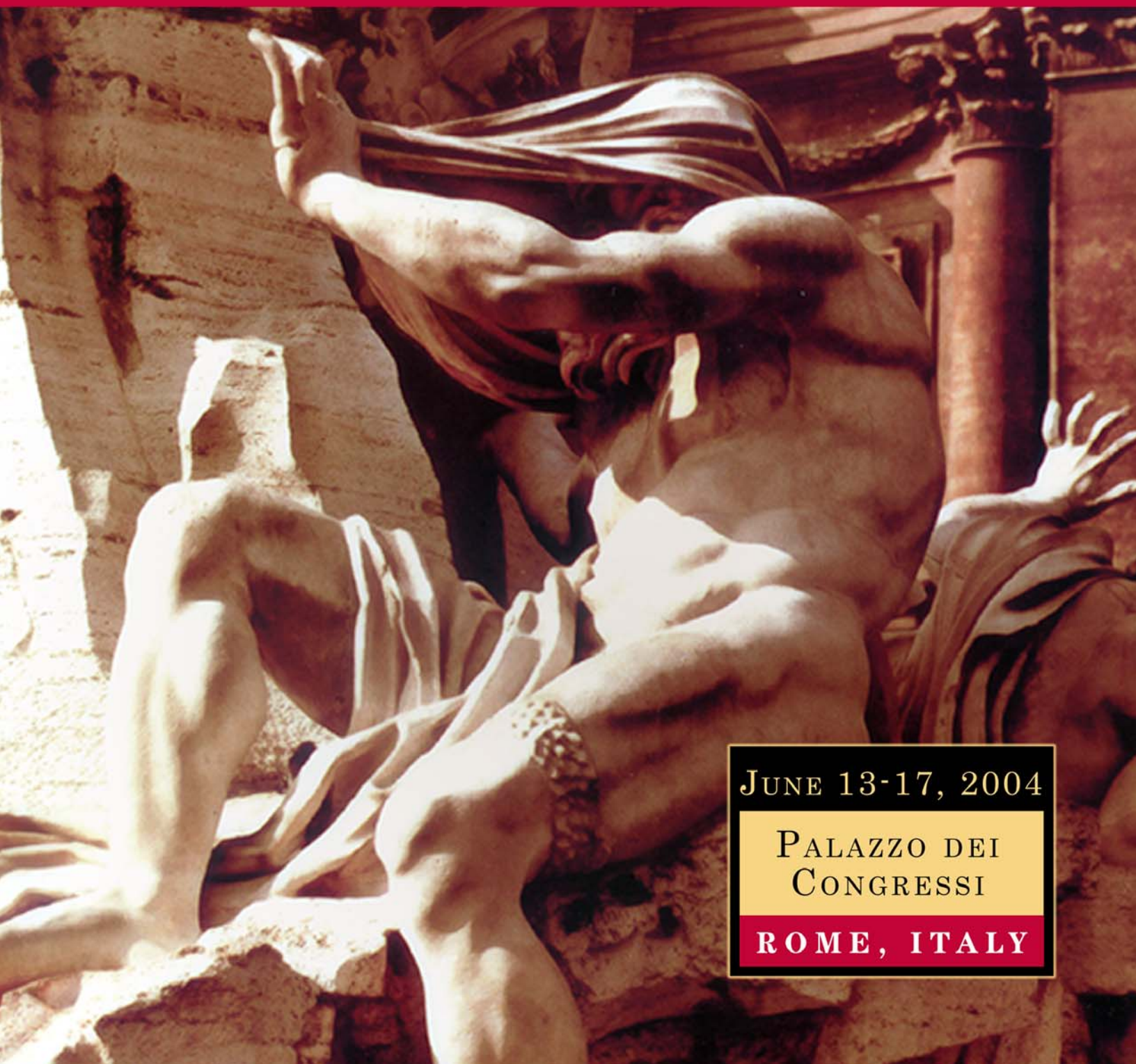
The *Movement* Disorder Society

FINAL PROGRAM

8th International Congress of Parkinson's Disease and Movement Disorders



June 13-17, 2004

Palazzo dei Congressi

ROME, ITALY

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THE CONGRESS IS UNDER THE AUSPICES OF:

The President of the Italian Republic
The Prime Minister of the Italian government
The National Institute of Health (ISS)
The National Research Council (CNR)
The City of Rome
The School of Medicine and Surgery, University of Rome "La Sapienza"
The Italian Society of Neurology

Dear Colleagues,

On behalf of the Officers and International Executive Committee of The *Movement* Disorder Society, welcome to the 8th International Congress of Parkinson's Disease and Movement Disorders.

I would like to thank the faculty and the members of the Congress Scientific Program Committee for this exemplary scientific program and for their contribution to the International Congress.

The International Congress week begins with a variety of Kickoff Seminars, which are supported through unrestricted educational grants from industry. The week continues with a wide array of plenary sessions, parallel sessions, seminars and video dinners. Poster sessions are unopposed and, to further serve our participants, time has also been allotted for 16 poster platform presentations.

I would like to thank the International Congress Oversight Committee and the Congress Organizing Committee for all of their hard work over the past two years. Their dedication in planning has allowed us to offer International Congress attendees the best that Rome has to offer. The social program will include a Sunday Opening Ceremony which will be held on the roof top terrace of the Palazzo dei Congressi. This event offers attendees the opportunity to greet each other while taking in the exquisite tastes and talents of Italy. The Congress Gala Event takes place at the Palazzo Brancaccio, offering a mix of Roman Patrician architecture, as well as enchanting gardens.

In closing, I would like to thank all of the International Congress attendees for their participation in the success of the 8th International Congress.

Sincerely,

C. Warren Olanow
President

INVITATION

Dear Colleagues,

It is my pleasure to welcome you to the 8th International Congress of Parkinson's Disease and Movement Disorders. As one of the world's most beautiful cities, Rome is an ideal venue for the International Congress and I hope that you will enjoy all that the city has to offer.

The photograph on the front page shows the Statue of the Nile, part of the Fountain of the Rivers created in 1651 by Gian Lorenzo Bernini and commissioned by Pope Innocenzo X. The Fountain is located in the middle of Piazza Navona and is one of the finest symbols of Baroque art. Its importance lies in the way the sculptor has fused figures, statues, landscapes, and water into one of his most imposing creations. The mass of rocks and grottoes forming the fountain is surmounted by a tall obelisk. At the four corners are colossal figures of the rivers Danube, Ganges, Nile and Plate, representing the four quarters of the globe.

With this symbolism in mind, I welcome participants from all over the world. The aim of the International Congress is to increase our knowledge in the field of Movement Disorders by sharing global research and perspectives. In line with MDS tradition, the scientific program is informative, comprehensive and innovative, including plenary session lectures, parallel sessions, platform presentations, abstract poster presentations, seminars and video dinners.

On behalf of the Congress Organizing Committee and The *Movement* Disorder Society, welcome to Rome.

Alfredo Berardelli

Professor Alfredo Berardelli
Chair, Congress Organizing Committee

ACKNOWLEDGEMENTS

The *Movement* Disorder Society wishes to acknowledge and thank the following companies for their support:

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ORGANIZATION

The *Movement* Disorder Society (MDS) is an international, professional society of clinicians, scientists, and other healthcare professionals, who are interested in Parkinson's disease, related neurodegenerative and neurodevelopmental disorders, hyperkinetic Movement Disorders, and abnormalities in muscle tone and motor control. The spectrum of clinical disorders represented by the Society includes but is not limited to:

Ataxia
Blepharospasm
Dysphonia
Dystonic disorders
Gait disorders
Huntington's disease
Myoclonus
Parkinson's disease
Spasticity
Tardive dyskinesia
Tics and Tourette syndrome
Tremor

The *Movement* Disorder Society (MDS) was founded in 1985 on the initiative of Professors Stanley Fahn and C. David Marsden, whose leadership and vision guided the expansion of clinical expertise and research in this field. The organization merged in 1988 with the International Medical Society for Motor Disturbances.

PURPOSE, MISSION AND GOALS

Purpose:

The object and mission of the Society shall be to advance the neurological sciences pertaining to Movement Disorders; to operate exclusively for scientific, scholarly and educational purposes; to encourage research; to provide forums, such as medical journals, scientific symposia and International Congresses, for sharing ideas and for advancing the related clinical and scientific disciplines; to encourage interest and participation in the activities of the Society among healthcare and allied professionals and scientists; and to collaborate with other related professional and lay organizations.

Mission and Goals:

To disseminate knowledge about Movement Disorders by:

- Providing educational programs for clinicians, scientists and the general public designed to advance scientific and clinical knowledge about Movement Disorders
- Sponsoring congresses and symposia on Movement Disorders
- Collaborating with other international organizations and lay groups
- Publishing journals, videotapes and other collateral materials committed to high scientific standards and peer review

To promote research into causes, prevention and treatment of Movement Disorders by:

- Using the Society's influence and resources to enhance support for research
- Facilitating the dissemination of information about research
- Encouraging the training of basic and clinical scientists in Movement Disorders and related disorders

To formulate and promote public policy that will favorably affect the care of patients with Movement Disorders by:

- Working with regulatory agencies to assist them in the approval process of safe and effective therapeutic interventions
- Informing the public (media) and patient support groups of new research and therapeutic advances
- Playing a proactive role in the development of policies that affect support of research and patient care
- Developing standards of training in the specialty

MDS OFFICERS

President
C. Warren Olanow, USA

President-Elect
Andrew J. Lees, United Kingdom

Secretary
Andres M. Lozano, Canada

Secretary-Elect
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Treasurer
Wolfgang H. Oertel, Germany

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Past President
Werner Poewe, Austria

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Kapil D. Sethi, USA
Caroline M. Tanner, USA
Marie Vidailhet, France

International Congress Oversight Committee
Chair: Mark Hallett, USA
Wolfgang H. Oertel, Germany
C. Warren Olanow, USA
Werner Poewe, Austria
Eduardo Tolosa, Spain

Congress Scientific Program Committee
Chair: C. Warren Olanow, USA
Co-Chair 2004: Anthony H.V. Schapira, United Kingdom
Co-Chair 2005: Anthony E. Lang, Canada
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Alfredo Berardelli, Italy
Cynthia L. Comella, USA
Bruno Dubois, France
John A. Hardy, USA
Etienne C. Hirsch, France
Joseph Jankovic, USA
Yoshikuni Mizuno, Japan
José A. Obeso, Spain
Olivier Rascol, France
Peter Riederer, Germany
John C. Rothwell, United Kingdom

Congress Organizing Committee
Chair: Alfredo Berardelli, Italy
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Alberto Albanese, Italy
Paolo Barone, Italy
Ubaldo Bonuccelli, Italy
Carlo Colosimo, Italy
Giovanni Fabbrini, Italy
Mario Manfredi, Italy
Stefano Ruggieri Italy
Fabrizio Stocchi, Italy
Mario Zappia, Italy

Past Presidents

2001-2002	Werner Poewe, Austria
1999-2000	Mark Hallett, USA
1997-1998	Eduardo Tolosa, Spain
1995-1996	Joseph Jankovic, USA
1991-1994	C. David Marsden, United Kingdom
1988-1991	Stanley Fahn, USA

International Medical Society for Motor Disturbances

Past Presidents

1993-1994	C. Warren Olanow, USA
1991-1992	Bastian Conrad, Germany
1989-1990	Mark Hallett, USA
1987-1988	Mario Manfredi, Italy
1985-1986	C. David Marsden, United Kingdom

MDS International Secretariat
The *Movement* Disorder Society
555 East Wells Street, 11th Floor
Milwaukee, WI 53202-3823
USA
Tel: +1 414-276-2145
Fax: +1 414-276-3349
E-mail: congress@movementdisorders.org
Web site: www.movementdisorders.org

ARISTEA - Local Organizing Secretariat
Via Tolmino, 5 - 00198 Rome, Italy
Tel: +39 06 845431
Fax: +39 06 84543700
E-mail: aristea.roma@aristea.com

MDS COMMITTEES AND TASK FORCES

Archives Committee
Chair: Werner Poewe
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Awards Committee
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Paolo Barone
Kailash P. Bhatia
Günther Deuschl
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Alexis Elbaz
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Francisco Cardoso
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Beom S. Jeon
Regina Katzenschlager
Eldad Melamed
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Bhim S. Singhal
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Carlo Colosimo
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Irene Litvan
Elan D. Louis
Yasushi Osaki
Young H. Sohn
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Chair: Anthony H.V. Schapira
Thomas Gasser
Etienne C. Hirsch
Joseph Jankovic
Karl D. Kieburtz
José A. Obeso
Fabrizio Stocchi
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Strategy and Planning Committee
Chair: Mark Hallett
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C. Warren Olanow
Werner Poewe
Staff Liaison: Caley Kleczka

Task Force for the Development of Rating Scales for Parkinson's Disease
Chair: Christopher Goetz
Werner Poewe
Olivier Rascol
Cristina Sampaio
Glenn Stebbins
Staff Liaisons: Caley Kleczka, Lisa Seidl

Task Force on Epidemiology
Chair: Caroline Tanner
Yoav Ben-Shlomo
Nadir Bharucha
James Bower
Piu Chan
Dusan Flisar
Amos Korczyn
Mathilde Leonardi
Elan D. Louis
Zvezdan Pirtosek
Gustavo Roman
Web Ross
Staff Liaison: Jenny Oliva

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Task Force on Evidence-Based Medicine in Movement Disorders
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Christopher Goetz
William Koller
Werner Poewe
Olivier Rascol
Staff Liaison: Jody McCarthy

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Co-Chair: Bruno Dubois
Co-Chair: Murat Emre
Co-Chair: Ian McKeith
Dag Aarsland
G. A. (Tony) Broe
Richard Brown
David John Burn
Jeffrey L. Cummings
Dennis Dickson
Charles Duyckaerts
Serge G. Gauthier
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Andrew J. Lees
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Yoshikuni Mizuno
C. Warren Olanow
Werner Poewe
Niall P. Quinn
Cristina Sampaio
Eduardo Tolosa
Staff Liaison: Caley Kleczka

UPDRS Revision Task Force
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Chair: Werner Poewe
Subcommittee Members: Bruno Dubois, Anette Schrag

UPDRS Part II
Chair: Matthew Stern
Subcommittee Members: Anthony Lang, Peter LeWitt

UPDRS Part III
Chair: Stanley Fahn
Subcommittee Members: Joseph Jankovic, C. Warren Olanow

UPDRS Part IV
Chair: Pablo Martinez-Martin
Subcommittee Members: Andrew Lees, Olivier Rascol, Bob Van Hilten

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Chair: Glenn Stebbins
Subcommittee Members: Robert Holloway, David Nyenhuis

Appendices
Chair: Cristina Sampaio
Subcommittee Members: Richard Dodel, Jaime Kulisevsky

Statistical Testing
Chair: Barbara C. Tilley
Subcommittee Members: Sue Leurgans, Jean Teresi

Staff Liaisons: Caley Kleczka, Lisa Seidl

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INTERNATIONAL CONGRESS REGISTRATION AND VENUE

BADGES

All International Congress attendees will receive a name badge with their registration materials. Badges should be worn at all times as they will be used to control access into all International Congress sessions and activities. Individuals will be identified as follows:

Red = Delegate
Yellow = Exhibitor
Orange = Exhibitor Delegate
Green = Guest
Purple = Press
Blue = Staff

LANGUAGE

The official language of the International Congress is English.

REGISTRATION DESK

Location: Ground Floor
Name badges, seminar and special event tickets and International Congress bags can be collected at the International Congress Registration Desk located in the entrance lobby of the Palazzo dei Congressi during the following hours:

Saturday, June 12	3:00 pm to 8:30 pm
Sunday, June 13	7:00 am to 7:30 pm
Monday, June 14	7:00 am to 7:30 pm
Tuesday, June 15	7:00 am to 7:30 pm
Wednesday, June 16	7:00 am to 7:30 pm
Thursday, June 17	7:00 am to 5:00 pm

SPECIAL ACCESSIBILITY NEEDS

Delegates requiring special arrangements in order to fully participate in the International Congress should speak to an MDS staff member at the Registration Desk located on the Ground Floor of the Palazzo dei Congressi.

VENUE

Palazzo dei Congressi
Piazzale J. F. Kennedy
00144 Rome, Italy
The average temperature in Rome in June ranges from a low of 61°F/16°C to a high of 77°F/25°C.

The Palazzo dei Congressi and the EUR

The Congress will take place in the fascinating setting of the Palazzo dei Congressi, located in the EUR district. This modern quarter is considered one of the most noteworthy areas of contemporary Italian urban and architectural culture. The monumental EUR (Esposizione Universale Roma) complex, was conceived in the late 1930s to host the Universal Exhibition which was to be held in 1942, but never took place. The monumental buildings in the urban district of EUR are spaciously set out in an architectural setting with wide tree-lined avenues, parks and a lake.

The Palazzo dei Congressi is one of the outstanding works of Italian architecture from the period between the two wars. It admirably synthesizes the ambitious project in line with the most modern architectural ideas of the time. The building was designed by Adalberto Libera in 1937 and was built in two phases: the first between 1939 and 1943, when building of the EUR district began, and the second between 1952 and 1954, when EUR became a modern residential quarter and one of Rome's most directional areas. The main features of architectural interest are the immense cubic hall, the Salone della Cultura, covered by an imposing vault in reinforced concrete, and the portico supported by granite columns. The Sala dei Congressi, situated behind the main body of the building, is decorated with frescos painted by Gino Severini, a famous Italian futurist artist who lived at the beginning of the XXth century. The cover suspended over the back of the atrium functions as a hanging garden and outdoor theatre thus creating fascinating space that draws inspiration from metaphysical aesthetics.

SOCIAL EVENTS

Sunday, June 13

Opening Ceremony
Location: Salone Della Cultura, Ground Floor
8:30 pm to 9:30 pm

Welcome Reception
Location: Rooftop Terrace
9:30 pm to 11:00 pm

All International Congress attendees and registered guests are invited to meet friends and colleagues during the traditional Opening Cermony and Welcome Reception. Following the Opening Ceremony, a moonlight Welcome Reception will be held on the Rooftop Terrace.

Monday, June 14

International Congress Banquet at the Brancaccio Palace
8:00 pm to 11:00 pm

Palazzo Brancaccio is the newest Roman patrician palace. Built by Princess Elizabeth and Salvatore Brancaccio in 1880, this palace is still considered one of the most beautiful places in Rome.

The evening's itinerary begins with cocktails and hors d'oeuvres; dinner will be served, and local entertainment will highlight the evening.

Transportation to the Brancaccio Palace is provided from the Palazzo dei Congressi and Sheraton Roma beginning at 7:30 pm. Shuttles will depart the Brancaccio Palace at the end of the event. A metro station, close to the Brancaccio Palace, is also available for those staying in the city center.

Tickets purchased in advance are enclosed in each delegate's registration materials. Additional tickets may be purchased, based on availability at the Registration Desk in the Palazzo dei Congressi.

Fee: $100 USD per person

ABSTRACTS-ON-DISK™

All abstracts published in the supplement to the MDS Journal will also be available by Abstracts-On Disk ™ sponsored by MDS and supported through an unrestricted educational grant from Medtronic Neurological. To obtain a copy, please visit the Medtronic Booth #129 and exchange the voucher located in your registration bag.

Abstract Volume

All abstracts accepted for poster presentation have been published in an abstract supplement to the MDS Journal, *Movement* Disorders. Each delegate will receive one copy with their registration materials. MDS members have already received an additional copy with their May journal issue.

CONTINUING MEDICAL EDUCATION

Objectives

As a result of participating in this activity, the attendee should be better able to:

- Describe the pathophysiology and neurobiology of Parkinson's disease and other Movement Disorders
- Discuss the diagnostic approaches and tools available for Parkinson's disease and other Movement Disorders
- Discuss the pharmacological and non-pharmacological treatment options available for Parkinson's disease and other Movement Disorders

Target Audience

The target audience of the 8th International Congress of Parkinson's Disease and Movement Disorders includes clinicians, researchers, post-doctoral fellows and medical school students with an interest in the current research and approaches for the treatment of Movement Disorders.

Availability of CME Credit

The scientific program of the 8th International Congress of Movement Disorders and Parkinson's Disease has been reviewed and approved for Category 1 credit toward the American Medical Association (AMA) Physician's Recognition Award. The *Movement* Disorder Society has approved this educational activity for a maximum of 39 Category 1 credits. Each physician should claim only those credits that he/she actually spent in the educational activity. One credit may be claimed for each hour of participation.

Reciprocity between the European and AMA PRA Credit Systems

A pilot CME credit reciprocity system between the European Union of Medical Specialists (UEMS) and the American Medical Association (AMA) has been extended until 2006. Under the terms of this joint agreement, the UEMS and AMA agree to the exchange and reciprocal recognition of AMA PRA Category 1 and EACCME (European Accreditation Council for Continuing Medical Education) credits earned through participation in approved live educational activities.

Requesting CME Credit Certificates

In order to receive a CME Certificate authenticating participation in this educational activity, International Congress participants must complete and submit a CME Request Form following the last session attended **EACH DAY** of the Congress. Completed CME Request Forms should be handed to meeting room attendants along with completed evaluation forms. Alternatively, completed CME Request Forms can be returned to the CME Desk situated near the Registration Desk on the ground floor of the Congressi.

Participants can find CME Request Forms for each day of the International Congress in their International Congress registration bags. International Congress registration bags are collected upon registering at the Registration Desk on the ground floor. Additional CME Request Forms can be obtained from all meeting room attendants or from the CME Desk near the Registration Desk.

Faculty Financial Disclosure Information

It is the policy of The *Movement* Disorder Society (MDS) to ensure balance, independence, objectivity, and scientific rigor in all sponsored educational activities. All faculty participating in any MDS sponsored activities are required to disclose to the activity audience any real or apparent conflict(s) or interest that may have a direct bearing on the subject matter of the continuing medical education (CME) activity. This pertains to relationships with pharmaceutical companies, biomedical device manufacturers, or other corporations whose products or services are related to the subject matter of the presentation topic. The intent of this policy is not to prevent a speaker with a potential conflict of interest from making a presentation. It is merely intended that any potential conflict should be identified openly so that the listeners may form their own judgments about the presentation with the full disclosure of the facts. It remains for the audience to determine whether the speaker's outside interest may reflect a possible bias in either the exposition or the conclusions presented.

Please see the yellow insert in your International Congress registration bag for complete information regarding faculty disclosure of commercial relationships.

Faculty Disclosure of Unlabeled Product Use Discussion

Presentations which provide information in whole or in part related to non-approved uses for drug products and/or devices must clearly acknowledge the unlabeled indications or the investigative nature of their proposed uses to the audience. Speakers who plan to discuss non-approved uses for commercial products and/or devices must advise the International Congress audience of their intent.

Please see the yellow insert in your International Congress registration bag for complete information regarding faculty disclosure of unlabeled product use discussion.

Continuing Medical Education for Italian Physicians

For information regarding Continuing Medical Education for Italian Physicians, please contact Maddalena Redini at the Technical Secretariat for CME Accreditation, the Italian Society of Neurology, at telephone +39 50 879740 or by e-mail at neuro@sirius.pisa.it.

Evaluations

Please take time to complete the evaluation forms provided for each session you attend. Your input and comments are essential in planning future educational programs for MDS. When completed, evaluations may be returned to your meeting room attendants or to the MDS Registration Desk.

INTERNATIONAL CONGRESS INFORMATION

EXHIBITION

Location: Ground Floor

Please allow adequate time in your daily schedule to visit the exhibits located throughout the Ground Level of the Palazzo dei Congressi. The exhibition is an integral component of your International Congress experience, offering you the opportunity to speak with representatives of companies that provide services and market products directly related to Movement Disorders. Representatives will be available to discuss these services and products during the following hours:

Monday, June 14	8:00 am to 5:00 pm
Tuesday, June 15	8:00 am to 5:00 pm
Wednesday, June 16	8:00 am to 5:00 pm
Thursday, June 17	8:00 am to 5:00 pm

INTERNET CAFÉ

Location: First Floor

Internet access will be available to meeting attendees on the First Floor of the Palazzo dei Congressi. The Internet Café is supported through an unrestricted educational grant from Cephalon, Inc. Please limit your internet use to 15 minutes so that other attendees can also access this service.

LUNCH OPTIONS

Rooftop Terrace Restaurant

Location: Rooftop Terrace

Served lunch will be available at the Rooftop Terrace Restaurant. Tickets are required for lunch and may be purchased at the Registration Desk.

Quick Lunch

Location: Ground Floor

Lunch bags containing sandwiches, fruit and snacks may be purchased. Tickets are required for lunch and may be purchased at the Registration Desk.

MDS EXHIBIT AND INFORMATION STAND

Location: Registration Area, Ground Floor

The *Movement* Disorder Society (MDS) is an international society of healthcare professionals committed to research and patient care in the fields of Parkinson's disease and other disorders of movement and motor control.

MDS supports and promotes a wide range of educational programming and other initiatives to advance scientific understanding and standards of care as they pertain to Movement Disorders. For this, MDS provides forums such as a high ranking journal, scientific symposia and international congresses.

Attendees are invited to take advantage of MDS member benefits by applying to the Society. Learn more about MDS initiatives and speak with a representative at the MDS Exhibit and Information Stand located in the Registration Area during the following hours:

Saturday, June 12	3:00 pm to 8:30 pm
Sunday, June 13	8:00 am to 5:30 pm
Monday, June 14	8:00 am to 5:30 pm
Tuesday, June 15	8:00 am to 5:30 pm
Wednesday, June 16	8:00 am to 5:30 pm
Thursday, June 17	8:00 am to 5:00 pm

NO CAMERAS

Cameras are not permitted in any 8th International Congress educational session or in the poster areas.

OPTIONAL TOURS

Tours have been arranged by:
ARISTEA
Via Tolmino, 5
00198 Roma - Italy
Tel. +39 06 845431
Fax +39 06 84543700

Please visit the Tours Desk in the Registration Area on the Ground Floor to collect your tour tickets. Additional tour tickets may be purchased at this desk, based on availability.

PRESS ROOM

Location: Press Room, Ground Floor

Members of the working media may register without charge for the 8th International Congress in the Press Room. Press must register, provide credentials and wear their badge for admittance into MDS sessions.

Press Room hours are as follows:

Sunday, June 13	8:00 am to 5:00 pm
Monday, June 14	8:00 am to 5:00 pm
Tuesday, June 15	8:00 am to 5:00 pm
Wednesday, June 16	8:00 am to 5:00 pm
Thursday, June 17	8:00 am to 5:00 pm

SCIENTIFIC PROGRAM

Kickoff Seminars

Kickoff Seminars emphasize pharmacological treatment approaches for Movement Disorders, as well as diagnostic strategy overviews and updates. These industry supported seminars are open to all International Congress registrants.

Plenary and Parallel Sessions

Plenary and Parallel Sessions continue to offer a variety of popular topics in lecture format and panel discussion from renowned neurologists and Movement Disorder specialists from around the world. Each presenter offers his/her perspective and information on the latest studies and research on Parkinson's disease and other Movement Disorders. These main sessions are open to all International Congress registrants.

Seminars

Sessions offering Italian cuisine are featured throughout the International Congress week, similar to the popular Wine and Cheese Seminars from the 7th International Congress in Miami in 2002. Each session offers an expert's view on Movement Disorders through a variety of topics. Seminars have limited registration to encourage discussion and interaction with presenters.

Fee: $55 USD/ $40 USD for junior participants and allied health professionals.

Video Dinners

Due to outstanding reviews from the Miami International Congress, Video Dinners are again offered. Video presentations of atypical Movement Disorders engage delegates and generate clinical discussions. To ensure greater interaction, video sessions participation is limited. Dinner is served during the sessions.

Fee: $80 USD/ $55 USD for junior participants and allied health professionals.

Platform Presentations

16 abstracts have been selected for oral platform presentation at the International Congress. The abstracts selected feature newsworthy and cutting-edge information about Parkinson's disease and Movement Disorders. The Platform Presentations are held as main sessions, and are open to all International Congress delegates.

Abstract Poster Sessions

Delegate feedback from past International Congresses has indicated a great interest in Poster Sessions. Poster Sessions are featured each day utilizing the following schedule:

Poster Session 1

Location: First Floor

Monday, June 14
Poster Viewing: 8:30 am to 5:00 pm
Authors Present Odd Numbers: 12:00 pm to 1:00 pm
Authors Present Even Numbers: 4:00 pm to 5:00 pm
Abstracts 1-344

Poster Session 2

Location: First Floor

Tuesday, June 15
Poster Viewing: 8:30 am to 5:00 pm
Authors Present Odd Numbers: 11:30 am to 12:30 pm
Authors Present Even Numbers: 4:00 pm to 5:00 pm
Abstracts 345-694

Poster Session 3

Location: First Floor

Wednesday, June 16
Poster Viewing: 8:30 am to 5:00 pm
Authors Present Odd Numbers: 11:30 am to 12:30 pm
Authors Present Even Numbers: 4:00 pm to 5:00 pm
Abstracts 695-1017

Poster Session 4

Location: First Floor

Thursday, June 17
Poster Viewing: 8:30 am to 4:30 pm
Authors Present Odd Numbers: 12:00 pm to 1:00 pm
Authors Present Even Numbers: 1:00 pm to 2:00 pm
Abstracts 1018-1338

SOCIAL EVENTS

Sunday, June 13

Opening Ceremony

Location: Salone Della Cultura, Ground Floor
8:30 pm to 9:30 pm

Welcome Reception

Location: Rooftop Terrace
9:30 pm to 11:00 pm

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The evening's itinerary begins with cocktails and hors d'oeuvres; dinner will be served, and local entertainment will highlight the evening.

Transportation to the Brancaccio Palace is provided from the Palazzo dei Congressi and Sheraton Roma beginning at 7:30 pm. Shuttles will depart the Brancaccio Palace at the end of the event. A metro station, close to the Brancaccio Palace, is also available for those staying in the city center.

Tickets purchased in advance are enclosed in each delegate's registration materials. Additional tickets may be purchased, based on availability at the Registration Desk in the Palazzo dei Congressi.

Fee: $100 USD per person

SPEAKER READY ROOM

Location: Slide Review Room, Ground Floor

All speakers must check in at the Speaker Ready Room with presentation materials on the day prior to their scheduled presentation. Equipment is available for faculty to review their presentations. Audio visual personnel will be available for assistance.

The Speaker Ready Room hours are as follows:

Saturday, June 12	5:00 pm to 8:00 pm
Sunday, June 13	7:00 am to 8:00 pm
Monday, June 14	7:00 am to 6:00 pm
Tuesday, June 15	7:00 am to 8:00 pm
Wednesday, June 16	7:00 am to 8:00 pm
Thursday, June 17	7:00 am to 4:30 pm

TRANSPORTATION

Shuttle service is offered between the Sheraton Roma Hotel and the Palazzo dei Congressi. Delegates commuting from the city center receive metro passes in their on-site registration materials.

To reach the Palazzo dei Congressi from the City Center by subway take the B Line to Fermi Station. Please refer to the metro map on page 51. A shuttle from Fermi Station to the Palazzo dei Congressi is available.

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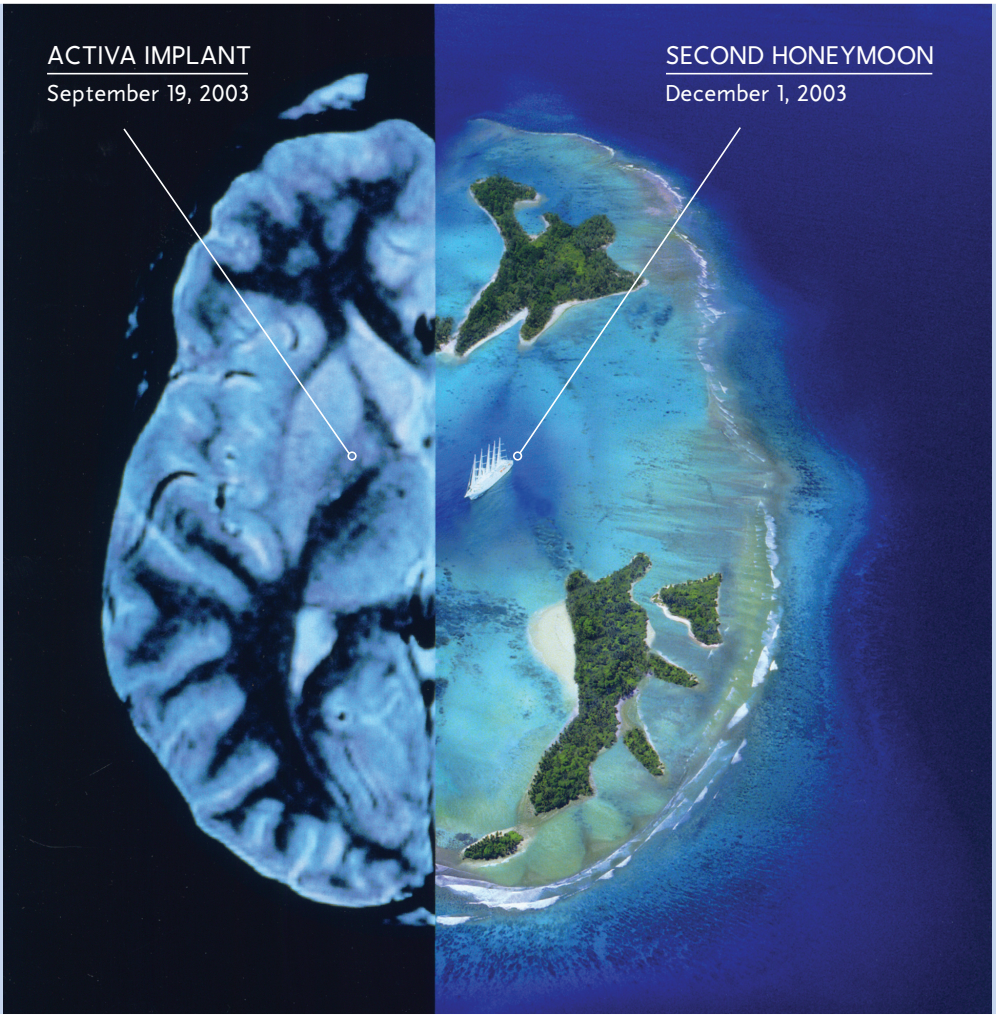

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* *Results were for a subset of patients whose data were verified against medical records. Data on file at Medtronic, Inc.*

** *PD symptom improvement with medication off. Results were for a subset of patients whose data were verified against medical records. Data on file at Medtronic, Inc.*

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Activa® Parkinson's Control Therapy and Tremor Control Therapy:
Product technical manual must be reviewed prior to use for detailed disclosure.

Indications:
Parkinson's Control Therapy: Bilateral stimulation of the internal globus pallidus (GPi) or the subthalamic nucleus (STN) using Medtronic Activa® Parkinson's Control Therapy is indicated for adjunctive therapy in reducing some of the symptoms of advanced, levodopa-responsive Parkinson's disease that are not adequately controlled with medication.
Tremor Control Therapy: Unilateral thalamic stimulation by the Medtronic Activa® Tremor Control System is indicated for the suppression of tremor in the upper extremity. The system is intended for use in patients who are diagnosed with Essential Tremor or Parkinsonian tremor not adequately controlled by medications and where the tremor constitutes a significant functional disability. The safety or effectiveness of this therapy has not been established for bilateral stimulation.

Contraindications:
Contraindications include patients who will be exposed to MRI using a full body radio-frequency (RF) coil or a head transmit coil that extends over the chest area, patients for whom test stimulation is unsuccessful, or patients who are unable to properly operate the neurostimulator. Also, diathermy (e.g., shortwave diathermy, microwave diathermy or therapeutic ultrasound diathermy) is contraindicated because diathermy's energy can be transferred through the implanted system (or any of the separate implanted components), which can cause tissue damage and can result in severe injury or death. Diathermy can damage parts of the neurostimulation system.

Warnings/Precautions/Adverse Events:
There is a potential risk of tissue damage using stimulation parameter settings of high amplitudes and wide pulse widths. Extreme care should be used with lead implantation in patients with a heightened risk of intracranial hemorrhage. Do not place the lead-extension connector in the soft tissues of the neck. Placement in this location has been associated with an increased incidence of lead fracture. Theft detectors and security screening devices may cause stimulation to switch ON or OFF, and may cause some patients to experience a momentary increase in perceived stimulation. Although some MRI procedures can be performed safely with an implanted Activa System, clinicians should carefully weigh the decision to use MRI in patients with an implanted Activa System. MRI can cause induced voltages in the neurostimulator and/or lead possibly causing uncomfortable, jolting, or shocking levels of stimulation. MRI image quality may be reduced for patients who require the neurostimulator to control tremor, because the tremor may return when the neurostimulator is turned off.

Severe burns could result if the neurostimulator case is ruptured or pierced. The Activa System may be affected by or adversely affect medical equipment such as cardiac pacemakers or therapies, cardioverter/defibrillators, external defibrillators, ultrasonic equipment, electrocautery, or radiation therapy. Safety and effectiveness has not been established for patients with neurological disease other than Parkinson's disease or Essential Tremor, previous surgical ablation procedures, dementia, coagulopathies, or moderate to severe depression; or for patients who are pregnant, under 18 years, over 75 years of age (Parkinson's Control Therapy) or over 80 years of age (Tremor Control Therapy). Adverse events related to the therapy, device, or procedure can include: stimulation not effective, cognitive disorders, pain, dyskinesia, dystonia, speech disorders including dysarthria, infection, paresthesia, intracranial hemorrhage, electromagnetic interference, cardiovascular events, visual disturbances, sensory disturbances, device migration, paresis/asthenia, abnormal gait, incoordination, headaches, lead repositioning, thinking abnormal, device explant, hemiplegia, lead fracture, seizures, respiratory events, and shocking or jolting stimulation.

Rx only

PROGRAM AT A GLANCE

	Sunday, June 13		Monday, June 14	Tuesday, June 15	Wednesday, June 16	Thursday, June 17	
7:00 AM			Committees and Workgroups	Committees and Workgroups	Committees and Workgroups	Committees and Workgroups	7:00 AM
8:00 AM							8:00 AM
9:00 AM	Kickoff Seminar 1A	Kickoff Seminar 1B	Plenary Session 1	Parallel Sessions 1 & 2 Platform Presentations	Plenary Session 3	Seminar Series	9:00 AM
10:00 AM	Kickoff Seminar 3	Kickoff Seminar 2		MDS Business Meeting		Parallel Sessions 5 & 6	10:00 AM
11:00 AM			Marsden Lecture		Fahn Lecture		11:00 AM
12:00 PM		Kickoff Seminar 4	Junior Awards Poster Session 1 Odd Numbers	Poster Session 2 Odd Numbers Lunch Break	Poster Session 3 Odd Numbers Lunch Break	Poster Session 4 and Lunch	12:00 PM
1:00 PM	Kickoff Seminar 5		Lunch Break				1:00 PM
2:00 PM		Kickoff Seminar 6	Plenary Session 2	Parallel Sessions 3 & 4	Plenary Session 4	Parallel Sessions 7 & 8	2:00 PM
3:00 PM		Kickoff Seminar 7					3:00 PM
4:00 PM	Kickoff Seminar 8		Poster Session 1 Even Numbers	Poster Session 2 Even Numbers	Poster Session 3 Even Numbers		4:00 PM
5:00 PM		Kickoff Seminar 9	Seminar Series	Seminar Series	Seminar Series		5:00 PM
6:00 PM	Kickoff Seminar 10	Kickoff Seminar 11					6:00 PM
7:00 PM				Video Dinners	Video Dinners		7:00 PM
8:00 PM			Congress Banquet				8:00 PM
	Opening Ceremony						
9:00 PM	Welcome Reception						9:00 PM
10:00 PM							10:00 PM

SUNDAY, JUNE 13, 2004

KICKOFF SEMINARS

8:30 am to 9:30 am

Kickoff Seminar 1A: Managing Parkinson's disease: turning off to on

Location: Salone Della Cultura, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Bertek Pharmaceuticals, Inc.

Chairs: William Koller
New York, NY, USA

Fabrizio Stocchi
Rome, Italy

The history of apomorphine
Andrew Lees
London, United Kingdom

Apomorphine as a rescue agent in Parkinson's disease
Mark Stacy
Durham, NC, USA

Panel discussion

At the conclusion of this session, participants should be able to: 1. Describe the motor complications associated with advanced Parkinson's disease; 2. Discuss the history of apomorphine and its use as a "rescue agent" in Parkinson's disease; 3. Explain when "rescue therapy" is needed in Parkinson's disease; 4. Use an apomorphine injectable pen and instruct patients in its appropriate handling.

8:30 am to 9:30 am

Kickoff Seminar 1B: Essential tremor: new insights into cause and treatment

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Ortho-McNeil Pharmaceutical.

Chair: Mark Hallett
Bethesda, MD, USA

Medical and surgical treatment of essential tremor
Joseph Jankovic
Houston, TX, USA

Pathophysiology of essential tremor
Günther Deuschl
Kiel, Germany

Panel discussion

10:00 am to 11:00 am

Kickoff Seminar 2: Depression in Parkinson's disease: role of dopamine agonists

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Boehringer Ingelheim International GmbH.

Chairs: Heinz Reichmann
Dresden, Germany

Yoshikuni Mizuno
Tokyo, Japan

Depression in Parkinson's disease: clinical features and significance
Christopher G. Goetz
Chicago, IL, USA

Role of dopamine agonists in the treatment of depression in Parkinson's disease
Paolo Barone
Napoli, Italy

Panel discussion

At the conclusion of the session, participants should be able to: 1.Describe how to identify and diagnose depression in patients with Parkinson's disease; 2. Describe which rating scales should be used to evaluate the severity of depression in PD; 3. Describe what are the effective therapeutic approaches for depression in PD.

10:00 am to 12:00 pm

Kickoff Seminar 3: Restless legs syndrome: advances in diagnosis and treatment

Location: Salone Della Cultura, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Pfizer, Inc.

Chairs: Claudia Trenkwalder
Kassel, Germany

Jacques Montplaisir
Montreal, Canada

Update on etiology and pathogenesis
Ray Chaudhuri
London, United Kingdom

Diagnosis and differential diagnosis
Diego Garcia Borreguero
Madrid, Spain

Non-dopaminergic treatment
Arthur S. Walters
Edison, NJ, USA

Dopaminergic treatment
Per Odin
Bremerhave, Germany

Panel discussion

In sessions not listing learning objectives, specific objectives will be shared with participants at the beginning of the session.

SCIENTIFIC PROGRAM

11:30 am to 12:30 pm

Kickoff Seminar 4: Neuroimaging

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from from Amersham Health.

Chairs: Andrew Lees
London, United Kingdom

Wolfgang Oertel
Marburg, Germany

Use of imaging for diagnosis and assessment of therapy in Parkinson's disease

David J. Brooks
London, United Kingdom

Neuroimaging in other Movement Disorders

A. Jon Stoessl
Vancouver, Canada

Panel discussion

At the conclusion of this session, participants should be able to: 1. Understand the basic principles of PET and SPECT imaging in Parkinson's disease; 2. Explain the role of SPECT and PET imaging in diagnosis and therapy of Parkinson's disease; 3. Define the role of SPECT and PET imaging in diagnosis of other Movement Disorders; 4. Highlight the clinical value of dopamine transporter imaging in the routine work-up of patients with Movement Disorders; 5. To assess the role of imaging as a surrogate marker for disease progression in parkinsonian syndromes.

1:00 pm to 3:00 pm

Kickoff Seminar 5: Dopamine Agonists—New perspectives in the treatment of Parkinson's disease and restless legs syndrome

Location: Salone Della Cultura, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from GlaxoSmithKline.

Chairs: Amos Korczyn
Ramat Aviv, Israel

Joaquim Ferreira
Torres Vedras, Portugal

Dopamine agonists—historical perspectives in Parkinson's disease

Kapil Sethi
Augusta, GA, USA

Restless legs syndrome—pathophysiology

Walter Paulus
Gottingen, Germany

Restless legs syndrome—diagnosis and significance to patients

Thomas Roth
Detroit, MI, USA

Restless legs syndrome—treatment

Richard Allen
Bethesda, MD, USA

Panel discussion

At the conclusion of this session, participants should be able to: 1. Discuss the syndrome, understand how to diagnose and treat it and explain the theories on it; 2. Describe pathogenisis.

1:30 pm to 2:30 pm

Kickoff Seminar 6: Transdermal delivery of dopaminergic drugs

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Schwarz Pharma.

Chairs: Niall Quinn
London, United Kingdom

William Weiner
Baltimore, MD, USA

CDS, delivery methods in Parkinson's disease

Peter LeWitt
Southfield, MI, USA

Novel transdermal delivery approaches for Parkinson's disease

Cheryl Waters
New York, NY, USA

Panel discussion

3:00 pm to 4:00 pm

Kickoff Seminar 7: Addressing dementia and neuropsychiatric issues in Parkinson's disease

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Novartis Pharma.

Chairs: Bruno Dubois
Paris, France

Ian McKeith
Newcastle Upon Tyne, United Kingdom

The challenges of dementia and neuropsychiatric symptoms in Parkinson's disease

Ray Watts
Birmingham, AL, USA

Treatment options in Parkinson's disease dementia and dementia with Lewy bodies

Murat Emre
Capa Istanbul, Turkey

Panel discussion

3:30 pm to 5:30 pm

Kickoff Seminar 8: New directions in the treatment of Parkinson's disease using MAO-B inhibitors and propargylamines

Location: Salone Della Cultura, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Teva Pharmaceutical Industries Ltd., Teva Neuroscience, Lundbeck and Eisai.

Chairs: Anthony E. Lang
Toronto, Canada

Werner Poewe
Innsbruck, Austria

Role in the treatment of early disease

Matthew B. Stern
Philadelphia, PA, USA

Role in the treatment of advanced disease

Olivier Rascol
Toulouse, France

Rationale and potential for modifying disease progression

Ira Shoulson
Rochester, NY, USA

Panel discussion

At the conclusion of the session, participants should be able to: 1. Describe the role of MAO-B inhibitors and propargylamines in the treatment of de novo patients with Parkinson's disease; 2. Define efficacy and safety of MAO-B inhibitors and propargylamines across the various stages of Parkinson's disease; 3. Discuss issues in the design of clinical trials of disease modifying agents in Parkinson's disease and identify the rationale for MAO-B inhibitors and propargylamines.

4:30 pm to 5:30 pm

Kickoff Seminar 9: Is botulinum toxin toxic?

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Allergan, Inc.

Chairs: Alfredo Berardelli
Rome, Italy

Cynthia L. Comella
Chicago, IL, USA

Are there long-term toxicity issues?

Markus Naumann
Wuerzburg, Germany

Immunogenicity and long-term botulinum toxin administration

Joseph Jankovic
Houston, TX, USA

Panel discussion

At the conclusion of this session, participants should be able to: 1. List factors associated with the occurrence of BTX-A adverse events; 2. Discuss long term benefits and safety of botulinum toxin for cervical dystonia; 3. List risk factors for the development of immunogenicity against botulinum toxin.

SCIENTIFIC PROGRAM

6:00 pm to 8:00 pm

Kickoff Seminar 10: Dopamine agonists as potential disease modifying therapy in Parkinson's disease

Location: Salone Della Cultura, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Pfizer, Inc.

Chairs: Oscar Gershanik
Buenos Aires, Argentina

Anthony H.V. Schapira
London, United Kingdom

Etiology of Parkinson's disease
Etienne Hirsch
Paris, France

Motor and non-motor complications of levodopa-treated Parkinson's disease
Eduardo Tolosa
Barcelona, Spain

Dopamine agonists in the prevention and treatment of motor and non-motor complications
José Obeso
Pamplona, Spain

Dopamine agonists as putative neuroprotective agents
Ken Marek
New Haven, CT, USA

Panel discussion

At the conclusion of this session, participants should be able to: 1. Describe some of the hypothesis that presently try to explain the etiology and pathogenesis of Parkinson's disease; 2. Discuss the controversies related to the putative neuroprotective effects of dopamine agonists and the tools used in their evaluation; 3. Recognize the motor and non-motor complications that affect PD patients under long-term levodopa treatment.

6:00 pm to 8:00 pm

Kickoff Seminar 11: Levodopa-CDS in the treatment of Parkinson's disease: the role of COMT-inhibition

Location: Assembly Hall, Ground Floor

Sponsored by The Movement Disorder Society. Supported through an unrestricted educational grant from Novartis Pharma/Orion Pharma.

Chairs: Yves Agid
Paris, France

C. Warren Olanow
New York, NY, USA

Levodopa-related motor complications
Eldad Melamed
Petah Tiqva, Israel

CDS approaches to animal models in Parkinson's disease
Peter Jenner
London, United Kingdom

CDS approaches to treating Parkinson's disease patients
Fabrizio Stocchi
Rome, Italy

COMT inhibitors in the treatment of Parkinson's disease
Robert Hauser
Tampa, FL, USA

Panel discussion

MONDAY, JUNE 14, 2004

8:30 am to 11:00 am
Plenary Session 1: Etiopathogenesis of cell death in Parkinson's disease
Location: Salone Della Cultura, Ground Floor

Chair: C. Warren Olanow
New York, NY, USA

Co-chair: Etienne Hirsch
Paris, France

Etiology: Update on genetic and environmental factors of cell death
J.W. Langston
Sunnyvale, CA, USA

Genetic causes ofParkinson's disease
John Hardy
Bethesda, MD, USA

Pathogenesis: Role of mitochondria, oxidative stress, inflammation and excitotoxicity in neurodegeneration
Serge Przedborski
New York, NY, USA

The UPS and models of Parkinson's disease
Kevin McNaught
New York, NY, USA

At the conclusion of this session, participants should be able to: 1. List the major factors involved in the etiopathogenesis of Parkinson's disease; 2. Describe the mechanisms potentially involved in the mechanism of neuronal degeneration in Parkinson's disease; 3. Explain the role of protein processing in the etiopathogenesis of Parkinson's disease.

11:00 am to 11:30 am
C. David Marsden Lecture
The value of transgenic and gene targeted models for experimental therapeutics of neurodegenerative diseases
Location: Salone Della Cultura, Ground Floor

Donald Price
Baltimore, MD, USA

11:30 am to 12:00 pm
Junior Awards
Location: Salone Della Cultura, Ground Floor

12:00 pm to 1:00 pm
Abstract Poster Session 1
Location: Poster Area, First Floor

Abstract Numbers 1-344
Authors present odd numbers

1:00 pm to 1:30 pm
Lunch
Location: Rooftop Terrace and Various Locations

1:30 pm to 4:00 pm
Plenary Session 2: The basal ganglia pathophysiological model: contributions and limitations
Location: Salone Della Cultura, Ground Floor

Chair: José Obeso
Pamplona, Spain

Co-chair: Nobuo Yanagisawa
Kawasaki-City, Japan

Introduction: the model
Nobuo Yanagisawa
Kawasaki-City, Japan

Anatomical chemical organization of the basal ganglia: misconceptions
Hagai Bergman
Jerusalem, Israel

Dopamine depletion and modification of basal ganglia activity
Erwan Bezard
Bordeaux, France

Functional imaging of the basal ganglia
Joel Perlmutter
St. Louis, MO, USA

Neuronal activity and Movement Disorders: firing, rhythms and patterns
Peter Brown
London, United Kingdom

Consequence of lesion of the basal ganglia in man
John Rothwell
London, United Kingdom

Conclusion: lessons from the model
José Obeso
Pamplona, Spain

4:00 pm to 5:00 pm
Abstract Poster Session 1
Location: Poster Area, First Floor

Abstract Numbers 1-344
Authors present even numbers

SCIENTIFIC PROGRAM

5:00 pm to 6:30 pm

Seminar Series

Sessions featuring Italian cuisine are featured on Monday. Each session offers an expert's view on Movement Disorders through a variety of topics. To encourage discussion and interaction, the seminar series have limited registration and a ticket is required for admission.

Fee: $55 USD/ $40 USD for junior participants and allied health professionals.

S101 Advances in stiff person syndrome

Location: Meeting Room 5, First Floor

Philip Thompson
North Terrace, Adelaide, Australia

Hans Meinck
Heidelberg, Germany

At the conclusion of this session, participants should be able to: 1. Identify the clinical manifestations of the stiff man syndrome and its variants; 2. Describe the appropriate diagnostic tests to confirm the diagnosis status and to rule out other relevant diseases; 3. Discuss the therapeutic options.

S102 Ataxias

Location: Meeting Room 1, First Floor

S.H. Subramony
Jackson, MS, USA

Stefan Pulst
Los Angeles, CA, USA

At the conclusion of this session, participants should be able to: 1. Recognize the clinical manifestations of inherited ataxias and discuss their differential diagnosis; 2. Describe the use of genetic tests and be familiar with their interpretation; 3. Discuss the pathogenesis of recessive and dominant ataxias.

S103 Autonomic nervous system function in neurodegenerative disease

Location: Meeting Room 6, First Floor

Horacio Kaufmann
New York, NY, USA

Ronald Pfeiffer
Memphis, TN, USA

At the conculsion of this session, participants should be able to: 1. Recognize autonomic dysfunction is a frequent and sometimes dominant feature of the "synucleinpathic" neurodegenerative Movement Disorders such such as Parkinson's disease multiple system atrophy, and dementia with Lewy bodies; 2. Identify the specific cardiovascular, sexual, urinary and gastrointestinal features of autonomic dysfunction in neurodegenerative Movement Disorders; 3. Discuss appropriate diagnostic and treatment approaches for the cardiovascular, sexual, urinary and gastrointestinal manifestations of autonomic dysfunction in neurodegenerative Movement Disorders.

S104 Case management: Parkinson's disease

Location: Meeting Room 3, First Floor

Christopher Goetz
Chicago, IL, USA

Cheryl Waters
New York, NY, USA

At the conclusion of this session, participants should be able to: 1. Define treatment options for Parkinson's disease based on current evidence from clinical trials; 2. Discuss treatment options for Parkinson's disease, combining evidence from clinical trials with practice experience; 3. Recognize management options that are problem-specific for the treatment of Parkinson's disease at different phases of disease progression.

S105 Parkinson's disease in the elderly (diagnosis and management)

Location: Meeting Room 7, Ground Floor

Giovanni Fabbrini
Rome, Italy

François Tison
Pessac, France

At the conclusion of this session, participants should be able to: 1. Discuss the general principals of differential diagnosis in the elderly parkinsonian patients; 2. Describe the clinical phenotype and the clinical problems of aged parkinsonian patients, with regard to the biological pattern of neurodegeneration observed in the elderly, the general comorbidities, the incidence of dementia and psychiatric disturbances; 3. Discuss the currently available treatment in elderly parkinsonian patients, with regard to the paucity of controlled studies, the differences in the pharmokinetic and pharmacodynamic of antiparkinsonian drugs in this population.

S106 Parkinsonism - PSP/CBGD: clinical update

Location: Meeting Room 2, First Floor

Peter Pramstaller
Bolzano, Italy

Lawrence Golbe
New Brunswick, NJ, USA

At the conclusion of this session, participants should be able to: 1. Recognize the clinical features of progressive supranuclear palsy and corticobasal degeneration and be able to apply formal clinical diagnostic criteria to distinguish PSP and CBD from each other and from competing diagnostic considerations; 2. Explain the clinical deficits of PSP and CBD to patients and caregivers in order to help them avoid complications of the illness, including those caused by unnecessary diagnostic testing and useless treatments; 3. Describe current understanding of the etiology and pathogenesis of the brain degeneration in PSP and CBD, including what is known of the genetic and toxic factors so that they can provide informed answers to patients' and families' questions regarding familial and occupational risks.

S107 Pediatric Movement Disorders

Location: Meeting Room 4, First Floor

Robert Surtees
London, United Kingdom

Terence Sanger
Stanford, CA, USA

At the conclusion of this session, participants should be able to: 1. Recognize the most common pediatric Movement Disorders; 2. List the most common treatments of childhood Movement Disorders; 3. Explain the differential diagnosis of the most common pediatric Movement Disorders.

S108 Restless legs syndrome

Location: Meeting Room 8, Ground Floor

Richard Allen
Arnold, MD, USA

Claudia Trenkwalder
Kassel, Germany

At the conclusion of this session, participants should be able to: 1. Describe and define the key features of restless legs syndrome including the essential definition criteria and the role of sleep disturbance; 2. Discuss the differential diagnosis that are important to differentiate RLS from i.e. polyneuropathy, sleep apnea with PLM, PLMD and to explain possible pathophysiological concepts of RLS; 3. Indicate the appropriate treatment strategies for RLS including dopaminergic medication, opirids, gabapentin and others.

Because
she thinks *the* world *of* him

For patients like Edward living with Parkinson's disease, it's the simple tasks that are important, like helping to fix his granddaughter's bike. However, living with PD makes it increasingly difficult to do even the simplest things in life.[1] REQUIP can help. With REQUIP, patients like Edward are able to maintain their ability to perform activities of daily living while significantly reducing the risk of dyskinesia vs L-dopa.[2]

Make a difference for your patients with Parkinson's disease.

Safety and effectiveness in the pediatric population have not been established.

REQUIP has been associated with sedating effects, including somnolence, and the possibility of falling asleep while engaged in activities of daily living, including operation of a motor vehicle. Syncope or symptomatic hypotension may occur more frequently during initial treatment or with an increase in dose. Hallucinations may occur at any time during treatment. REQUIP may potentiate the dopaminergic side effects of L-dopa and may cause and/or exacerbate pre-existing dyskinesias.

FOR THE TREATMENT OF PARKINSON'S DISEASE

REQUIP®
ropinirole HCl

Please see brief summary of complete Prescribing Information on adjacent page.

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A Progressive Therapy for a **Progressive Disease**

REQUIP® (ropinirole hydrochloride) Tablets **BRIEF SUMMARY**

The following is a brief summary only; see full prescribing information for complete product information.

INDICATIONS AND USAGE: REQUIP is indicated for the treatment of the signs and symptoms of idiopathic Parkinson's disease. The effectiveness of REQUIP was demonstrated in randomized, controlled trials in patients with early Parkinson's disease who were not receiving concomitant L-dopa therapy as well as in patients with advanced disease or concomitant L-dopa.

CONTRAINDICATIONS: REQUIP is contraindicated for patients known to have hypersensitivity to the product.

WARNINGS: Falling Asleep During Activities of Daily Living: Patients treated with REQUIP have reported falling asleep while engaged in activities of daily living, including the operation of motor vehicles which sometimes resulted in accidents. Although many of these patients reported somnolence while on REQUIP, some perceived that they had no warning signs such as excessive drowsiness, and believed that they were alert immediately prior to the event. Some of these events have been reported as late as one year after initiation of treatment. Somnolence is a common occurrence in patients receiving REQUIP. Many clinical experts believe that falling asleep while engaged in activities of daily living always occurs in a setting of pre-existing somnolence although patients may not give such a history. For this reason, prescribers should continually reassess patients for drowsiness or sleepiness especially since some of the events occur well after the start of treatment. Prescribers should also be aware that patients may not acknowledge drowsiness or sleepiness until directly questioned about drowsiness or sleepiness during specific activities. Before initiating treatment with REQUIP, patients should be advised of the potential to develop drowsiness and specifically asked about factors that may increase the risk with REQUIP such as concomitant sedating medications, the presence of sleep disorders, and concomitant medications that increase ropinirole plasma levels (e.g., ciprofloxacin—see PRECAUTIONS, Drug Interactions). If a patient develops significant daytime sleepiness or episodes of falling asleep during activities that require active participation (e.g., conversations, eating, etc.), REQUIP should ordinarily be discontinued. (See DOSAGE AND ADMINISTRATION for guidance in discontinuing REQUIP.) If a decision is made to continue REQUIP, patients should be advised to not drive and to avoid other potentially dangerous activities. There is insufficient information to establish that dose reduction will eliminate episodes of falling asleep while engaged in activities of daily living. Syncope: Syncope, sometimes associated with bradycardia, was observed in association with ropinirole in both early Parkinson's disease (without L-dopa) patients and advanced Parkinson's disease (with L-dopa) patients. In the two double-blind placebo-controlled studies of REQUIP in patients with Parkinson's disease who were not being treated with L-dopa, 11.5% (18 of 157) of patients on REQUIP had syncope compared to 1.4% (2 of 147) of patients on placebo. Most of these cases occurred more than 4 weeks after initiation of therapy with REQUIP, and were usually associated with a recent increase in dose. Of 208 patients being treated with both L-dopa and REQUIP, in placebo-controlled advanced Parkinson's disease trials, there were reports of syncope in 6 (2.9%) compared to 2 of 120 (1.7%) of placebo/ L-dopa patients. Because the studies of REQUIP excluded patients with significant cardiovascular disease, it is not known to what extent the estimated incidence figures apply to Parkinson's disease patients as a whole. Therefore, patients with severe cardiovascular disease should be treated with caution. Two of 47 Parkinson's disease patient volunteers enrolled in phase 1 studies had syncope following a 1-mg dose. In phase 1 studies including 110 healthy volunteers, one patient developed hypotension, bradycardia, and sinus arrest of 26 seconds accompanied by syncope; the patient recovered spontaneously without intervention. One other healthy volunteer reported syncope. **Symptomatic Hypotension:** Dopamine agonists, in clinical studies and clinical experience, appear to impair the systemic regulation of blood pressure, with resulting postural hypotension, especially during dose escalation. Parkinson's disease patients, in addition, appear to have an impaired capacity to respond to a postural challenge. For these reasons, Parkinson's patients being treated with dopaminergic agonists ordinarily (1) require careful monitoring for signs and symptoms of postural hypotension, especially during dose escalation, and (2) should be informed of this risk (see PRECAUTIONS, Information for Patients). Although the clinical trials were not designed to systematically monitor blood pressure, there were individual reported cases of postural hypotension in early Parkinson's disease (without L-dopa) patients treated with REQUIP. Most of these cases occurred more than 4 weeks after initiation of therapy with REQUIP, and were usually associated with a recent increase in dose. In phase 1 studies of REQUIP that included 110 healthy volunteers, nine subjects had documented symptomatic postural hypotension. These episodes appeared mainly at doses above 0.6 mg and these doses are higher than the starting doses recommended for Parkinson's disease patients. In eight of these nine individuals, the hypotension was accompanied by bradycardia, but did not develop into syncope. (See Syncope above.) None of these events resulted in death or hospitalization. One of 47 Parkinson's disease patient volunteers enrolled in phase 1 studies had documented hypotension following a 2-mg dose on two occasions. **Hallucinations:** In double-blind, placebo-controlled, early therapy studies in patients with Parkinson's disease who were not treated with L-dopa, 5.2% (8 of 157) of patients treated with REQUIP reported hallucinations, compared to 1.4% of patients on placebo (2 of 147). Among those patients receiving both REQUIP and L-dopa, in advanced Parkinson's disease (with L-dopa) studies, 10.1% (21 of 208) were reported to experience hallucinations, compared to 4.2% (5 of 120) of patients treated with placebo and L-dopa. Hallucinations were of sufficient severity to cause discontinuation of treatment in 1.3% of the early Parkinson's disease (without L-dopa) patients and 1.9% of advanced Parkinson's disease (with L-dopa) patients compared to 0% and 1.7% of placebo patients, respectively.

PRECAUTIONS: General: ***Dyskinesia:*** REQUIP may potentiate the dopaminergic side effects of L-dopa and may cause and/or exacerbate pre-existing dyskinesia. Decreasing the dose of L-dopa may ameliorate this side effect. ***Renal and Hepatic:*** No dosage adjustment is needed in patients with mild to moderate renal impairment (creatinine clearance of 30 to 50 mL/min.). Because the use of REQUIP in patients with severe renal or hepatic impairment has not been studied, administration of REQUIP to such patients should be carried out with caution. ***Events Reported with Dopaminergic Therapy:*** *Withdrawal-Emergent Hyperpyrexia and Confusion:* Although not reported with REQUIP, a symptom complex resembling the neuroleptic malignant syndrome (characterized by elevated temperature, muscular rigidity, altered consciousness, and autonomic instability), with no other obvious etiology, has been reported in association with rapid dose reduction, withdrawal of, or changes in anti-Parkinsonian therapy. *Fibrotic Complications:* Cases of retroperitoneal fibrosis, pulmonary infiltrates, pleural effusion, and pleural thickening have been reported in some patients treated with ergot-derived dopaminergic agents. While these complications may resolve when the drug is discontinued, complete resolution does not always occur. Although these adverse events are believed to be related to the ergoline structure of these compounds, whether other, nonergot derived dopamine agonists can cause them is unknown. In the development program for REQUIP, a 69 year-old man with obstructive lung disease was treated with REQUIP for 16 months and developed pleural thickening and effusion accompanied by lower extremity edema, cardiomegaly, pleuritic pain, and shortness of breath. Pleural biopsy demonstrated chronic inflammation and sclerosis. The effusion resolved after medical therapy and discontinuation of REQUIP. The patient was lost to follow-up. The relationship of these events to REQUIP cannot be established. ***Retinal pathology in albino rats:*** Retinal degeneration was observed in albino rats in the 2-year carcinogenicity study at all doses tested (equivalent to 0.6 to 20 times the maximum recommended human dose on a mg/m² basis), but was statistically significant at the highest dose (50 mg/kg/day). Additional studies to further evaluate the specific pathology (e.g., loss of photoreceptor cells) have not been performed. Similar changes were not observed in a 2-year carcinogenicity study in albino mice or in rats or monkeys treated for 1 year. The potential significance of this effect in humans has not been established, but cannot be disregarded because disruption of a mechanism that is universally present in vertebrates (e.g., disk shedding) may be involved. ***Binding to melanin:*** REQUIP binds to melanin-containing tissues (i.e., eyes, skin) in pigmented rats. After a single dose, long-term retention of drug was demonstrated, with a half-life in the eye of 20 days. It is not known if REQUIP accumulates in these tissues over time. **Information for Patients:** Patients should be instructed to take REQUIP only as prescribed. REQUIP can be taken with or without food. Since ingestion with food reduces the maximum concentration (C_{max}) of REQUIP, patients should be advised that taking REQUIP with food may reduce the occurrence of nausea. However, this has not been established in controlled clinical trials. Patients should be informed that hallucinations can occur and that the elderly are at a higher risk than younger patients with Parkinson's disease. Patients should be advised that they may develop postural (orthostatic) hypotension with or without symptoms such as dizziness, nausea, syncope, and sometimes sweating. Hypotension and/or orthostatic symptoms may occur more frequently during initial therapy or with an increase in dose at any time (cases have been seen after weeks of treatment). Accordingly, patients should be cautioned against rising rapidly after sitting or lying down, especially if they have been doing so for prolonged periods, and especially at the initiation of treatment with REQUIP. Patients should be alerted to the potential sedating effects associated with REQUIP including somnolence and the possibility of falling asleep while engaged in activities of daily living. Since somnolence is a frequent adverse event with potentially serious consequences, patients should neither drive a car nor engage in other potentially dangerous activities until they have gained sufficient experience with REQUIP to gauge whether or not it affects their mental and/or motor performance adversely. Patients should be advised that if increased somnolence or episodes of falling asleep during activities of daily living (e.g., watching television, passenger in a car, etc.) are experienced at any time during treatment, they should not drive or participate in potentially dangerous activities until they have contacted their physician. Because of possible additive effects, caution should be advised when patients are taking other sedating medications or alcohol in combination with REQUIP and when taking concomitant medications that increase plasma levels of ropinirole (e.g., ciprofloxacin). Because of the possible additive sedative effects, caution should also be used when patients are taking alcohol or other CNS depressants (e.g., benzodiazepines, antipsychotics, antidepressants, etc.) in combination with REQUIP. Because of the possibility that ropinirole may be excreted in breast milk, patients should be advised to notify their physicians if they intend to breast-feed or are breast-feeding an infant. Because ropinirole has been shown to have adverse effects on embryo-fetal development, including teratogenic effects, in animals, and because experience in humans is limited, patients should notify their physician if they become pregnant or intend to become pregnant during therapy (see PRECAUTIONS, Pregnancy). **Drug Interactions:** ***Pharmacokinetic Interaction:*** In vitro metabolism studies showed that CYP1A2 was the major enzyme responsible for metabolism of ropinirole. There is thus the potential for substrates or inhibitors of this enzyme when coadministered with ropinirole to alter its clearance. Therefore, if therapy with a drug known to be a potent inhibitor of CYP1A2 is stopped or started during treatment with REQUIP, adjustment of the dose of REQUIP may be required. ***L-dopa:*** Co-administration of carbidopa + L-dopa (Sinemet® 10/100 mg b.i.d.) with ropinirole (2.0 mg t.i.d.) had no effect on the steady-state pharmacokinetics of ropinirole (n = 28 patients). Oral administration of REQUIP 2.0 mg t.i.d. increased mean steady state C_{max} of L-dopa by 20% but its AUC was unaffected (n = 23 patients). ***Digoxin:*** Co-administration of REQUIP (2.0 mg t.i.d.) with digoxin (0.125-0.25 mg q.d.) did not alter the steady-state pharmacokinetics of digoxin in 10 patients. ***Theophylline:*** Administration of theophylline (300 mg b.i.d., a substrate of CYP1A2) did not alter the steady-state pharmacokinetics of ropinirole (2 mg t.i.d.) in 12 patients with Parkinson's disease. Ropinirole (2 mg t.i.d.) did not alter the pharmacokinetics of theophylline (5 mg/kg i.v.) in 12 patients with Parkinson's disease. ***Ciprofloxacin:*** Co-administration of ciprofloxacin (500 mg b.i.d.), an inhibitor of CYP1A2, with ropinirole (2 mg t.i.d.) increased ropinirole AUC by 84% on average, and C_{max} by 60% (n = 12 patients). ***Estrogens:*** Population pharmacokinetic analysis revealed that estrogens (mainly ethinylestradiol: intake 0.6-3 mg over 4-month to 23-year period) reduced the oral clearance of ropinirole by 38% in 16 patients. Dosage adjustment may not be needed for REQUIP in patients on estrogen therapy because patients must be carefully titrated with ropinirole to tolerance or adequate effect. However, if estrogen therapy is stopped or started during treatment with REQUIP, then adjustment of the dose of REQUIP may be required. ***Dopamine Antagonists:*** Since ropinirole is a dopamine agonist, it is possible that dopamine antagonists, such as neuroleptics (phenothiazines, butyrophenones, thioxanthenes) or metoclopramide, may diminish the effectiveness of REQUIP. Patients with major psychotic disorders treated with neuroleptics should only be treated with dopamine agonists if the potential benefits outweigh the risks. Population analysis showed that commonly administered drugs, e.g., selegiline, amantadine, tricyclic antidepressants, benzodiazepines, ibuprofen, thiazides, antihistamines, and anticholinergics did not affect the oral clearance of ropinirole. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Two-year carcinogenicity studies were conducted in Charles River CD-1 mice at doses of 5, 15, and 50 mg/kg/day and in Sprague-Dawley rats at doses of 1.5, 15, and 50 mg/kg/day (top doses equivalent to 10 times and 20 times, respectively, the maximum recommended human dose of 24 mg/day on a mg/m² basis). In male rats, there was a significant increase in testicular Leydig cell adenomas at all doses tested, i.e., ≥1.5 mg/kg (0.6 times the maximum recommended human dose on a mg/m² basis). This finding is of questionable significance because the endocrine mechanisms believed to be involved in the production of Leydig cell hyperplasia and adenomas in rats are not relevant to humans. In the female mouse, there was an increase in benign uterine endometrial polyps at a dose of 50 mg/kg/day (10 times the maximum recommended human dose on a mg/m² basis). Ropinirole was not mutagenic or clastogenic in the *in vitro* Ames test, the *in vitro* chromosome aberration test in human lymphocytes, the *in vitro* mouse lymphoma (L1578Y cells) assay, and the *in vitro* mouse micronucleus test. When administered to female rats prior to and during mating and throughout pregnancy, ropinirole caused disruption of implantation at doses of 20 mg/kg/day (8 times the maximum recommended human dose on a mg/m² basis) or greater. This effect is thought to be due to the prolactin-lowering effect of ropinirole. In humans, chorionic gonadotropin, not prolactin, is essential for implantation. In rat studies using low doses (5 mg/kg) during the prolactin-dependent phase of early pregnancy (gestation days 0-8), ropinirole did not affect female fertility at dosages up to 100 mg/kg/day (40 times the maximum recommended human dose on a mg/m² basis). No effect on male fertility was observed in rats at dosages up to 125 mg/kg/day (50 times the maximum recommended human dose on a mg/m² basis). **Pregnancy: Pregnancy Category C:** In animal reproduction studies, ropinirole has been shown to have adverse effects on embryo-fetal development, including teratogenic effects. Ropinirole given to pregnant rats during organogenesis (20 mg/kg on gestation days 6 and 7 followed by 20, 60, 90, 120 or 150 mg/kg on gestation days 8 through 15) resulted in decreased fetal body weight at 60 mg/kg/day, increased fetal death at 90 mg/kg/day and digital malformations at 150 mg/kg/day (24, 36, and 60 times the maximum recommended clinical dose on a mg/m² basis, respectively). The combined administration of ropinirole (10 mg/kg/day, 8 times the maximum recommended human dose on a mg/m² basis) and L-dopa (250 mg/kg/day) to pregnant rabbits during organogenesis produced a greater incidence and severity of fetal malformations (primarily digit defects) than were seen in the offspring of rabbits treated with L-dopa alone. No indication of an effect on development of the conceptus was observed in rabbits when a maternally toxic dose of ropinirole was administered alone (20 mg/kg/day; 16 times the maximum recommended human dose on a mg/m² basis). In a perinatal-postnatal study in rats, 10 mg/kg/day (4 times the maximum recommended human dose on a mg/m² basis) of ropinirole impaired growth and development of nursing offspring and altered neurological development of female offspring. There are no adequate and well-controlled studies using REQUIP in pregnant women. REQUIP should be used during pregnancy only if the potential benefit outweighs the potential risk to the fetus. Nursing Mothers: REQUIP inhibits prolactin secretion in humans and could potentially inhibit lactation. Studies in rats have shown that REQUIP and/or its metabolite(s) is excreted in breast milk. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from REQUIP a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness in the pediatric population have not been established.

ADVERSE REACTIONS: During the pre-marketing development of REQUIP, patients received REQUIP either without L-dopa (early Parkinson's disease studies) or as concomitant therapy with L-dopa (advanced Parkinson's disease studies). Because these 2 populations may have differential risks for various adverse events, this section will, in general, present adverse event data for these 2 populations separately. The prescriber should be aware that the following figures cannot be used to predict the incidence of adverse events in the course of usual medical practice where patient characteristics and other factors differ from those that prevailed in the clinical studies. Similarly, the cited frequencies cannot be compared with figures obtained from other clinical investigations involving different treatments, uses and investigators. However, the cited figures do provide the prescribing physician with some basis for estimating the relative contribution of drug and non-drug factors to the adverse-events incidence rate in the population studied. **Early Parkinson's disease (without L-dopa):** The most commonly observed adverse events (≥5%) in the double-blind, placebo-controlled early Parkinson's disease trials associated with the use of REQUIP (n = 157) not seen at an equivalent frequency among the placebo-treated patients (n = 147) were, in order of decreasing incidence: nausea, dizziness, somnolence, headache, vomiting, syncope, fatigue, dyspepsia, viral infection, constipation, pain, increased sweating, asthenia, dependent leg edema, orthostatic symptoms, abdominal pain, pharyngitis, confusion, hallucinations, urinary tract infections, and abnormal vision. Approximately 24% of 157 patients treated with REQUIP who participated in the double-blind, placebo-controlled early Parkinson's disease (without L-dopa) trials discontinued treatment due to adverse events compared to 13% of 147 patients who received placebo. The adverse events most commonly causing discontinuation of treatment by patients treated with REQUIP were: nausea (6.4%), dizziness (3.8%), aggravated Parkinson's disease (1.3%), hallucinations (1.3%), somnolence (1.3%), vomiting (1.3%), and headache (1.3%). Of these, hallucinations appear to be dose-related. While other adverse events leading to discontinuation may be dose-related, the titration design utilized in these trials precluded an adequate assessment of the dose response. Treatment-emergent adverse events that occurred in ≥2% of patients with early Parkinson's disease (without L-dopa) treated with REQUIP participating in the double-blind, placebo-controlled studies and were numerically more common in the group treated with REQUIP are listed below in order of decreasing incidence: nausea (60% vs. 22%), dizziness (40% vs. 22%), somnolence (40% vs. 6%), vomiting (12% vs. 7%), syncope (12% vs. 1%), fatigue (11% vs. 4%), viral infection (11% vs. 3%), dyspepsia (10% vs. 5%), pain (8% vs. 4%), leg edema (7% vs. 1%), orthostatic symptoms (6% vs. 1%), increased sweating (6% vs. 4%), pharyngitis (6% vs. 4%), abnormal vision (6% vs. 3%), dependent edema (6% vs. 3%), abdominal pain (6% vs. 3%), asthenia (6% vs. 1%), urinary tract infections (5% vs. 4%), dry mouth (5% vs. 3%), hypertension (5% vs. 3%), confusion (5% vs. 1%), hallucinations (5% vs. 1%), rhinitis (4% vs. 3%), sinusitis (4% vs. 3%), chest pain (4% vs. 2%), hypesthesia (4% vs. 2%), anorexia (4% vs. 1%), palpitation (3% vs. 2%), flushing (3% vs. 1%), malaise (3% vs. 1%), flatulence (3% vs. 1%), increased alkaline phosphatase (3% vs. 1%), amnesia (3% vs. 1%), impotence (3% vs. 1%), bronchitis (3% vs. 1%), eye abnormality (3% vs. 1%), yawning (3% vs. 0%), dyspnea (3% vs. 0%), peripheral ischemia (3% vs. 0%), hyperkinesia (2% vs. 1%), extrasystoles (2% vs. 1%), hypotension (2% vs. 0%), vertigo (2% vs. 0%), atrial fibrillation (2% vs. 0%), tachycardia (2% vs. 0%), impaired concentration (2% vs. 0%), xerophthalmia (2% vs. 0%). Other events reported by 1% or more of early Parkinson's disease (without L-dopa) patients treated with REQUIP, but that were equally or more frequent in the placebo group were: headache, upper respiratory infection, insomnia, arthralgia, tremor, back pain, anxiety, dyskinesias, aggravated Parkinsonism, depression, falls, myalgia, leg cramps, paresthesias, nervousness, diarrhea, arthritis, hot flushes, weight loss, rash, cough, hyperglycemia, muscle spasm, arthrosis, abnormal dreams, dystonia, increased salivation, bradycardia, gout, basal cell carcinoma, gingivitis, hematuria, and rigors. Among the treatment-emergent adverse events in patients treated with REQUIP, hallucinations appear to be dose-related. The incidence of adverse events was not materially different between women and men. **Advanced Parkinson's disease (with L-dopa):** The most commonly observed adverse events (≥5%), in the double-blind, placebo-controlled advanced Parkinson's disease (with L-dopa) trials associated with the use of REQUIP (n = 208) as an adjunct to L-dopa not seen at an equivalent frequency among the placebo-treated patients (n = 120) were, in order of decreasing incidence: dyskinesias, nausea, dizziness, aggravated Parkinsonism, somnolence, headache, insomnia, injury, hallucinations, falls, abdominal pain, upper respiratory infection, confusion, increased sweating, vomiting, viral infection, increased drug level, arthralgia, tremor, anxiety, urinary tract infection, constipation, dry mouth, pain, hypokinesia, and paresthesia. Approximately 24% of 208 patients who received REQUIP in the double-blind, placebo-controlled advanced Parkinson's disease (with L-dopa) trials discontinued treatment due to adverse events compared to 18% of 120 patients who received placebo. The events most commonly (≥1%) causing discontinuation of treatment by patients treated with REQUIP were: dizziness (2.9%), dyskinesias (2.8%), vomiting (2.4%), confusion (2.4%), nausea (1.9%), hallucinations (1.9%), anxiety (1.9%), and increased sweating (1.8%). Of these, hallucinations and dyskinesias appear to be dose-related. Treatment-emergent adverse events that occurred in ≥2% of patients with advanced Parkinson's disease (with L-dopa) treated with REQUIP participating in the double-blind, placebo-controlled studies and were numerically more common in the group treated with REQUIP were in order of decreasing incidence: dyskinesias (34% vs. 13%), nausea (30% vs. 18%), dizziness (26% vs. 16%), somnolence (20% vs. 8%),

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headache (17% vs. 12%); falls (10% vs. 7%); hallucinations (10% vs. 4%); abdominal pain (9% vs. 8%); upper respiratory infection (9% vs. 8%); confusion (9% vs. 2%); arthralgia (7% vs. 5%); vomiting (7% vs. 4%); increased drug level (7% vs. 3%); increased sweating (7% vs. 2%); tremor (6% vs. 3%); anxiety (6% vs. 3%); urinary tract infection (6% vs. 3%); exacerbation (6% vs. 3%); hypokinesia (5% vs. 4%); pain (5% vs. 3%); paresthesia (5% vs. 3%); diarrhea (5% vs. 3%); nervousness (5% vs. 3%); dry mouth (5% vs. 1%); amnesia (5% vs. 1%); syncope (3% vs. 2%); abnormal dreaming (3% vs. 2%); dyspnea (3% vs. 2%); arthritis (3% vs. 1%); paresis (3% vs. 0%); hypotension (2% vs. 1%); dysphagia (2% vs. 1%); flatulence (2% vs. 1%); increased saliva (2% vs. 1%); weight decrease (2% vs. 1%); pyuria (2% vs. 1%); urinary incontinence (2% vs. 1%); diplopia (2% vs. 1%); and anemia (2% vs. 0%). Other events reported by 1% or more of patients treated with both REQUIP and L-dopa, but equally or more frequent in the placebo/L-dopa group were: myocardial infarction, orthostatic symptoms, virus infections, asthenia, dyspepsia, myalgia, back pain, depression, leg cramps, fatigue, rhinitis, chest pain, hematuria, vertigo, tinnitus, leg edema, hot flushes, abnormal gait, hyperkinesia, and pharyngitis. Among the treatment-emergent adverse events in patients treated with REQUIP, hallucinations and dyskinesias appear to be dose-related.

Other Adverse Events Observed During All Phase 2/3 Clinical Trials: REQUIP has been administered to 1,599 individuals in clinical trials. During these trials, all adverse events were recorded by the clinical investigators using terminology of their own choosing. To provide a meaningful estimate of the proportion of individuals having adverse events, similar types of events were grouped into a smaller number of standardized categories using modified WHOART dictionary terminology. These categories are used in the listing below. The frequencies presented represent the proportion of the 1,599 individuals exposed to REQUIP who experienced events of the type cited on at least one occasion while receiving REQUIP. All reported events that occurred at least twice (or once for serious or potentially serious events), except those already listed above, trivial events, and terms too vague to be meaningful are included, without regard to determination of a causal relationship to REQUIP, except that events very unlikely to be drug-related have been deleted. Events are further classified within body system categories and enumerated in order of decreasing frequency using the following definitions: frequent adverse events are defined as those occurring in at least 1/100 patients and infrequent adverse events are those occurring in 1/100 to 1/1000 patients and rare events are those occurring in fewer than 1/1000 patients. ***Body as a Whole:*** *infrequent* – cellulitis, peripheral edema, fever, influenza-like symptoms, enlarged abdomen, precordial chest pain, and generalized edema; *rare* – ascites. ***Cardiovascular:*** *infrequent* – cardiac failure, bradycardia, tachycardia, supraventricular tachycardia, angina pectoris, bundle branch block, cardiac arrest, cardiomegaly, aneurysm, mitral insufficiency; *rare* – ventricular tachycardia. ***Central/Peripheral Nervous System:*** *frequent* – neuralgia; *infrequent* – involuntary muscle contractions, hypertonia, dysphonia, abnormal coordination, extrapyramidal disorder, migraine, choreoathetosis, coma, stupor, aphasia, convulsions, hypotonia, peripheral neuropathy, paralysis; *rare* – grand mal convulsions, hemiparesis, hemiplegia. ***Endocrine:*** *infrequent* – hypothyroidism, gynecomastia, hyperthyroidism; *rare* – goiter, SIADH. ***Gastrointestinal:*** *infrequent* – increased hepatic enzymes, bilirubinemia, cholecystitis, cholelithiasis colitis, dysphagia, periodontitis, fecal incontinence, gastroesophageal reflux, hemorrhoids, toothache, eructation, gastritis, esophagitis, hiccups, diverticulitis, duodenal ulcer, gastric ulcer, melena, duodenitis, gastrointestinal hemorrhage, glossitis, rectal hemorrhage, pancreatitis, stomatitis and ulcerative stomatitis, tongue edema; *rare* – biliary pain, hemorrhagic gastritis, hematemesis, salivary duct obstruction. ***Hematologic:*** *infrequent* – purpura, thrombocytopenia, hematoma, Vitamin B12 deficiency, hypochromic anemia, eosinophilia, leukocytosis, leukopenia, lymphocytosis, lymphopenia, lymphedema. ***Metabolic/Nutritional:*** *frequent* – increased BUN; *infrequent* – hypolycemia, increased alkaline phosphatase, increased LDH, weight increase, hyperphosphatemia, hyperuricemia, diabetes mellitus, glycosuria, hypokalemia, hypercholesterolemia, hyperkalemia, acidosis, hyponatremia; *thirst, increased CPK, dehydration; rare* – hypochloremia. ***Musculoskeletal:*** *infrequent* – aggravated arthritis, tendinitis, osteoporosis, bursitis, polymyalgia rheumatica, muscle weakness, skeletal pain, torticollis; *rare* – Dupuytren's contracture requiring surgery. ***Neoplasm:*** *infrequent* – malignant breast neoplasm; *rare* – bladder carcinoma, benign brain neoplasm, esophageal carcinoma, malignant laryngeal neoplasm, lipoma, rectal carcinoma, uterine neoplasm. ***Psychiatric:*** *infrequent* – increased libido, agitation, apathy, impaired concentration, depersonalization, paranoid reaction, personality disorder, euphoria, delirium, dementia, delusion, emotional lability, decreased libido, manic reaction, somnambulism, aggressive reaction, neurosis; *rare* – suicide attempt. ***Genito-urinary:*** *infrequent* – amenorrhea, vaginal hemorrhage, penile disorder, prostatic disorder, salpingitis, epididymitis, perineal pain, dysuria, micturition frequency, albuminuria, nocturia, polyuria, renal calculus; *rare* – breast enlargement, mastitis, uterine hemorrhage, ejaculation disorder, Peyronie's Disease, pyelonephritis, acute renal failure, uremia. ***Resistance Mechanism:*** *infrequent* – herpes zoster, otitis media, sepsis, abscess, herpes simplex, fungal infection, genital moniliasis. ***Respiratory:*** *infrequent* – asthma, epistaxis, laryngitis, pleurisy, pulmonary edema. ***Skin/Appendage:*** *infrequent* – pruritis, dermatitis, eczema, skin ulceration, alopecia, skin hypertrophy, skin discoloration, urticaria, fungal dermatitis, furunculosis, hypokeratosis, photosensitivity reaction, psoriasis, maculopapular rash, psoriform rash, seborrhea. ***Special Senses:*** *infrequent* – tinnitus, earache, decreased hearing, abnormal lacrimation, conjunctivitis, blepharitis, glaucoma, abnormal accommodation, blepharospasm, eye pain, photophobia; *rare* – scotoma. ***Vascular Extracardiac:*** *infrequent* – varicose veins, phlebitis, peripheral gangrene; *rare* – limb embolism, pulmonary embolism, gangrene, subarachnoid hemorrhage, deep thrombophlebitis, leg thrombophlebitis, thrombosis.

Falling Asleep During Activities of Daily Living: Patients treated with REQUIP have reported falling asleep while engaged in activities of daily living, including operation of a motor vehicle which sometimes resulted in accidents (see boxed WARNING).

OVERDOSAGE: There were no reports of intentional overdose of REQUIP in the premarketing clinical trials. 27 patients accidentally took more than their prescribed dose of REQUIP, with 16 patients ingesting more than 24 mg/day. The largest overdose reported in premarketing clinical trials was 405 mg taken over a 7-day period (62.1 mg/day). Of patients who received a dose greater than 24 mg/day, one experienced mild oro-facial dyskinesia, another patient experienced intermittent nausea. Other symptoms reported with accidental overdoses were: agitation, increased dyskinesia, grogginess, sedation, orthostatic hypotension, chest pain, confusion, vomiting and nausea. **Overdose Management:** Symptoms of REQUIP overdose are likely to be related to its dopaminergic activity. General supportive measures are recommended. Maintain vital signs and consider removal of any unabsorbed material (e.g., by gastric lavage).

DOSAGE AND ADMINISTRATION: The dosage should be gradually increased to achieve a maximum therapeutic effect, balanced against the principal side effects of nausea, dizziness, somnolence and dyskinesia. REQUIP should be taken three times daily. REQUIP can be taken with or without food. Since ingestion with food reduces the maximum concentration (C_{max}) of REQUIP, tell patients that taking REQUIP with food may reduce the occurrence of nausea. However, this has not been established in controlled clinical trials. The recommended starting dose is 0.25 mg three times daily. Based on individual patient response, dosage should then be titrated as described in the table below. After week 4, if necessary, increase daily dosage by 1.5 mg per day on a weekly basis up to a dose of 9 mg per day, and then by up to 3 mg per day weekly to a total dose of 24 mg per day.

Ascending-Dose Schedule of Requip

Week	Dosage	Total Daily Dose
1	0.25 mg three times daily	0.75 mg
2	0.5 mg three times daily	1.5 mg
3	0.75 mg three times daily	2.25 mg
4	1.0 mg three times daily	3.0 mg

doses greater than 24 mg/day have not been tested in clinical trials. When REQUIP is administered as adjunct therapy to L-dopa, decrease the concurrent L-dopa dose gradually as tolerated. L-dopa dosage reduction was allowed during the advanced Parkinson's disease (with L-dopa) study if dyskinesias or other dopaminergic effects occurred. Overall, reduction of L-dopa dose was sustained in 67% of patients treated with REQUIP and in 57% of patients on placebo. On average, the L-dopa dose was reduced by 31% in patients treated with REQUIP. Discontinue REQUIP gradually over a 7-day period. Reduce the frequency of administration from three times daily to twice daily for 4 days. For the remaining 3 days, reduce the frequency to once daily prior to complete withdrawal of REQUIP.

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Research Triangle Park, NC 27709

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SCIENTIFIC PROGRAM

TUESDAY, JUNE 15, 2004

8:30 am to 10:30 am

Parallel Session 1: Platform Presentations: Parkinson's disease

Location: Salone Della Cultura, Ground Floor

Chair: Cynthia Comella
Chicago, IL, USA

Co-chair: Wolfgang Oertel
Marburg, Germany

Identification of PARK6, a novel mitochondrial protein causing Parkinson's disease
Enza Maria Valente
Abstract Number: P1042

Endocannabinoid levels are altered in parkinsonism and L-DOPA-induced dyskinesia in the MPTP-lesioned macaque
Susan Fox
Abstract Number: P1163

Parkinsonian signs in older people in the community and risk of incident dementia: A prospective longitudinal population-based study
Elan Louis
Abstract Number: P954

Combined use of NMDA and AMPA antagonists further reduces levodopa-induced dyskinesias in MPTP-lesioned primates
Francesco Bibbiani
Abstract Number: P606

Predicting incident non-motor complications of dopaminergic therapy in patients with early Parkinson's disease: A secondary analysis of the CALM-PD trial
Kevin Biglan
Abstract Number: P576

Is levodopa-induced dyskinesias risk decreased in parkinsonian patients initially treated with dopamine agonist? A longitudinal study among 425 patients
Franck Durif
Abstract Number: P608

Neuronal activity of *zona incerta* in Parkinson's disease patients
Marcelo Merello
Abstract Number: P894

Predicting success after deep brain stimulation placement in the subthalamic nucleus in Parkinson's disease patients
Roy Bakay
Abstract Number: P947

Parallel Session 2: Platform Presentations: Other Movement Disorders

Location: Assembly Hall, Ground Floor

Chair: Peter Riederer
Wuerzburg, Germany

Co-chair: Murat Emre
Capa Istanbul, Turkey

Nicotine corrects impaired motor-motor and afferent sensory inhibition in patients with Gilles de la Tourette syndrome
Michael Orth
Abstract Number: P156

Is Obsessive Compulsive Disorder (OCD) a sensorimotor integration dysfunction? Evidence from a gating study in a SEP paradigm
Simone Rossi
Abstract Number: P124

Long term prognosis of psychogenic Movement Disorders
Madhavi Thomas
Abstract Number: P1263

Misdiagnosis of fragile X associated tremor/ataxia syndrome (FXTAS)
Maureen Leehey
Abstract Number: P985

Characteristics of two distinct clinical phenotypes observed in pathologically proven progressive supranuclear palsy: Richardson's syndrome and PSP-Parkinsonism
David Williams
Abstract Number: P955

Pallidal stimulation to treat tardive dyskinesia: Preliminary report of the French multicentric study STARDYS
Philippe Damier
Abstract Number: P900

Gait and motor disturbances are correlated with age-related white matter changes - Cross-sectional results of the LADIS (Leukoaraiosis And DISability) project
Hansjoerg Baezner
Abstract Number: P982

Experimental evidence for a toxic etiology of Guadeloupean parkinsonism
Annie Lannuzel
Abstract Number: P977

10:30 am to 11:30 am

MDS Business Meeting

Location: Salone Della Cultura, Ground Floor

11:30 am to 12:30 pm

Abstract Poster Session 2

Location: Poster Area, First Floor

Abstract Numbers 345-694
Authors present odd numbers

12:30 pm to 1:30 pm

Lunch

Location: Rooftop Terrace and Various Locations

1:30 pm to 4:00 pm

Parallel Session 3: Cognitive and behavioral dysfunction in Movement Disorders

Location: Salone Della Cultura, Ground Floor

Chair: Bruno Dubois
Paris, France

Co-chair: I.G. McKeith
Newcastle Upon Tyne, United Kingdom

Cognitive changes and dementia in Parkinson's disease
Murat Emre
Istanbul, Turkey

Anatomical and physiological basis of cognitive and behavioral changes in Movement Disorders
Peter Strick
Pittsburg, PA, USA

Motivation, apathy and the basal ganglia
Richard Levy
Paris, France

Executive function and basal ganglia
Adrian Owen
Cambridge, United Kingdom

Reward and the basal ganglia
Mandar Jog
London, Canada

At the conclusion of this session, participants should be able to: 1. Describe the pattern of cognitive changes and dementia associated with Parkinson's disease and related disorders; 2. Recognize the role of the basal ganglia in the regulation of motivation and the mechanism of apathy in patients with lesions of the basal ganglia; 3. Recognize the involvement of the basal ganglia in executive functions and other frontal lobe-related processes.

SCIENTIFIC PROGRAM

Parallel Session 4: Update on other Movement Disorders

Location: Assembly Hall, Ground Floor

Chair: Ira Shoulson
Rochester, NY, USA

Co-chair: Anne Young
Boston, MA, USA

Update on dystonia
Enza Maria Valente
Rome, Italy

Update on Huntington's disease
Elena Cattaneo
Milano, Italy

Update on Friedreich's ataxia
Anthony H. V. Schapira
London, United Kingdom

Update on psychogenic Movement Disorders
Mark Hallett
Bethesda, MD, USA

Update on essential tremor
Elan Louis
New York, NY, USA

At the conclusion of this session, participants should be able to: 1 Describe the latest research in Huntington's disease; 2. Describe the latest update in essential tremor; 3. Describe the latest research in dystonia; 4. Describe the latest update in psychogenic Movement Disorders; 5. Describe the latest update in essential tremor.

4:00 pm to 5:00 pm

Abstract Poster Session 2

Location: Poster Area, First Floor

Abstract Numbers 345-694
Authors present even numbers

5:00 pm to 6:30 pm

Seminar Series

Sessions featuring Italian cuisine are featured on Tuesday. Each session offers an expert's view on Movement Disorders through a variety of topics. To encourage discussion and interaction, Seminar Series have limited registration and a ticket is required for admission.

Fee: $55 USD/ $40 USD for junior participants and allied health professionals.

S201 Sleep problems in Parkinson's disease

Location: Meeting Room 1, First Floor

Paolo Barone
Napoli, Italy

David Rye
Atlanta, GA, USA

S202 Rare genetic Movement Disorders (PKAN, Wilson's, acanthocytosis, etc.)

Location: Meeting Room 3, First Floor

Peter LeWitt
Southfield, MI, USA

Kailash Bhatia
London, United Kingdom

At the conclusion of this session, participants should be able to: 1. Recognize typical clinical features of several rare genetic Movement Disorders, including Wilson's disease, neuroacanthocytosis, PKAN, neuroferritinopathy and others; 2. Discuss the diagnostic options for differentiating these Movement Disorders, and define these various available genetic tests; 3. Indicate the available management strategies and the natural history of these Movement Disorders.

S203 Facial dyskinesias

Location: Meeting Room 5, First Floor

Ryuji Kaji
Tokushima City, Japan

Josep Valls-Solé
Barcelona, Spain

At the conclusion of this session, participants should be able to: 1. Describe the most relevant syndromes presenting with facial dyskinesias; 2. Recognize the most relevant clinical and electrophysiological features that characterize each of the syndromes presenting with facial dyskinesias; 3. Discuss the key electrophysiological features useful for differential diagnosis between disorders presenting with facial dyskinesias.

S204 Dementia with Lewy bodies

Location: Meeting Room 6, First Floor

Daniel Perl
New York, NY, USA

David John Burn
Newcastle Upon Tyne, United Kingdom

S205 Magnetic stimulation in Movement Disorders

Location: Meeting Room 7, Ground Floor

Antonio Currà
Rome, Italy

Robert Chen
Toronto, Canada

At the conclusion of this session, participants should be able to: 1. Describe basic principles, utility, safety and limitations of transcranial magnetic stimulation (TMS); 2. Discuss the main TMS findings in common Movement Disorders such a Parkinson's disease, dystonia, chorea, tremor, myoclonus and tics; 3. Identify the current and possible future clinical and research applications of TMS in Movement Disorders.

S206 Epidemiology and genetics of Parkinson's disease

Location: Meeting Room 8, Ground Floor

Vincenzo Bonifati
Rome, Italy

Caroline Tanner
Sunnyvale, CA, USA

At the conclusion of this session, participants should be able to: 1. Describe the demographics and international distribution of Parkinson's disease; 2. Describe the genetic determinants of parkinsonism; 3. Understand the factors proposed to increase or decrease susceptibility to developing Parkinson's disease.

S207 The New UPDRS

Location: Meeting Room 2, First Floor

Stanley Fahn
New York, NY, USA

Christopher G. Goetz
Chicago, IL, USA

At the conclusion of this session, participants should be able to: 1. Define the hallmarks of the original and new versions of the UPDRS; 2. Identify the new changes and their rationales; 3. Recognize the plans for clinimetric testing of the new UPDRS so that the old and new versions can be compared.

S208 Principles of animal models in Movement Disorders

Location: Meeting Room 4, First Floor

Ted M. Dawson
Baltimore, MD, USA

Jie Shen
Boston, MA, USA

7:00 pm to 9:00 pm

Video Dinners

Video presentations of atypical Movement Disorders engage delegates and generate clinical discussions. To ensure greater interaction, Video Dinners are limited to a maximum number of participants, and a ticket is required for admission. As the title indicates, dinner is served during the sessions.

Fee: $80 USD/ $55 USD for junior participants and allied health professionals.

V101 Atypical parkinsonism

Location: Meeting Room 1, First Floor

Eduardo Tolosa
Barcelona, Spain

Niall Quinn
London, United Kingdom

At the conclusion of this session, participants should be able to: 1. Recognize clinical features suggestive of atypical parkinsonism; 2. Identify individual specific causes of atypical parkinsonism; 3. Discuss the differential diagnosis between different causes of atypical parkinsonism.

V102 Dystonia

Location: Meeting Room 3, First Floor

Susan Bressman
Englewood, NJ, USA

Joseph Jankovic
Houston, TX, USA

At the conclusion of this session, participants should be able to: 1. Recognize the phenomenology of generalized, segmental and focal dystonia, as illustrated by videos; 2. Discuss the etiologic, including genetic, classification of dystonia; 3. Discuss therapeutic strategies in dystonia, including pharmacological, chemodeneration, and surgical approaches.

V103 Gait disorders

Location: Meeting Room 2, First Floor

John Nutt
Portland, OR, USA

Roger Elble
Springfield, IL, USA

At the conclusion of this session, participants should be able to: 1. Describe the clinical differences between highest-level and lower-level gait disorders; 2. Recognize the common and uncommon gait disorders caused by pathology of the central and peripheral nervous system; 3. Discuss unusual gait disorders submitted by anyone attending the seminar.

V104 Myoclonus/startle and other jerks

Location: Meeting Room 4, First Floor

Steven Frucht
New York, NY, USA

Hiroshi Shibasaki
Bethesda, MD, USA

At the conclusion of this session, participants should be able to: 1. Recognize the major forms of myoclonus and startle syndromes; 2. List the possible etiologies of form of myoclonus and startle; 3. Describe the various treatment options for these disorders.

SCIENTIFIC PROGRAM

WEDNESDAY, JUNE 16, 2004

8:30 am to 11:00 am
Plenary Session 3: Experimental interventional therapeutics for Movement Disorders
Location: Salone Della Cultura, Ground Floor

Chair: Olle Lindvall
Lund, Sweden

Co-chair: John Nutt
Portland, OR, USA

Gene therapy
Jeffrey Kordower
Chicago, IL, USA

Stem cells
Ole Isacson
Belmont, MA, USA

Transplantation strategies
Patrik Brundin
Lund, Sweden

Trophic factors
Clive Svendsen
Madison, WI, USA

Clinical point of view
Olle Lindvall
Lund, Sweden

John Nutt
Portland, OR, USA

At the conclusion of this session, participants should be able to: 1. Describe the three basic mechanisms of neurorestorative therapies, namely gene therapy, neural grafting and administration of neurotrophic factors; 2. Discuss the advantages and disadvantages of stem cells relative to fetal cells for neural grafting; 3. List techniques to deliver genes and neurotropic factors to the central nervous system.

11:00 am to 11:30 am
Stanley Fahn Lecture
Molecular pathogenesis of dominately inherited ataxias
Location: Salone Della Cultura, Ground Floor

Huda Zoghbi
Houston, TX, USA

11:30 am to 12:30 pm
Abstract Poster Session 3
Location: Poster Area, First Floor

Abstract Numbers 695-1017
Authors present odd numbers

12:30 pm to 1:30 pm
Lunch
Location: Rooftop Terrace and Various Locations

1:30 pm to 4:00 pm
Plenary Session 4: Modern concepts in the diagnosis and treatment of parkinsonism
Location: Salone Della, Cultura, Ground Floor

Chair: Anthony E. Lang
Toronto, Canada

Co-chair: Joseph Jankovic
Houston, TX, USA

Neuroprotective trials in Parkinson's disease: design issues and prospects
Karl Kieburtz
Rochester, NY, USA

New approaches in symptomatic treatment
Olivier Rascol
Toulouse, France

Atypical parkinsonism
Andrew Lees
London, United Kingdom

New developments in neuroimaging
A. Jon Stoessl
Vancouver, Canada

Parkinsonism and dementia
David Burn
Newcastle Upon Tyne, United Kingdom

At the conclusion of this session, participants should be able to: Identify the possible targets for neuroprotection in Parkinson's disease and understand the research design issues that must be considered in evaluating putative neuroprotective and disease-modifying strategies; 2. Describe the clinical and neuropathological aspects of disorders presenting as atypical parkinsonism and dementia associated with parkinsonism and discuss the approaches available to diagnosis and management of these disorders; 3. Discuss new developments in the neuroimaging of parkinsonian disorders and new approaches to the symptomatic treatment of Parkinson's disease.

4:00 pm to 5:00 pm
Abstract Poster Session 3
Location: Poster Area, First Floor

Abstract Numbers 695-1017
Authors present even numbers

5:00 pm to 6:30 pm
Seminar Series
Sessions featuring Italian cuisine are featured on Wednesday. Each session offers an expert's view on Movement Disorders through a variety of topics. To encourage discussion and interaction, Seminar Series have limited registration and a ticket is required for admission.

Fee: $55 USD/ $40 USD for junior participants and allied health professionals.

S301 Clinical/epidemiology of dystonia

Location: Meeting Room 5, First Floor

Thomas Warner
London, United Kingdom

Gianni Defazio
Bari, Italy

At the conclusion of this session, participants should be able to: 1. Recognize and diagnose the various clinical forms of dystonia; 2. Describe the epidemiology and prevalence of dystonia; 3. Identify the genetic and environmental risk factors that lead to dystonia.

S302 Systemic and infectious diseases that cause Movement Disorders

Location: Meeting Room 1, First Floor

Jorge Luis Juncos
Atlanta, GA, USA

Francisco Cardoso
Belo Horizonte MG, Brazil

At the conclusion of this session, participants should be able to: 1. Describe the phenomenology of Movement Disorders associated with infectious diseases; 2. List the infectious agents that can cause Movement Disorders; 3. Discuss the management of Movement Disorders associated with infectious diseases.

S303 Drug induced Movement Disorders

Location: Meeting Room 3, First Floor

William Weiner
Baltimore, MD, USA

Daniel Tarsy
Boston, MA, USA

At the conclusion of this session, participants should be able to: 1. Recognize the Movement Disorders caused by antipsychotic drugs, antidepressants, stimulants, lithium and other medications; 2. Discuss the pathophysiologic basis for the antipsychotic drug-induced Movement Disorders; 3. Discuss the prevention and management of drug-induced Movement Disorders.

S304 Parkinsonism - MSA: clinical update

Location: Meeting Room 6, First Floor

Gregor Wenning
Innsbruck, Austria

Irene Litvan
Louisville, KY, USA

At the conclusion of this session participants should be able to: 1. Describe typical and atypical presentations of MSA; 2. Describe appropriate investigations; 3. Describe therapeutic management.

S305 New developments in the pathology of Parkinson's disease

Location: Meeting Room 7, Ground Floor

Heiko Braak
Frankfurt, Germany

Glenda Halliday
Randwick, Australia

At the conclusion of this session, participants should be able to: 1. Identify the main cellular pathologies found in idiopathic Parkinson's disease, discuss their intracellular origins and determine any relationship between them; 2. Describe the new neuropathological staging scheme for idiopathic Parkinson's disease identify the brain regions involved, and discuss their clinical significance; 3. Identify the cortical, basal ganglia and thalamic regions involved in movement control, discuss their functional connectivity, identify all pathological abnormalities in the circuits in idiopathic Parkinson's disease and discuss their clinical significance.

S306 Young onset parkinsonism

Location: Meeting Room 4, First Floor

Anette Schrag
London, United Kingdom

Christoph Lücking
Munich, Germany

At the conclusion of this session, participants should be able to: 1. Describe to clinical and neuropathological characteristics of young onset Parkinson's disease; 2. Describe the role of genetics in Parkinson's disease (with particular reference to young onset Parkinson's disease); 3. Discuss the molecular pathophysiology of Parkinson's disease based on the genes involved.

S307 Ubiquitin proteasome system in Parkinson's disease

Location: Meeting Room 2, First Floor

Mark Cookson
Bethesda, MD, USA

Michael Sherman
Watertown, MA, USA

At the conclusion of this session, participants should be able to: 1. Describe the molecular components of the ubiquitin-proteasome system (UPS); 2. Explain the potential roles of molecular chaperones in mitigating the damage caused by misfolded proteins; 3. Discuss the relevance of the UPS in the molecular pathophysiology of Parkinson's disease.

S308 Botulinum toxin mechanisms and applications

Location: Meeting Room 8, Ground Floor

Reiner Benecke
Rostock, Germany

Dirk Dressler
Rostock, Germany

SCIENTIFIC PROGRAM

7:00 pm to 9:00 pm

Video Dinners

Video presentations of atypical Movement Disorders engage delegates and generate clinical discussions. To ensure greater interaction, Video Dinners are limited to a maximum number of participants, and a ticket is required for admission. As the title indicates, dinner is served during the sessions.

Fee: $80 USD/ $55 USD for junior participants and allied health professionals.

V201 Paroxysmal Movement Disorders

Location: Meeting Room 1, First Floor

Kailash Bhatia
London, United Kingdom

Kapil Sethi
Augusta, GA, USA

V202 Psychogenic Movement Disorders

Location: Meeting Room 2, First Floor

Anthony E. Lang
Toronto, Canada

John Morris
Sydney, Australia

At the conclusion of this session, participants should be able to: 1. Identify the key clinical features of psychogenic Movement Disorders; 2. Recognize the difference between psychogenic Movement Disorders and Movement Disorders associated with organic disease of the nervous system; 3. Define some of the underlying mechanisms of psychogenic Movement Disorders.

V203 Rare examples of Parkinsonism

Location: Meeting Room 3, First Floor

Andrew Lees
London, United Kingdom

Nir Giladi
Tel Aviv, Israel

At the conclusion of this session, participants should be able to: 1. Recognize some unusual and rare causes of Parkinson's syndrome; 2. Recognize some rare presentations of Parkinson's disease; 3. Receive factual information relating to the nature and diagnosis of the rare cases to be presented by video.

V204 Unusual Movement Disorders

Location: Meeting Room 4, First Floor
Rajesh Pahwa
Kansas City, KS, USA

Oscar Gershanik
Buenos Aires, Argentina

At the conclusion of this session, participants should be able to: 1. Indicate what are the necessary steps that have to be made for the systematic analysis of the phenomenology of a patient with an unusual Movement Disorder; 2. Identify the different types of abnormal involuntary movements that can be observed in the cases shown during the video session as the first necessary step towards the recognition of an unusual Movement Disorder. The presenters will ask the audience to carefully observe the peculiar features that distinguish one Movement Disorder from the other and subsequently reach a reasoned identification with the help of categorical descriptions. 3. Recognize a wide variety of diseases that can present with unusual Movement Disorders and frequently constitute a diagnostic challenge for the general neurologist.

SCIENTIFIC PROGRAM

THURSDAY, JUNE 17, 2004

8:30 am to 10:00 am

Seminar Series

Sessions featuring an Italian continental breakfast are featured on Thursday. Each session offers an expert's view on Movement Disorders through a variety of topics. To encourage discussion and interaction, Seminar Series have limited registration and a ticket is required for admission.

Fee: $55 USD/ $40 USD for junior participants and allied health professionals.

S401 Basic genetics in Movement Disorders

Location: Meeting Room 5, First Floor

John Hardy
Bethesda, MD, USA

Andrew Singleton
Bethesda, MD, USA

At the conclusion of this session, participants should be able to: 1. Describe the major types of molecular genetic studies aimed at identifying genes that cause Movement Disorders; 2. Identify families and populations of interest for molecular genetics studies; 3. Recognize the first steps a clinician should take to begin molecular genetic analysis of a family with an inherited Movement Disorder.

S402 Differential diagnosis and management of choreas

Location: Meeting Room 3, First Floor

Kathleen Shannon
Chicago, IL, USA

Francisco Cardoso
Belo Horizonte MG, Brazil

At the conclusion of this session, participants should be able to: 1. Recognize common and rare choreic syndromes; 2. Describe the appropriate diagnostic work-up for chorea depending on characteristics of disease presentation and history; 3. Discuss the appropriate pharmacological approaches to the treatment of choreic disease.

S403 Clinical management of dystonia

Location: Meeting Room 1, First Floor

Alberto Albanese
Milano, Italy

Marie Vidailhet
Paris, France

At the conclusion of this session, participants should be able to identify and choose the most appropriate treatment for dystonia, based on classification and on clinical features.

S404 Management of motor complications in Parkinson's disease

Location: Meeting Room 2, First Floor

Paul Krack
Grenoble, France

Ray Watts
Birmingham, AL, USA

At the conclusion of this session, participants should be able to: 1. Describe the clinical characteristics of motor fluctuations and dyskinesias and explain their pathophysiology; 2. List all available strategies to prevent motor complications in the first place and to treat motor complications, including available oral medications, drug infusion, and surgical treatment; 3. Identify the optimal strategy in a given patient.

S405 Management of psychiatric disturbances in Parkinson's disease

Location: Meeting Room 6, First Floor

E. Ch. Wolters
Amsterdam, Netherlands

Jorge Luis Juncos
Atlanta, GA, USA

At the conclusion of this session, participants should be able to: 1. Understand the pathophysiology of PD-psychosis; 2. Understand the phamacotherapeutic strategies in PD-psychosis; 3. Understand the pharmacotherapeutic choices in PD-psychosis.

S406 Mitochondrial functions in Movement Disorders: therapeutic implication

Location: Meeting Room 7, Ground Floor

Cliff Shults
San Diego, CA, USA

M. Flint Beal
New York, NY, USA

At the conclusion of this session, participants should be able to: 1. Explain the various neuroprotective properties of coenzyme Q10; 2. Describe the results of coenzyme Q10 for neuroprotection in animal studies; 3. Describe the results of clinical trials of coenzyme Q10 in neurodegererative diseases.

S407 Targeting the basal ganglia for functional surgery

Location: Meeting Room 4, First Floor

Philip Starr
San Francisco, CA, USA

Maria Rodriguez-Oroz
Pamplona, Spain

At the conclusion of this session, participants should be able to: 1. Describe MRI-based target localization for GPI and STN; 2. Recognize electrophysiologic characteristics of the GPI and STN in Parkinson's disease; 3. Recognize stimulation-induced adverse effects during intra-operative test stimulation through DBS leads.

S408 Tourette syndrome update
Location: Meeting Room 8, Ground Floor

David Lichter
Clarence, NY, USA

Michael Trimble
London, United Kingdom

At the conclusion of this session, participants should be able to: 1. Discuss the diagnostic criteria for Tourette syndrome (TS) and tools available for assessment of tics and comorbid neuropsychiatric conditions in TS, especially obsessive compulsive disorder (OCD) and attention deficit hyperactivity disorder (ADHD); 2. Describe current concepts of pathophysiology and neurobiology of TS, as derived from post-mortem, neurochemical, neuroimaging, neurophysiologic, genetic and clinical stdies in TS patients, as well as studies in experimental animals; 3. Discuss the pharmacological and non-pharmalogical options available for treatment of tics, OCD, ADHD and other comorbid disorders in TS patients.

10:00 am to 12:00 pm

Parallel Session 5: Dyskinesias in Parkinson's disease

Location: Salone Della Cultura, Ground Floor

Chair: Stanley Fahn
New York, NY, USA

Co-chair: Peter Jenner
London, United Kingdom

Introduction and primate model
Peter Jenner
London, United Kingdom

Rodent model of dyskinesia
Angela Cenci
Lund, Sweden

Pathophysiologic basis of dyskinesia
Jonathan Brotchie
Toronto, Canada

Molecular mechanisms
Thomas Chase
Bethesda, MD, USA

Graft-related dyskinesias
C. Warren Olanow
New York, NY, USA

Pathophysiology of graft-related dyskinesias
José Obeso
Pamplona, Spain

Conclusion
Stanley Fahn
New York, NY, USA

At the conclusion of this session, participants should be able to: 1. Have an understanding of the pathophysiology of levodopa-induced dyskinesias; 2. Recognize and understand the cause of graft-induced dyskinesias; 3. Better identify and treat dopa-induced dyskinesias.

Parallel Session 6: Pathophysiology of Movement Disorders

Location: Assembly Hall, Ground Floor

Chair: Alfredo Berardelli
Rome, Italy

Co-chair: John Rothwell
London, United Kingdom

Introduction: how does the experimental model for Movement Disorders fit with data in individual diseases?
Alfredo Berardelli
Rome, Italy

Basic mechanisms of basal ganglia plasticity
Paolo Calabresi
Rome, Italy

The role of sensory deficits in the pathology of dystonia
Giovanni Abbruzzese
Genova, Italy

Are we nearer to understanding the mechanisms of tremor?
Günther Deuschl
Kiel, Germany

The use and physiological mechanisms of alternative cues to treat patients with Movement Disorders
Robert Iansek
Cheltenham, Australia

Conclusion
John Rothwell
London, United Kingdom

At the conclusion of this session, participants should be able to: 1. Describe the contribution of basal ganglia and cortical plasticity to the presentation of clinical symptoms of patients with Movement Disorders; 2. To describe the possible mechanisms of tremor and the roles of different CNS regions in different types of tremor; 3. Discuss how and why it may be necessary to treat some of Movement Disorder symptoms with alternative cues for movement.

12:00 pm to 2:00 pm

Abstract Poster Sessions and Lunch

Poster Location: Poster Area, First Floor
Lunch Location: Rooftop Terrace and Various Locations

Abstract Numbers 1018-1338
Authors present odd numbers from 12:00 pm to 1:00 pm
Authors present even numbers from 1:00 pm to 2:00 pm

SCIENTIFIC PROGRAM

2:00 pm to 4:30 pm

Parallel Session 7: Controversies

Location: Salone Della Cultura, Ground Floor

Chair: Yves Agid
Paris, France

Co-chair: Donald Calne
Vancouver, Canada

Initial therapy in Parkinson's disease should be with a dopamine agonist: YES
Werner Poewe
Innsbruck, Austria

Initial therapy in Parkinson's disease should be with a dopamine agonist: NO
William Weiner
Baltimore, MD, USA

Imaging endpoints reflect Parkinson's disease progression: YES
David Brooks
London, United Kingdom

Imaging endpoints reflect Parkinson's disease progression: NO
J. Eric Ahlskog
Rochester, MN, USA

Immunology in Movement Disorders: PANDAS and Tourette's: YES
Gavin Giovannoni
London, United Kingdom

Immunology in Movement Disorders: PANDAS and Tourette's: NO
Harvey Singer
Baltimore, MD, USA

Do you need Lewy bodies to diagnose Parkinson's disease? YES
Dennis Dickson
Jacksonville, FL, USA

Do you need Lewy bodies to diagnose Parkinson's disease? NO
Yoshikuni Mizuno
Tokyo, Japan

Can you have Parkinson's disease with a normal F-dopa/PET or DAT/SPECT?: YES
Eldad Melamed
Petah Tiqva, Israel

Can you have Parkinson's disease with a normal F-dopa/PET or DAT/SPECT?: NO
Kenneth Marek
New Haven, CT, USA

At the conclusion of this session, participants should be able to: 1. Discuss PET is Parkinson's disease; 2. Explain what Lewy bodies signify in Parkinson's disease; 3. Discuss initial treatment of Parkinson's disease.

Parallel Session 8: Surgery

Location: Assembly Hall, Ground Floor

Chair: Alim Benabid
Grenoble, France

Co-chair: William Koller
New York, NY, USA

Ablative surgery for Parkinson's disease
Jerrold Vitek
Atlanta, GA, USA

Deep brain stimulation for Parkinson's disease
Jens Volkmann
Kiel, Germany

Issues in surgery for Parkinson's disease: a neurologist's point of view
Pierre Pollak
Grenoble, France

Surgery for dystonia and tremor
Marcelo Merello
Buenos Aires, Argentina

The future of ablative and deep brain stimulation surgery in Movement Disorders
Andres Lozano
Toronto, Canada

At the conclusion of this session, participants should be able to: 1. Recognize the indications and patient selections of surgical treatment of Movement Disorders; 2. Appreciate the clinical response that can be expected with DBS treatment of Parkinson's disease; 3. Understand the current knowledge regarding the mechanism of action of DBS of the subthalamus in Parkinson's disease.

THE VISION

Through innovative research, strategic partnerships, and an unsurpassed commitment to disease education, Pfizer Neuroscience is dedicated to being the leading provider of neurologic and psychiatric medicines that make a meaningful difference in the lives of patients and their families around the world.

NEUROSCIENCE

FACULTY

Key: KS = Kickoff Seminar, PS = Plenary Session, PRS = Parallel Session, S = Seminar, V = Video Dinner

Giovanni Abbruzzese
Genova, Italy
PRS06

Yves Agid
Paris, France
KS11, PRS07

J. Eric Ahlskog
Rochester, MN, USA
PRS07

Alberto Albanese
Milano, Italy
S403

Richard P. Allen
Arnold, MD, USA
KS05, S108

Paolo Barone
Napoli, Italy
KS02, S201

M. Flint Beal
New York, NY, USA
S406

Alim L. Benabid
Grenoble, France
PRS08

Reiner Benecke
Rostock, Germany
S308

Alfredo Berardelli
Roma, Italy
KS09, PRS06

Hagai Bergman
Jerusalem, Isreal
PS02

Erwan Bezard
Bordeaux, France
PS02

Kailash P. Bhatia
London, United Kingdom
S202, V201

Vincenzo Bonifati
Roma, Italy
S206

Heiko Braak
Frankfurt, Germany
S305

Susan B. Bressman
Englewood, NJ, USA
V102

David J. Brooks
London, United Kingdom
KS04, PRS07

Jonathan M. Brotchie
Toronto, Canada
PRS05

Peter Brown
London, United Kingdom
PS02

Patrik Brundin
Lund, Sweden
PS03

David John Burn
Newcastle Upon Tyne, United Kingdom
PS04, S204

Paolo Calabresi
Rome, Italy
PRS06

Donald B. Calne
Vancouver, Canada
PRS07

Francisco Cardoso
Belo Horizonte MG, Brazil
S302, S402

Elena Cattaneo
Milano, Italy
PRS04

Angela M. Cenci
Lund, Sweden
PRS05

Thomas N. Chase
Bethesda, MD, USA
PRS05

Ray Chaudhuri
London, United Kingdom
KS03

Robert Chen
Toronto, Canada
S205

Cynthia L. Comella
Chicago, IL, USA
KS09, PRS01

Mark Cookson
Bethesda, MD, USA
S307

Antonio Currà
Venafro, Italy
S205

Ted M. Dawson
Baltimore, MD, USA
S208

Gianni Defazio
Bari, Italy
S301

Günther Deuschl
Kiel, Germany
KS1B, PRS06

Dennis Dickson
Jacksonville, FL, USA
PRS07

Dirk W. Dressler
Rostock, Germany
S308

Bruno Dubois
Paris, France
KS07, PRS03

Roger J. Elble
Springfield, IL, USA
V103

Murat Emre
Capa Istanbul, Turkey
KS07, PRS02, PRS03

Giovanni Fabbrini
Rome, Italy
S105

Stanley Fahn
New York, NY, USA
PRS05, S207

Joaquim Ferreira
Torres Vedras, Portugal
KS05

Steven Frucht
New York, NY, USA
V104

Diego Garcia Borreguero
Madrid, Spain
KS03

Oscar S. Gershanik
Buenos Aires, Argentina
KS10, V204

Nir Giladi
Tel Aviv, Israel
V203

Gavin Giovannoni
London, United Kingdom
PRS07

Christopher G. Goetz
Chicago, IL, USA
KS02, S104, S207

Lawrence I. Golbe
New Brunswick, NJ, USA
S106

Mark Hallett
Bethesda, MD, USA
KS1B, PRS04

Glenda M. Halliday
Randwick, Australia
S305

John A. Hardy
Bethesda, MD, USA
PS01, S401

Robert Hauser
Tampa, FL, USA
KS11

Etienne C. Hirsch
Paris, France
KS10, PS01

Robert Iansek
Cheltenham, Australia
PRS06

Ole Isacson
Belmont, MA, USA
PS03

Joseph Jankovic
Houston, TX, USA
KS1B, KS09, PS04, V102

Peter Jenner
London, United Kingdom
KS10, PRS05

Mandar Jog
London, Canada
PRS03

Jorge Luis Juncos
Atlanta, GA, USA
S302, S405

Ryuji Kaji
Tokushima City, Japan
S203

Horacio Kaufman
New York, NY, USA
S103

Karl D. Kieburtz
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PS04

William C. Koller
New York, NY, USA
KS1A, PRS08

Amos Korczyn
Ramat-Aviv, Isreal
KS05

Jeffrey H. Kordower
Chicago, IL, USA
PS03

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Grenoble, France
S404

Anthony E. Lang
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J. William Langston
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PS01

Andrew J. Lees
London, United Kingdom
KS1A, KS04, PS04, V203

Richard Levy
Paris, France
PRS03

Peter A. LeWitt
Southfield, MI, USA
KS06, S202

David G. Lichter
Clarence, NY, USA
S408

Olle Lindvall
Lund, Sweden
PS03

Irene Litvan
Louisville, KY, USA
S304

Elan D. Louis
New York, NY, USA
PRS04

Andres M. Lozano
Toronto, Canada
PRS08

Christoph Lücking
Munich, Germany
S306

FACULTY

Key: KS = Kickoff Seminar, PS = Plenary Session, PRS = Parallel Session, S = Seminar, V = Video Dinner

Kenneth Marek
New Haven, CT, USA
KS10, PRS07

I.G. McKeith
Newcastle Upon Tyne, United Kingdom
KS07, PRS03

Kevin McNaught
New York, NY, USA
PS01

Hans Michael Meinck
Heidelberg, Germany
S101

Eldad Melamed
Petah Tiqva, Israel
KS11, PRS07

Marcelo Merello
Buenos Aires, Argentina
PRS08

Yoshikuni Mizuno
Tokyo, Japan
KS02, PRS07

Jacques Montplaisir
Montreal, Canada
KS03

John G.L. Morris
Sydney, Australia
V202

Markus Naumann
Wuerzburg, Germany
KS09

John G. Nutt
Portland, OR, USA
PS03, V103

José A. Obeso
Pamplona, Spain
KS10, PRS05, PS02

Per Odin
Bremerhave, Germany
KS03

Wolfgang H. Oertel
Marburg, Germany
KS04, PRS01

C. Warren Olanow
New York, NY, USA
KS11, PRS05, PS01

Adrian M. Owen
Cambridge, United Kingdom
PRS03

Rajesh Pahwa
Kansas City, KS, USA
V204

Walter Paulus
Gottingen, Germany
KS05

Daniel P. Perl
New York, NY, USA
S204

Joel S. Perlmutter
St. Louis, MO, USA
PS02

Ronald Pfeiffer
Memphis, TN, USA
S103

Werner Poewe
Innsbruck, Austria
KS08, PRS07

Pierre Pollak
Grenoble, France
PRS08

Peter Paul Pramstaller
Bolzano, Italy
S106

Donald L. Price
Baltimore, MD, USA
C. David Marsden Lecturer

Serge Przedborski
New York, NY, USA
PS01

Stefan Pulst
Los Angeles, CA, USA
S102

Niall P. Quinn
London, United Kingdom
KS06, V101

Olivier Rascol
Toulouse, France
KS08, PS04

Heinz Reichmann
Dresden, Germany
KS02

Peter Riederer
Wuerzburg, Germany
PRS02

Maria Rodriguez-Oroz
Pamplona, Spain
S407

Thomas Roth
Detroit, MI, USA
KS05

John C. Rothwell
London, United Kingdom
PRS06, PS02

David Rye
Atlanta, GA, USA
S201

Terence Sanger
Stanford, CA, USA
S107

Anthony H.V. Schapira
London, United Kingdom
KS10, PRS04

Anette Schrag
London, United Kingdom
S306

Kapil D. Sethi
Augusta, GA, USA
KS05, V201

Kathleen M. Shannon
Chicago, IL, USA
S402

Jie Shen
Boston, MA, USA
S208

Michael Y. Sherman
Watertown, MA, USA
S307

Hiroshi Shibasaki
Bethesda, MD, USA
V104

Ira Shoulson
Rochester, NY, USA
KS08, PRS04

Cliff Shults
San Diego, CA, USA
S406

Harvey S. Singer
Baltimore, MD, USA
PRS07

Andrew Singleton
Bethesda, MD, USA
S401

Mark Stacy
Durham, NC, USA
KS1A

Philip Starr
San Francisco, CA, USA
S407

Matthew Stern
Philladelphia, PA, USA
KS08

Fabrizio Stocchi
Rome, Italy
KS1A, KS11

A. Jon Stoessl
Vancouver, Canada
KS04, PS04

Peter L. Strick
Pittsburg, PA, USA
PRS03

S.H. Subramony
Jackson, MS, USA
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Robert A. H. Surtees
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Clive N. Svendsen
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PS03

Caroline M. Tanner
Sunnyvale, CA, USA
S206

Daniel Tarsy
Boston, MA, USA
S303

Philip D. Thompson
North Terrace, Adelaide, Australia
S101

Francois Tison
Pessac, France
S105

Eduardo Tolosa
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Claudia M. Trenkwalder
Kassel, Germany
KS03, S108

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Jerrold Lee Vitek
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PRS08

Jens Volkmann
Kiel, Germany
PRS08

Arthur S. Walters
Edison, NJ, USA
KS03

Thomas T. Warner
London, United Kingdom
S301

Cheryl H. Waters
New York, NY, USA
KS06, S104

Ray L. Watts
Birmingham, AL, USA
KS07, S404

William J. Weiner
Baltimore, MD, USA
KS06, PRS07, S303

Gregor K. Wenning
Innsbruck, Austria
S304

E. Ch. Wolters
Amsterdam, Netherlands
S405

Nobuo Yanagisawa
Kawasaki-city, Japan
PS02

Anne B. Young
Boston, MA, USA
PRS04

Huda Zoghbi
Houston, TX, USA
Stanley Fahn Lecturer

European Federation of Neurological Societies

Co-Sponsored by the European Section of the Movement Disorder Society (MDS – ES)

9th CONGRESS OF THE EUROPEAN FEDERATION OF NEUROLOGICAL SOCIETIES

Preliminary Scientific Programme

Athens, Greece, September 17-20, 2005

Main Topics

- Vascular cognitive impairment
- ALS
- Neuroprotection - neurodegeneration in MS
- The mysteries of Parkinsonism – New insights
- Headache - advances in pathophysiology and management
- Neurological disorders and sleep apnea
- Update on carotid artery disease
- Eye movements – A window to brain function
- Burden and costs of neurological diseases

Teaching Courses

- Movement disorders
- Stroke
- Epilepsy
- Dementia
- Treatment strategies in multiple sclerosis
- From headache syndromes to headache management
- From diagnosis to treatment in neuromuscular diseases
- Autonomic nervous system
- Critical care
- Neurooncology
- Neurootology - vertigo

EFNS 2005

www.efns.org/efns2005

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Fax: +43 1 889 05 03 12
E-mail: headoffice@efns.org

COMMITTEE & TASK FORCE MEETINGS

MONDAY, JUNE 14

7:00 am to 8:30 am

Awards Committee
Location: Meeting Room 5, First Floor

Education Committee
Location: Meeting Room 3, First Floor

Financial Affairs Committee
Location: Meeting Room 6, First Floor

Journal Oversight Committee
Location: Meeting Room 4, First Floor

12:30 pm to 1:30 pm

International Congress Oversight Committee
Location: Officers/IEC Workroom, Ground Floor

4:30 pm to 7:30 pm

Task Force on PD Dementia
Location: Officers/IEC Workroom, Ground Floor

6:00 pm to 7:30 pm

Membership Committee
Location: Lounge 1, Ground Floor

TUESDAY, JUNE 15, 2004

7:00 am to 8:30 am

Continuing Medical Education (CME) Committee
Location: Meeting Room 3, First Floor

Industrial Relations Committee
Location: Meeting Room 4, First Floor

Liaison/Public Relations Committee
Location: Meeting Room 6, First Floor

WEDNESDAY, JUNE 16

7:00 am to 8:30 am

Bylaws Committee
Location: Meeting Room 6, First Floor

Neurosurgery Section Task Force
Location: Meeting Room 2, First Floor

UPDRS Revision Task Force Steering Committee / Task Force for the Development of Rating Scales for PD
Location: Meeting Room 5, First Floor

7:30 am to 8:30 am

Scientific Issues Committee
Location: Meeting Room 4, First Floor

12:00 pm to 1:30 pm

EBMR Task Force
Location: Meeting Room 6, First Floor

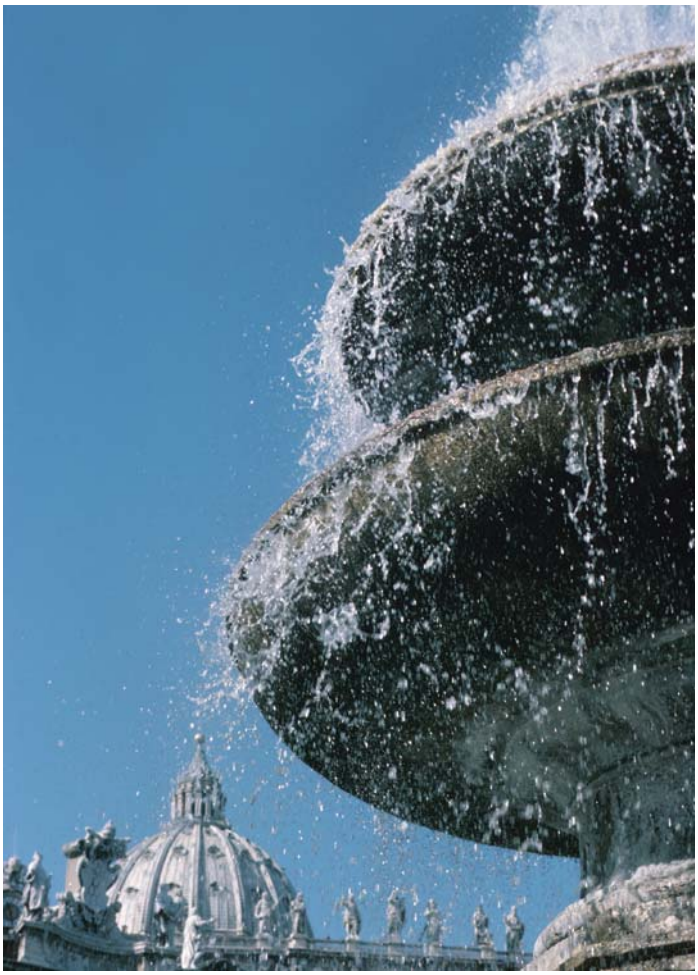
Task Force on Epidemiology
Location: Meeting Room 5, First Floor

Young Members
Location: Meeting Room 1, First Floor

MDS ANNUAL BUSINESS MEETING

Tuesday, June 15
10:30 am to 11:30 am

Salone Della Cultura, Ground Floor



EXHIBITION

General information and Exhibit Hall Hours

Please allow adequate time in your daily schedule to visit the Exhibit Hall, located in the Palazzo dei Congressi. The exhibition is an integral component of your International Congress experience, offering you the opportunity to speak with representatives of companies that provide services or market products directly related to Movement Disorders. Delegates may enter the Exhibit Hall during the following hours:

Monday, June 14	8:00 am to 5:00 pm
Tuesday, June 15	8:00 am to 5:00 pm
Wednesday, June 16	8:00 am to 5:00 pm
Thursday, June 17	8:00 am to 5:00 pm

Exhibitor Registration

Exhibitors may register at the Exhibitor Registration Desk, located in the Palazzo dei Congressi during the following hours:

Saturday, June 12	3:00 pm to 8:30 pm
Sunday, June 13	6:30 am to 6:00 pm
Monday, June 14	6:30 am to 6:00 pm
Tuesday, June 15	6:30 am to 6:00 pm
Wednesday, June 16	6:30 am to 6:00 pm
Thursday, June 17	6:30 am to 6:00 pm

Exhibitor Badge Policy

Exhibit booth personnel must show an official MDS exhibitor name badge in order to gain access to the Exhibit Hall during installation, show, or dismantlement hours. Badges should be worn at all times as security guards will monitor Exhibit Hall entrances for proper identification. Exhibitors will be identified as follows:

Exhibitor Badge (Yellow) - Allows admittance to the exhibit hall area only.

Exhibitor Delegate Badge (Orange) - Allows the delegate to enter the exhibit hall as an exhibitor and attend scientific sessions including poster presentations (access to Wine and Cheese Seminars and Video Dinners at an additional cost).

Endorsement Disclaimer

Products and services displayed in the Exhibit Hall or advertised in the program occur by contractual business arrangements between the MDS and participating companies and organizations. These arrangements do not constitute nor imply an endorsement by the MDS of these products and services.

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Fax: +44 1494 473593
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Booth Number: 117

Allergan is the manufacturer of BOTOX®, Botulinum Toxin Type-A, Purified Neurotoxin Complex. There are presently a number of BOTOX® clinical trials underway for a wide variety of uses. Allergan, Inc. headquartered in Irvine, California, is a technology-driven, global health care company providing eye care and specialty pharmaceutical products worldwide.

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Fax: +44 1494 798700
Web site: www.amershamhealth.com
Booth Number: 214

Bertek Pharmaceuticals, Inc.

Marketing
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USA
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Fax: +1 (919) 993-5907
Web site: www.bertek.com
Booth Number: 123

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Web site: www.boehringer-ingelheim.com
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United Kingdom
Phone: +44 191 296 9307
Fax: +44 191 296 9368
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Dystonia Medical Research Foundation
One East Wacker Dr. #2430
Chicago, IL 60601
USA
Phone: +1 (312) 755-0198
Fax: +1 (312) 803-0138
Web site: www.dystonia-foundation.org
Booth Number: 113
The Dystonia Medical Research Foundation is mandated to advance research for more effective treatments and ultimately a cure; to promote awareness and education; and to support the needs and well-being of affected individuals and families.

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European Dystonia Federation
69 East King Street
Helensburgh, G84 7RE
United Kingdom
Phone: +44 1436 678799
Fax: +44 1436 678799
Web site: www.dystonia-europe.org
Booth Number: 212
Alliance of 19 national dystonia patient support organizations in Europe.

European Federation of Neurological Societies
Alser Strasse 4
Vienna, 10900
Austria
Phone: +43 1 889 0503
Fax: +43 1 889 050313
Web site: www.efns.org
Booth Number: 235
The aim of the European Federation of Neurological Societies is to advance the development of the neurological sciences in Europe. 38 European national neurological associations are registered members of the EFNS. The EFNS welcomes individual members from all over the world. For more information visit www.efns.org.

FHC, Inc.
9 Main St.
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USA
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Web site: www.fh-co.com
Booth Number: 210
FHC's microTargeting® products are used for intraoperative micro/macro-electrode recording, micro/macro-stimulation, and data analysis in functional neurosurgery to treat Movement Disorders. These products include FDA cleared and CE marked microelectrodes, microdrive systems and the microTargeting® Platform that mounts on implanted fiducial markers and eliminates the need for a sterotactic frame.

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Teningen D-79331
Germany
Phone: +49 7641 9414 60
Fax: +49 7641 9414 94
Web site: www.inomed.com
Booth Number: 232
inomed GmbH maufactures equipment for intraoperative neurophysiological monitoring, neurological diagnostics and invasive pain therapy. Products include ISIS IOM and ISIS MER System for intraoperative neurophysiological monitoring and Micro Electrode recording.

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Medtronic Neurological
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USA
Phone: +1 (763) 505-5000
Fax: +1 (763) 505-1000
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National Spasmodic Torticollis Association
9920 Halbert Ave.
Fountain Valley, CA 92708
USA
Phone: +1 (714) 378-7837
Fax: +1 (714) 378-7830
Web site: www.torticollis.org
Booth Number: 110
The National Spasmodic Torticollis Association is a non-profit organization dedicated to: providing information and support to ST patients, educating the public and the medical community about ST, advocating for the rights of those with ST and promoting research on ST.

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Fax: +41 61 324 6652
Web site: www.novartis.com

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FIN-02200 Espoo
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Fax: +1 (212) 808-8833
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Rochester, MN 55902
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Phone: +1 (507) 287-6465
Fax: +1 (507) 287-6312
Web site: www.rls.org
Booth Number: 247
The Restless Legs Syndrome Foundation is a non-profit organization dedicated to increasing universal awareness, developing effective treatments, and finding a definitive cure for restless legs syndrome (RLS). The organization provides information about RLS, develops local support groups, publishes a quarterly newsletter, and funds research for the study of RLS.

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16th International Congress on Parkinson's Disease and Related Disorders
Paulsbornerstr. 44
Berlin, 14193
Germany
Phone: +49 30 300 6690
Web site: www.parkinson-berlin.de
Booth Number: 229
The 16th International Congress on Parkinson's Disease and related Disorders will be held in Berlin from 5th to 9th of June, 2005. The theme "Present and Future Perspectives of Parkinson's Syndrome" lends a certain futurological perspective to this Congress, but also includes the retrospective viewpoint. We will discuss the possibilities and limitations of the classification, etiopathogenesis and therapy of Parkinson's disease in the present and in the future.

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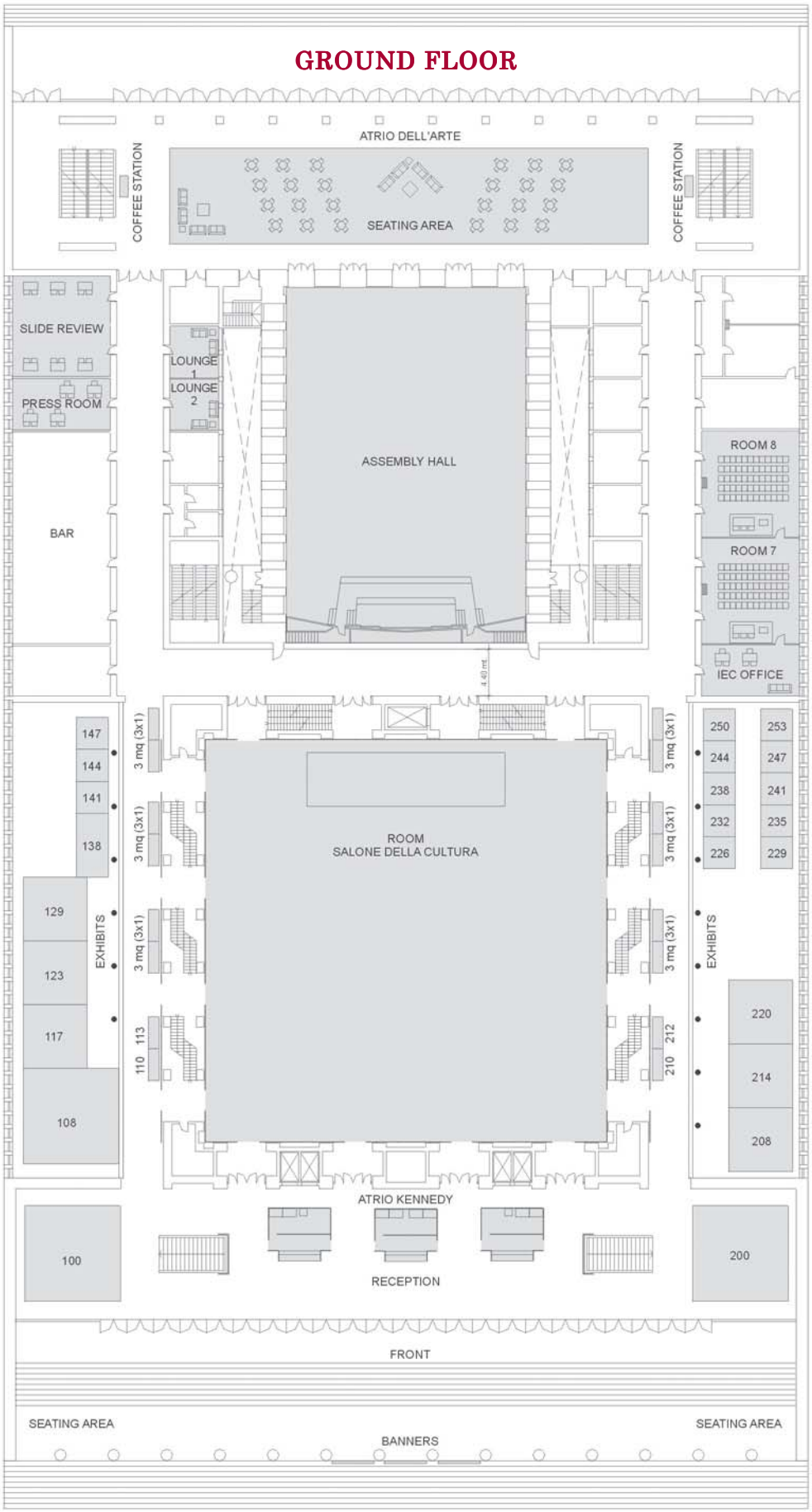

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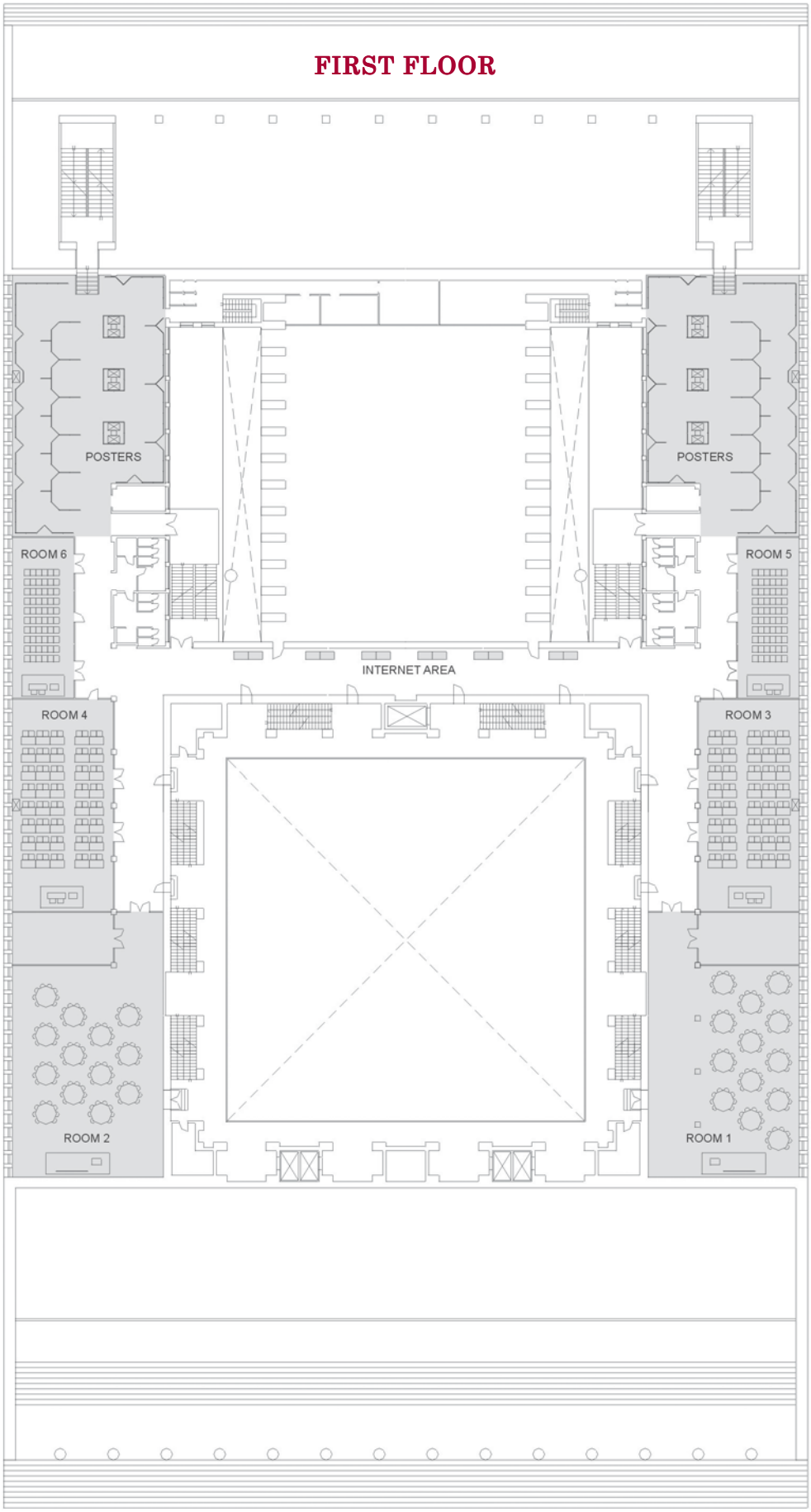

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12.5/50/200 mg, 25/100/200 mg, 37.5/150/200 mg

Enhance the Benefits of Levodopa

STALEVO tablets are indicated to treat patients with idiopathic Parkinson's disease: 1. To substitute (with equivalent strength of each of the 3 components) for immediate-release carbidopa/levodopa and entacapone previously administered as individual products. 2. To replace immediate-release carbidopa/levodopa therapy (without entacapone) when patients experience the signs and symptoms of end-of-dose "wearing off" (only for patients taking a total daily dose of levodopa of 600 mg or less and not experiencing dyskinesia). STALEVO is contraindicated for use concomitantly with nonselective monoamine oxidase (MAO) inhibitors, with selegiline at doses >10 mg/day, in patients with narrow-angle glaucoma, and in patients with suspicious, undiagnosed skin lesions or a history of melanoma. Because STALEVO contains entacapone, it should not be used concurrently with COMTAN® (entacapone). The most common side effects of STALEVO therapy are dopaminergic in nature (eg, dyskinesia, nausea). These side effects may be manageable with alteration in the drug-dosing schedule, ie, extending the dosing interval, reducing the number of doses per day, or changing to a STALEVO strength containing less levodopa. However, rapid withdrawal or abrupt reduction of STALEVO therapy should be avoided. Other common side effects include diarrhea, hyperkinesia, urine discoloration, hypokinesia, abdominal pain, dizziness, constipation, fatigue, pain, and hallucinations. Other less frequent side effects can include other mental disturbances, orthostatic hypotension, rhabdomyolysis, severe diarrhea, dark saliva, and symptoms resembling neuroleptic malignant syndrome. Drugs metabolized by the COMT enzymes (eg, isoproterenol, epinephrine) should be used with caution in patients receiving STALEVO. STALEVO should be used with caution in patients with severe cardiovascular or pulmonary disease, bronchial asthma, renal, hepatic, or endocrine disease, and in patients with a history of myocardial infarction or peptic ulcer.

STALEVO provides dosing convenience in a single tablet[1]

Three dosage strengths—each with a 1:4 ratio of carbidopa to levodopa

	Carbidopa	Levodopa	Entacapone
 STALEVO 50	12.5 mg	50 mg	200 mg
 STALEVO 100	25.0 mg	100 mg	200 mg
 STALEVO 150	37.5 mg	150 mg	200 mg

actual size

- Individual tablets should not be fractionated
- Only 1 STALEVO tablet should be administered at each dosing interval
- Except for COMTAN® (entacapone), standard drugs for PD may be used concomitantly with STALEVO (dose adjustments for those drugs may be required)

Stalevo™
(carbidopa, levodopa and entacapone) tablets
12.5/50/200 mg, 25/100/200 mg, 37.5/150/200 mg

Enhance the Benefits of Levodopa

References: 1. STALEVO prescribing information. East Hanover, NJ: Novartis Pharmaceuticals Corp; June 2003. **2.** Larsen JP, Wirn-Petersen J, Sidén Å, et al. The sustainability and efficacy of entacapone over 3 years in patients with Parkinson's disease. *Eur J Neurol.* 2002;9:137-144.

1/04

C-STA-1008

Stalevo™ 50
Stalevo™ 100
Stalevo™ 150

(carbidopa, levodopa and entacapone)
Tablets

Rx only

BRIEF SUMMARY: Please see package insert for full prescribing information.

INDICATIONS: Stalevo™ (carbidopa, levodopa and entacapone) is indicated to treat patients with idiopathic Parkinson's disease: 1. To substitute (with equivalent strength of each of the three components) for immediate release carbidopa/levodopa and entacapone previously administered as individual products. 2. To replace immediate-release carbidopa/levodopa therapy (without entacapone) when patients experience the signs and symptoms of end-of-dose "wearing-off" (only for patients taking a total daily dose of levodopa of 600 mg or less and not experiencing dyskinesias, *see DOSAGE AND ADMINISTRATION in the full prescribing information*). **CONTRAINDICATIONS:** Stalevo™ (carbidopa, levodopa and entacapone) tablets are contraindicated in patients who have demonstrated hypersensitivity to any component (carbidopa, levodopa, or entacapone) of the drug or its excipients. Monoamine oxidase (MAO) and COMT are the two major enzyme systems involved in the metabolism of catecholamines. It is theoretically possible, therefore, that the combination of entacapone and a non-selective MAO inhibitor (e.g., phenelzine and tranylcypromine) would result in inhibition of the majority of the pathways responsible for normal catecholamine metabolism. As with carbidopa-levodopa, nonselective monoamine oxidase (MAO) inhibitors are contraindicated for use with Stalevo. These inhibitors must be discontinued at least two weeks prior to initiating therapy with Stalevo. Stalevo may be administered concomitantly with the manufacturer's recommended dose of MAO inhibitors with selectivity for MAO type B (e.g., selegiline HCl). *(See PRECAUTIONS, Drug Interactions.)* Stalevo is contraindicated in patients with narrow-angle glaucoma. Because levodopa may activate malignant melanoma, Stalevo should not be used in patients with suspicious, undiagnosed skin lesions or a history of melanoma. **WARNINGS:** The addition of carbidopa to levodopa reduces the peripheral effects (nausea, vomiting) due to decarboxylation of levodopa; however, carbidopa does not decrease the adverse reactions due to the central effects of levodopa. Because carbidopa as well as entacapone permits more levodopa to reach the brain and more dopamine to be formed, certain adverse CNS effects, e.g., dyskinesia (involuntary movements) may occur at lower dosages and sooner with levodopa preparations containing carbidopa and entacapone than with levodopa alone. The occurrence of dyskinesias may require dosage reduction *(see PRECAUTIONS, Dyskinesia)*. Stalevo™ (carbidopa, levodopa and entacapone) may cause mental disturbances. These reactions are thought to be due to increased brain dopamine following administration of levodopa. All patients should be observed carefully for the development of depression with concomitant suicidal tendencies. Patients with past or current psychoses should be treated with caution. Stalevo should be administered cautiously to patients with severe cardiovascular or pulmonary disease, bronchial asthma, renal, hepatic or endocrine disease. As with levodopa, care should be exercised in administering Stalevo to patients with a history of myocardial infarction who have residual atrial, nodal, or ventricular arrhythmias. In such patients, cardiac function should be monitored carefully during the period of initial dosage adjustment, in a facility with provisions for intensive cardiac care. As with levodopa, treatment with Stalevo may increase the possibility of upper gastrointestinal hemorrhage in patients with a history of peptic ulcer. **Neuroleptic Malignant Syndrome (NMS):** Sporadic cases of a symptom complex resembling NMS have been reported in association with dose reductions or withdrawal of therapy with carbidopa-levodopa. Therefore, patients should be observed carefully when the dosage of Stalevo is reduced abruptly or discontinued, especially if the patient is receiving neuroleptics. NMS is an uncommon but life-threatening syndrome characterized by fever or hyperthermia, Neurological findings, including muscle rigidity, involuntary movements, altered consciousness, mental status changes; other disturbances, such as autonomic dysfunction, tachycardia, tachypnea, sweating, hypo- or hypertension; laboratory findings, such as creatine phosphokinase elevation, leukocytosis, myoglobinuria, and increased serum myoglobin have been reported. The early diagnosis of this condition is important for the appropriate management of these patients. Considering NMS as a possible diagnosis and ruling out other acute illnesses (e.g., pneumonia, systemic infection, etc.) is essential. This may be especially complex if the clinical presentation includes both serious medical illness and untreated or inadequately treated extrapyramidal signs and symptoms (EPS). Other important considerations in the differential diagnosis include central anticholinergic toxicity, heat stroke, drug fever and primary central nervous system (CNS) pathology. The management of NMS should include: 1) intensive symptomatic treatment and medical monitoring and 2) treatment of any concomitant serious medical problems for which specific treatments are available. Dopamine agonists, such as bromocriptine, and muscle relaxants, such as dantrolene, are often used in the treatment of NMS, however, their effectiveness has not been demonstrated in controlled studies. **Drugs Metabolized by Catechol-O-Methyltransferase (COMT):** When a single 400 mg dose of entacapone was given together with intravenous isoproterenol (isoprenaline) and epinephrine without coadministered levodopa/dopa decarboxylase inhibitor, the overall mean maximal changes in heart rate during infusion were about 50% and 80% higher than with placebo, for isoproterenol and epinephrine, respectively. Therefore, drugs known to be metabolized by COMT, such as isoproterenol, epinephrine, norepinephrine, dopamine, dobutamine, alpha-methyldopa, apomorphine, isoetherine, and tolcapone should be administered with caution in patients receiving entacapone regardless of the route of administration (including inhalation), as their interaction may result in increased heart rates, possibly arrhythmias, and excessive changes in blood pressure. Ventricular tachycardia was noted in one 32-year-old healthy male volunteer in an interaction study after epinephrine infusion and oral entacapone administration. Treatment with propranolol was required. A causal relationship to entacapone administration appears probable but cannot be attributed with certainty. **PRECAUTIONS: General:** As with levodopa, periodic evaluations of hepatic, hematopoietic, cardiovascular, and renal function are recommended during extended therapy. Patients with chronic wide-angle glaucoma may be treated cautiously with Stalevo™ (carbidopa, levodopa and entacapone) provided the intraocular pressure is well controlled and the patient is monitored carefully for changes in intraocular pressure during therapy. **Hypotension/Syncope:** In the large controlled trials of entacapone, approximately 1.2% and 0.8% of 200 mg entacapone and placebo patients treated also with levodopa/dopa decarboxylase inhibitor, respectively, reported at least one episode of syncope. Reports of syncope were generally more frequent in patients in both treatment groups who had an episode of documented hypotension (although the episodes of syncope, obtained by history, were themselves not documented with vital sign measurement). **Diarrhea:** In clinical trials of entacapone, diarrhea developed in 69 of 903 (10.0%) and 16 of 400 (4.0%) of patients treated with 200 mg of entacapone or placebo in combination with levodopa/dopa decarboxylase inhibitor, respectively. In patients treated with entacapone, diarrhea was generally mild to moderate in severity (8.6%) but was regarded as severe in 1.3%. Diarrhea resulted in withdrawal in 10 of 403 (1.7%) patients, 7 (1.2%) with mild and moderate diarrhea and 3 (0.5%) with severe diarrhea. Diarrhea generally resolved after discontinuation of entacapone. Two patients with diarrhea were hospitalized. Typically, diarrhea presents within 4-12 weeks after entacapone is started, but it may appear as early as the first week and as late as many months after the initiation of treatment. **Hallucinations:** Dopaminergic therapy in Parkinson's disease patients has been associated with hallucinations. In clinical trials of entacapone, hallucinations developed in approximately 4.0% of patients treated with 200 mg entacapone or placebo in combination with levodopa/dopa decarboxylase inhibitor. Hallucinations led to drug discontinuation and premature withdrawal from clinical trials in 0.8% and 0% of patients treated with 200 mg entacapone and placebo, respectively. Hallucinations led to hospitalization in 1.0% and 0.3% of patients in the 200 mg entacapone and placebo groups, respectively. **Dyskinesia:** Entacapone may potentiate the dopaminergic side effects of levodopa and may therefore cause and/or exacerbate preexisting dyskinesia. Although decreasing the dose of levodopa may ameliorate this side effect, many patients in controlled trials continued to experience frequent dyskinesias despite a reduction in their dose of levodopa. The rates of withdrawal for dyskinesia were 1.5% and 0.8% for 200 mg entacapone and placebo, respectively. **Other Events Reported with Dopaminergic Therapy:** The events listed below are rare events known to be associated with the use of drugs that increase dopaminergic activity, although they are most often associated with the use of direct dopamine agonists. **Rhabdomyolysis:** Cases of severe rhabdomyolysis have been reported with entacapone when used in combination with levodopa. The complicated nature of these cases makes it impossible to determine what role, if any, entacapone played in their pathogenesis. Severe prolonged motor activity including dyskinesia may account for rhabdomyolysis. One case, however, included fever and alteration of consciousness. It is therefore possible that the rhabdomyolysis may be a result of the syndrome described in Hyperpyrexia and Confusion *(see PRECAUTIONS, Other Events Reported with Dopaminergic Therapy)*. **Hyperpyrexia and Confusion:** Cases of a symptom complex resembling the neuroleptic malignant syndrome characterized by elevated temperature, muscular rigidity, altered consciousness, and elevated CPK have been reported in association with the rapid dose reduction or withdrawal of other dopaminergic drugs. No cases have been reported following the abrupt withdrawal or dose reduction of entacapone treatment during clinical studies. Prescribers should exercise caution when discontinuing carbidopa, levodopa and entacapone combination treatment. When considered necessary, withdrawal should proceed slowly. If a decision is made to discontinue treatment with Stalevo, recommendations include monitoring the patient closely and adjusting other dopaminergic treatments as needed. This syndrome should be considered in the differential diagnosis for any patient who develops a high fever or severe rigidity. Tapering entacapone has not been systematically evaluated. **Fibrotic Complications:** Cases of retroperitoneal fibrosis, pulmonary infiltrates, pleural effusion, and pleural thickening have been reported in some patients treated with ergot derived dopaminergic agents. These complications may resolve when the drug is discontinued, but complete resolution does not always occur. Although these adverse events are believed to be related to the ergoline structure of these compounds, whether other, nonergot derived drugs (e.g., entacapone, levodopa) that increase dopaminergic activity can cause them is unknown. It should be noted that the expected incidence of

fibrotic complications is so low that even if entacapone caused these complications at rates similar to those attributable to other dopaminergic therapies, it is unlikely that it would have been detected in a cohort of the size exposed to entacapone. Four cases of pulmonary fibrosis were reported during clinical development of entacapone; three of these patients were also treated with pergolide and one with bromocriptine. The duration of treatment with entacapone ranged from 7-17 months.

Renal Toxicity: In a one-year toxicity study, entacapone (plasma exposure 20 times that in humans receiving the maximum recommended daily dose of 1600 mg) caused an increased incidence of nephrotoxicity in male rats that was characterized by regenerative tubules, thickening of basement membranes, infiltration of mononuclear cells and tubular protein casts. These effects were not associated with changes in clinical chemistry parameters, and there is no established method for monitoring for the possible occurrence of these lesions in humans. Although this toxicity could represent a species-specific effect, there is not yet evidence that this is so.

Hepatic Impairment: Patients with hepatic impairment should be treated with caution. The AUC and C_{max} of entacapone approximately doubled in patients with documented liver disease compared to controls. *(See CLINICAL PHARMACOLOGY, Pharmacokinetics, and DOSAGE AND ADMINISTRATION in the full prescribing information.)*

Biliary Obstruction: Caution should be exercised when administering Stalevo to patients with biliary obstruction, as entacapone is excreted mostly via the bile.

Information for Patients: The patient should be instructed to take Stalevo only as prescribed. The patient should be informed that Stalevo is a standard-release formulation of carbidopa-levodopa combined with entacapone that is designed to begin release of ingredients within 30 minutes after ingestion. It is important that Stalevo be taken at regular intervals according to the schedule outlined by the physician. The patient should be cautioned not to change the prescribed dosage regimen and not to add any additional anti-parkinsonian medications, including other carbidopa-levodopa preparations, without first consulting the physician. Patients should be advised that sometimes a "wearing-off" effect may occur at the end of the dosing interval. The physician should be notified for possible treatment adjustments if such response poses a problem to patient's every day life. Patients should be advised that occasionally, dark color (red, brown, or black) may appear in saliva, urine, or sweat after ingestion of Stalevo. Although the color appears to be clinically insignificant, garments may become discolored. The patient should be advised that a change in diet to foods that are high in protein may delay the absorption of levodopa and may reduce the amount taken up in the circulation. Excessive acidity also delays stomach emptying, thus delaying the absorption of levodopa. Iron salts (such as in multi-vitamin tablets) may also reduce the amount of levodopa available to the body. The above factors may reduce the clinical effectiveness of the levodopa, carbidopa-levodopa and Stalevo therapy. NOTE: The suggested advice to patients being treated with Stalevo is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects. Patients should be informed that hallucinations can occur. Patients should be advised that they may develop postural (orthostatic) hypotension with or without symptoms such as dizziness, nausea, syncope, and sweating. Hypotension may occur more frequently during initial therapy or when total daily levodopa dosage is increased. Accordingly, patients should be cautioned against rising rapidly after sitting or lying down, especially if they have been doing so for prolonged periods, and especially at the initiation of treatment with Stalevo. Patients should be advised that they should neither drive a car nor operate other complex machinery until they have gained sufficient experience on Stalevo to gauge whether or not it affects their mental and/or motor performance adversely. Because of the possible additive sedative effects, caution should be used when patients are taking other CNS depressants in combination with Stalevo. Patients should be informed that nausea may occur, especially at the initiation of treatment with Stalevo. Patients should be advised of the possibility of an increase in dyskinesia. Carbidopa-levodopa combination and entacapone are known to affect embryo-fetal development in the rabbit and in the rat, respectively. Accordingly, patients should be advised to notify their physicians if they become pregnant or intend to become pregnant during therapy *(see PREGNANCY, Pregnancy)*. Carbidopa and entacapone are known to be excreted into maternal milk in rats. Because of the possibility that carbidopa, levodopa and entacapone may be excreted into human maternal milk, patients should be advised to notify their physicians if they intend to breast-feed or are breast-feeding an infant.

Laboratory Tests: Abnormalities in laboratory tests may include elevations of liver function tests such as alkaline phosphatase, SGOT (AST), SGPT (ALT), lactic dehydrogenase, and bilirubin. Abnormalities in blood urea nitrogen and positive Coombs' test have also been reported. Commonly levels of blood urea nitrogen, creatinine, and uric acid are lower during administration of Stalevo than with levodopa. Stalevo may cause a false-positive reaction for urinary ketone bodies when a test tape is used for determination of ketonuria. This reaction will not be altered by boiling the urine specimen. False-negative tests may result with the use of glucose-oxidase methods of testing for glucosuria. Cases of falsely diagnosed pheochromocytoma in patients on carbidopa-levodopa therapy have been reported very rarely. Caution should be exercised when interpreting the plasma and urine levels of catecholamines and their metabolites in patients on carbidopa-levodopa therapy. Entacapone is a chelator of iron. The impact of entacapone on the body's iron stores is unknown; however, a tendency towards decreasing serum iron concentrations was noted in clinical trials. In a controlled clinical study serum ferritin levels (as marker of iron deficiency and subclinical anemia) were not changed with entacapone compared to placebo after one year of treatment and there was no difference in rates of anemia or decreased hemoglobin levels.

Drug Interactions: Caution should be exercised when the following drugs are administered concomitantly with Stalevo.

Anti-hypertensive agents: Symptomatic postural hypotension has occurred when carbidopa-levodopa was added to the treatment of a patient receiving anti-hypertensive drugs. Therefore, when therapy with Stalevo is started, dosage adjustment of the antihypertensive drug may be required.

MAO inhibitors: For patients receiving nonselective MAO inhibitors, *see CONTRAINDICATIONS*. Concomitant therapy with selegiline and carbidopa-levodopa may be associated with severe orthostatic hypotension not attributable to carbidopa-levodopa alone.

Tricyclic antidepressants: There have been rare reports of adverse reactions, including hypertension and dyskinesia, resulting from the concomitant use of tricyclic antidepressants and carbidopa-levodopa.

Dopamine D2 receptor antagonists (e.g., phenothiazines, butyrophenones, risperidone) and isoniazid: Dopamine D2 receptor antagonists (e.g., phenothiazines, butyrophenones, risperidone) and isoniazid may reduce the therapeutic effects of levodopa.

Phenytoin and papaverine: The beneficial effects of levodopa in Parkinson's disease have been reported to be reversed by phenytoin and papaverine. Patients taking these drugs with carbidopa-levodopa should be carefully observed for loss of therapeutic response.

Iron salts: Iron salts may reduce the bioavailability of levodopa, carbidopa and entacapone. The clinical relevance is unclear.

Metoclopramide: Although metoclopramide may increase the bioavailability of levodopa by increasing gastric emptying, metoclopramide may also adversely affect disease control by its dopamine receptor antagonistic properties.

Drugs known to interfere with biliary excretion, glucuronidation, and intestinal beta-glucuronidase (probenecid, cholestyramine, erythromycin, rifampicin, ampicillin and chloramphenicol): As most entacapone excretion is via the bile, caution should be exercised when drugs known to interfere with biliary excretion, glucuronidation, and intestinal beta-glucuronidase are given concurrently with entacapone. These include probenecid, cholestyramine, and some antibiotics (e.g., erythromycin, rifampicin, ampicillin and chloramphenicol).

Pyridoxine: Stalevo can be given to patients receiving supplemental pyridoxine. Oral coadministration of 10-25 mg of pyridoxine hydrochloride (vitamin B6) with levodopa may reverse the effects of levodopa by increasing the rate of aromatic amino acid decarboxylation. Carbidopa inhibits this action of pyridoxine; therefore, Stalevo can be given to patients receiving supplemental pyridoxine.

Effect of levodopa and carbidopa in Stalevo on the metabolism of other drugs: Inhibition or induction effect of levodopa and carbidopa has not been investigated.

Effect of entacapone in Stalevo on the metabolism of other drugs: Entacapone is unlikely to inhibit the metabolism of other drugs that are metabolized by major P450s including CYP1A2, CYP2A6, CYP2C8, CYP2C19, CYP2D6, CYP2E1 and CYP3A. *In vitro* studies of human CYP enzymes showed that entacapone inhibited the CYP enzymes 1A2, 2A6, 2C8, 2C19, 2D6, 2E1 and 3A only at very high concentrations (IC50 from 200 to over 1000 µM; an oral 200 mg dose achieves a highest level of approximately 5 µM in people); these enzymes would therefore not be expected to be inhibited in clinical use. However, no information is available regarding the induction effect from entacapone.

Drugs that are highly protein bound (such as warfarin, salicylic acid, phenylbutazone, and diazepam): ***Levodopa:*** Levodopa is bound to plasma protein only to a minor extent (about 10%-30%).

Carbidopa: Carbidopa is approximately 36% bound to plasma protein.

Entacapone: Entacapone is highly protein bound (98%). *In vitro* studies have shown no binding displacement between entacapone and other highly bound drugs, such as warfarin, salicylic acid, phenylbutazone, and diazepam.

Hormone Levels: Of the ingredients in Stalevo, levocopa is known to depress prolactin secretion and increase growth hormone levels.

Carcinogenesis: In a two-year bioassay of carbidopa-levodopa, no evidence of carcinogenicity was found in rats receiving doses of approximately two times the maximum daily human dose of carbidopa and four times the maximum daily human dose of levodopa. Two-year carcinogenicity studies of entacapone were conducted in mice and rats. Rats were treated once daily by oral gavage with entacapone doses of 20, 90, or 400 mg/kg. An increased incidence of renal tubular adenomas and carcinomas was found in male rats treated with the highest dose of entacapone. Plasma exposures (AUC) associated with this dose were approximately 20 times higher than estimated plasma exposures of humans receiving the maximum recommended daily dose of entacapone (MRDD = 1600 mg). Mice were treated once daily by oral gavage with doses of 20, 100 or 600 mg/kg of entacapone (0.05, 0.3, and 2 times the MRDD for humans on a mg/m² basis). Because of a high incidence of premature mortality in mice receiving the highest dose of entacapone, the mouse study is not an adequate assessment of carcinogenicity. Although no treatment related tumors were observed in animals receiving the lower doses, the carcinogenic potential of entacapone has not been fully evaluated. The carcinogenic potential of entacapone administered in combination with carbidopa-levodopa has not been evaluated.

Mutagenesis: Carbidopa was positive in the Ames test in the presence and absence of metabolic activation, was mutagenic in the *in vitro* mouse lymphoma/thymidine kinase assay in the absence of metabolic activation, and was negative in the *in vivo* mouse micronucleus test. Entacapone was mutagenic and clastogenic in the *in vitro* mouse lymphoma/thymidine kinase assay in the presence and absence of metabolic activation, and was clastogenic in cultured human lymphocytes in the presence of metabolic activation. Entacapone, either alone or in combination with carbidopa-levodopa, was not clastogenic in the *in vivo* mouse micronucleus test or mutagenic in the bacterial reverse mutation assay (Ames test).

Impairment of Fertility: In reproduction studies with carbidopa-levodopa, no effects on fertility were found in rats receiving doses of approximately two times the maximum daily human dose of carbidopa and four times the maximum daily human dose of levodopa. Entacapone did not impair fertility or general reproductive performance in rats treated with up to 700 mg/kg/day (plasma AUCs 28 times those in humans receiving the MRDD). Delayed mating, but no fertility impairment, was evident in female rats treated with 700 mg/kg/day of entacapone.

Pregnancy: Pregnancy Category C: Carbidopa-levodopa caused both visceral and skeletal malformations in rabbits at all doses and ratios of carbidopa-levodopa tested, which ranged from 10 times/5 times the maximum recommended human dose of carbidopa-levodopa to 20 times/10 times the maximum recommended human dose of carbidopa-levodopa. There was a decrease in the number of live pups delivered by rats receiving approximately two times the maximum recommended human dose of carbidopa and approximately five times the maximum recommended human dose of levodopa during organogenesis. No teratogenic effects were observed in mice receiving up to 20 times the maximum recommended human dose of carbidopa-levodopa. It has been reported from individual cases that levodopa crosses the human placental barrier, enters the fetus, and is metabolized. Carbidopa concentrations in fetal tissue appeared to be minimal. In embryofetal development studies, entacapone was administered to pregnant animals throughout organogenesis at doses of up to 1900 mg/kg/day in rats and 300 mg/kg/day in rabbits. Increased incidences of fetal variations were evident in litters from rats treated with the highest dose, in the absence of overt signs of maternal toxicity. The maternal plasma drug exposure (AUC) associated with this dose was approximately 34 times the estimated plasma exposure in humans receiving the maximum recommended daily dose (MRDD) of 1600 mg. Increased frequencies of abortions and late/total resorptions and decreased fetal weights were observed in the litters of rabbits treated with maternotoxic doses of 100 mg/kg/day (plasma AUCs 0.4 times those in humans receiving the MRDD) or greater. There was no evidence of teratogenicity in these studies. However, when entacapone was administered to female rats prior to mating and during early gestation, an increased incidence of fetal eye anomalies (macrophthalmia, microphthalmia, anophthalmia) was observed in the litters of dams treated with doses of 140 mg/kg/day (plasma AUCs 7 times those in humans receiving the MRDD) or greater. In the absence of maternotoxicity, administration of up to 700 mg/kg/day (plasma AUCs 28 times those in humans receiving the MRDD) to female rats during the latter part of gestation and throughout lactation, produced no evidence of developmental impairment in the offspring. There is no experience from clinical studies regarding the use of Stalevo in pregnant women. Therefore, Stalevo should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Women: In animal studies, carbidopa and entacapone were excreted into maternal rat milk. It is not known whether entacapone or carbidopa-levodopa are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Stalevo is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in pediatric patients have not been established.

ADVERSE REACTIONS: Carbidopa-Levodopa: The most common adverse reactions reported with carbidopa-levodopa have included dyskinesias, such as choreiform, dystonic, and other involuntary movements and nausea. The following other adverse reactions have been reported with carbidopa-levodopa: ***Body as a Whole:*** Chest pain, asthenia. ***Cardiovascular:*** Cardiac irregularities, hypotension, orthostatic effects including orthostatic hypotension, hypertension, syncope, phlebitis, palpitation. ***Gastrointestinal:*** Dark saliva, gastrointestinal bleeding, development of duodenal ulcer, anorexia, vomiting, diarrhea, constipation, dysapepsia, dry mouth, taste alterations. ***Hematologic:*** Agranulocytosis, hemolytic and non-hemolytic anemia, thrombocytopenia, leukopenia. ***Hypersensitivity:*** Angioedema, urticaria, pruritus, Henoch-Schönlein purpura, bullous lesions (including pemphigus-like reactions). ***Musculoskeletal:*** Back pain, shoulder pain, muscle cramps. ***Nervous System/Psychiatric:*** Psychotic episodes including delusions, hallucinations, and paranoid ideation, neuroleptic malignant syndrome *(see WARNINGS)*, bradykinetic episodes ("on-off" phenomenon), confusion, agitation, dizziness, somnolence, dream abnormalities including nightmares, insomnia, paresthesia, headache, depression with or without development of suicidal tendencies, dementia, increased libido. Convulsions also have occurred; however, a causal relationship with carbidopa-levodopa has not been established. ***Respiratory:*** Dyspnea, upper respiratory infection. ***Skin:*** Rash, increased sweating, alopecia, dark sweat. ***Urogenital:*** Urinary tract infection, urinary frequency, dark urine.

Laboratory Tests: Decreased hemoglobin and hematocrit; abnormalities in alkaline phosphatase, SGOT (AST), SGPT (ALT), lactic dehydrogenase, bilirubin, blood urea nitrogen (BUN), Coombs' test; elevated serum glucose; white blood cells, bacteria, and blood in the urine. Other adverse reactions that have been reported with levodopa alone and with various carbidopa-levodopa formulations, and may occur with Stalevo™ (carbidopa, levodopa and entacapone) are: ***Body as a Whole:*** Abdominal pain and distress, fatigue. ***Cardiovascular:*** Myocardial infarction. ***Gastrointestinal:*** Gastrointestinal pain, dysphagia, sialorrhea, flatulence, bruxism, burning sensation of the tongue, heartburn, hiccups. ***Metabolic:*** Edema, weight gain, weight loss. ***Musculoskeletal:*** Leg pain. ***Nervous System/Psychiatric:*** Ataxia, extrapyramidal disorder, flailing, anxiety, gait abnormalities, nervousness, decreased mental acuity, memory impairment, disorientation, euphoria, blepharospasm (which may be taken as an early sign of excess dosage; consideration of dosage reduction may be made at this time), trismus, increased tremor, numbness, muscle twitching, activation of latent Horner's syndrome, peripheral neuropathy. ***Respiratory:*** Pharyngeal pain, cough. ***Skin:*** Malignant melanoma *(see also CONTRAINDICATIONS)*, hair loss. ***Special Senses:*** Oculogyric crisis, diplopia, blurred vision, dilated pupils. ***Urogenital:*** Urinary retention, urinary incontinence, priapism. ***Miscellaneous:*** Bizarre breathing patterns, faintness, hoarseness, malaise, hot flashes, sense of stimulation.

Laboratory Tests: Decreased white blood cell count and serum potassium; increased serum creatinine and uric acid; protein and glucose in urine.

Entacapone: The most commonly observed adverse events (>5%) in the double-blind, placebo-controlled trials of entacapone (N=1003) associated with the use of entacapone alone and not seen at an equivalent frequency among the placebo-treated patients were: dyskinesia/hyperkinesia, nausea, urine discoloration, diarrhea, and abdominal pain. Approximately 14% of the 603 patients given entacapone in the double-blind, placebo-controlled trials discontinued treatment due to adverse events compared to 9% of the 400 patients who received placebo. The most frequent causes of discontinuation in decreasing order are: psychiatric reasons (2% vs. 1%), diarrhea (2% vs. 0%), dyskinesia/hypokinesia (2% vs. 1%), nausea (2% vs. 1%), abdominal pain (1% vs. 0%), and aggravation of Parkinson's disease symptoms (1% vs. 1%).

Adverse Event Incidence in Controlled Clinical Studies of Entacapone: Table 5 lists treatment emergent adverse events that occurred in at least 1% of patients treated with entacapone participating in the double-blind, placebo-controlled studies and that were numerically more common in the entacapone group, compared to placebo. In these studies, either entacapone or placebo was added to carbidopa-levodopa (or benserazide-levodopa).

Table 5: Summary of Patients with Adverse Events After Start of Trial Drug Administration At Least 1% in Entacapone Group and >Placebo

SYSTEM ORGAN CLASS, Preferred Term, Entacapone (n = 603) % of patients, Placebo (n = 400) % of patients.
SKIN AND APPENDAGES DISORDERS, Sweating Increased, 2, 1; **MUSCULOSKELETAL SYSTEM DISORDERS,** Back Pain, 7, 1 **CENTRAL & PERIPHERAL NERVOUS SYSTEM DISORDERS,** Dyskinesia, 25, 13; Hyperkinesia, 10, 5; Hypokinesia, 9, 8; Dizziness, 8, 6; **SPECIAL SENSES, OTHER DISORDERS,** Taste Perversion, 1, 0; **PSYCHIATRIC DISORDERS,** Anxiety, 2, 1; Somnolence, 2, 0; Agitation, 1, 0; **GASTROINTESTINAL SYSTEM DISORDERS,** Nausea, 14, 6; Diarrhea, 10, 4; Abdominal Pain, 8, 4; Constipation, 6, 4; **GASTROINTESTINAL SYSTEM DISORDERS,** Vomiting, 4, 1; Mouth Dry, 3, 0; Dyspepsia, 2, 1; Flatulence, 2, 0; Gastritis, 1, 0; Gastrointestinal Disorders NOS, 1, 0; **RESPIRATORY SYSTEM DISORDERS,** Dyspnea, 3, 1; **PLATELET, BLEEDING & CLOTTING DISORDERS,** Purpura, 2, 1; **URINARY SYSTEM DISORDERS,** Urine Discoloration, 10, 0; **BODY AS A WHOLE - GENERAL DISORDERS,** Back Pain, 4, 2; Fatigue, 6, 4; Asthenia, 2, 1; **RESISTANCE MECHANISM DISORDERS,** Infection Bacterial, 1, 0.

The prescriber should be aware that these figures cannot be used to predict the incidence of adverse events in the course of usual medical practice where patient characteristics and other factors differ from those that prevailed in the clinical studies. Similarly, the cited frequencies cannot be compared with figures obtained from other clinical investigations involving different treatments, uses, and investigators. The cited figures do, however, provide the prescriber with some basis for estimating the relative contribution of drug and nondrug factors to the adverse events observed in the population studied.

Effects of Gender and Age on Adverse Reactions: No differences were noted in the rate of adverse events attributable to entacapone alone by age or gender.

DRUG ABUSE AND DEPENDENCE: ***Controlled substance class:*** Stalevo™ (carbidopa, levodopa and entacapone) is not a controlled substance.

Physical and psychological dependence: Stalevo has not been systematically studied, in animal or humans, for its potential for abuse, tolerance or physical dependence. In premarketing clinical experience, carbidopa-levodopa did not reveal any tendency for a withdrawal syndrome or any drug-seeking behavior. However, there are rare post-marketing reports of abuse and dependence of medications containing levodopa. In general, these reports consist of patients taking increasing doses of medication in order to achieve a euphoric state. Store at 25°C (77°F); excursions permitted to 15°C-30°C (59°F-86°F). [See USP Controlled Room Temperature.] Dispense in light container (USP).

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MONDAY, JUNE 14

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P286 The cognitive profile of primary dystonia: Preliminary findings of a prospective study
J. Mueller, L. Bartha, W. Eisner, G.K. Wenning, T. Benke, W. Poewe

POSTER SESSION 1

P287 Deep brain stimulation on the cervico-axial dystonia, long-term results
D. Gaudin, L. ClF, P. Coubes, G. Bouvier

P288 Survey of sensory and motor tricks
S. Kanchana, H. Shill, M. Wong, M. Hallett

P289 Antibasal ganglia antibodies in atypical dystonia and tics: A prospective study
M.J. Edwards, E. Trikouli, D. Martino, A.J. Church, G. Giovannoni, K.P. Bhatia

P290 Genetic heterogeneity in rapid-onset dystonia-parkinsonism: Description of a new family
K. Kabakci, K. Isbruch, K. Hedrich, P.L. Kramer, M. Schwarz, C. Klein

P291 Efficacy and safety of a new botulinum toxin type A free of complexing proteins in treatment of cervical dystonia
R. Benecke

P292 Two-year follow-up of botulinum toxin B treatment in type A resistant primary dystonia
T.M. Entner, J. Mueller, G.K. Wenning, W. Poewe

P293 Psychogenic dystonia: Clinical characteristics and long term progression
M. Thomas, K.D. Vuong, J. Jankovic

P294 Integrated approach to cervical dystonia with botulinum toxin and neuromotor rehabilitation
C. Tassorelli, F. Mancini, G. Sandrini, R. Zangaglia, G. Nappi, C. Pacchetti

P295 Neuropathology in DYT1/torsina-linked dystonia
C.W. Olanow, K.S. McNaught

P296 Segmental dystonia responsive to amiodarone
K.P. Frei, M. Pathak, D.D. Truong

P297 Cervical dystonia associated with droopy shoulder syndrome
J.-S. Liu, M.-Y. Lan, C.-C. Chang, C.-S. Su, H.-S. Wu, Y.-Y. Chang

P298 Bilateral pallidotomy for generalized dystonia: A Cuban experience
C. Maragoto, G. Rodríguez, G. Lopez, R. Melo, L. Alvarez

P299 Psychogenic facial spasm (The Smirk)
D. Tarsy, A. Schrag, N. Quinn, K. Bhatia

P300 Clinical presentation of familial and sporadic primary dystonia in a cohort of adult patients
H. Shang, N. Clerc, D. Lang, J.-M. Burgunder, A. Kaelin-Lang

P301 Dystonia as a presentation of anti-Hu paraneoplastic syndrome
G.D. O' Connor, P. Hodnett, D. Schmidt, B. Sweeney

P302 Acquired paroxysmal torticollis and bilateral belpharospasm following bilateral cerebellar infarction
K. O'Rourke, M. Hutchinson

P303 Generalized dystonia associated with Lipoid Proteinosis (Urbach-Wiethe disease): A case report
H.A. Teive, E.R. Pereira, S. Raskin, T. Hamada, J.A. McGrath, L.C. Werneck

P304 Status dystonicus: Report of four cases
H.A. Teive, M.M. Souza, S.A. Antoniuk, E.R. Barbosa, M. Scaff, L.C. Werneck

P305 The age of onset in cervical dystonia is independent of level of education
J.P. O'Dwyer, M. Hutchinson

P306 Clinical and genetic variability in myoclonus-dystonia syndrome
B. Garavaglia, D. Ghezzi, G. Zorzi, C. Ciano, C. Barzaghi, N. Nardocci

P307 Unilateral pallidotomy for primary hemidystonia
A. Alkhani, S. Bohlega

P308 Brain activation patterns during motor tasks in patients with epsilon-sarcoglycan mutation positive myoclonus-dystonia
A.B. Deutschländer, T. Stephan, M. Naumann, T. Gasser, T. Brandt, F. Asmus

P309 Hallervorden-Spatz Syndrome in Thailand
K. Phanthumchinda, Y. Likitjaroen

P310 Sensory thresholds using grating orientation tasks at the fingertip in cervical dystonia
D. Weise, A. Schramm, K. Stefan, A. Wolters, K. Reiners, M. Naumann

P311 Disturbance of associative motor cortical plasticity in focal hand dystonia
J.P. O'Dwyer, M. Hutchinson

P312 Transcutaneous electrical nerve stimulation (TENS): A new therapeutic approach for writer's cramp dystonia
M. Tinazzi, S. Farina, A. Fiaschi, G. Moretto, L. Bertolasi, S. Zarattini, N. Smania

Gene Therapies and Cell-Based Therapies

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P313 Dopaminergic protection and regeneration by neurturin-expressing c17.2 neural stem cells in rat model of Parkinson's disease
S. Chen, W. Liu, G. Lu, B. Li

P314 Restoring reinnervation of the dopaminergic system indced by GDNF in an experimental model reproducing a presymptomatic phase of Parkinson's disease
C. Carcenac, M. Brizard, J. Mallet, M. Savasta

P315 Switching cell fate with human Neuro-D1
J. Sanchez-Ramos, S. Kamath, P. Walczak, N. Chen, R. Heller, T. Zigova

P316 Transplantation of dopamine neurons derived from primate embryonic stem cells
R. Sanchez Pernaute, L. Studer, D. Ferrari, A. Perrier, A. Ferree, O. Isacson

P317 Ectopic expression of α-synucleins promotes neuronal differentiation of murine neural stem cells (NSC)
M. Jungnitsch, A. Storch, J. Schwarz

P318 Electrophysiological characterization of human fetal mesencephalic progenitor cells and derived neurons
F. Wegner, S.C. Schwarz, E. Frick, J.W. Wieacker, A. Storch, J. Schwarz

P319 Human neural precursor cells reverse functional deficits in 6-OHDA lesioned rats
S.C. Schwarz, W. Jan, W. Dirk, E. Susanne, S. Alexander, S. Johannes

P320 Proliferation and differentiation of murine neural stem cells: Effect of oxygen *in vitro*
J. Milosevic, M. Poppe, S.C. Schwarz, A. Storch, J. Schwarz

P321 Reversal of L-DOPA-induced dyskinesia and motor impairments by AAV vector-mediated gene transfer of TH and GTPCH1 in a rat model of Parkinson's disease
C. Winkler, T. Carlsson, N. Muzyczka, R. Dengler, A. Bjorklund, D. Kirik

P322 Functional effects of GDNF gene therapy on motor performance in parkinsonian rats with intrastriatal dopaminergic transplants
C. Winkler, B. Georgievska, T. Carlsson, R. Dengler, A. Bjorklund, D. Kirik

Myoclonus

Poster numbers 323-333

P323 Propriospinal myoclonus (PSM): A motor phenomenon found in restless legs syndrome (RLS) and different from Periodic Limb Movements during Sleep (PLMS)
R. Vetrugno, G. Plazzi, F. Provini, E. Lugaresi, P. Montagna

P324 EEG-EMG and EMG-EMG frequency analysis in Dutch patients with 'familial cortical myoclonus or tremor with epilepsy'
A.-F. van Rootselaar, N.M. Maurits, J.H. Koelman, K.L. Leenders, P. Brown, M.A. Tijssen

P325 'Familial cortical myoclonus or tremor with epilepsy' and cerebellar pathology
A.-F. van Rootselaar, E. Aronica, E.N. Jansen Steur, J.M. Rozemuller-Kwakkel, R.A. de Vos, M.A. Tijssen

P326 Trunk tremor as part of the myoclonus-dystonia phenotype in a large Dutch family
E.M. Foncke, C. Klein, M. Gerritz, K. Hedrich, C.C. Tijssen, M.A. Tijssen

P327 Analysis of fragmentary myoclonus in sleep: Frequency, distribution in sleep stages and association with sleep disorders
A.B. Kunz, B. Hoegl, W. Poewe

P328 A case of Guillain-Barre syndrome with peripheral myoclonus and syndrome of painful legs and moving toes
M. Bozi, K. Filippopolitis, I. Hatzigeorgiou, M. Tzortzi, A. Georgali

P329 Myoclonus in cortico-basal degeneration: Is it of cortical origin?
Z. Mari, M. Matsuhashi, H. Shibasaki, M. Hallett

P330 'Familial cortical myoclonus or tremor with epilepsy': A review on the clinical, genetic and electrophysiological aspects
A.-F. van Rootselaar, I.N. van Schaik, P.M. Callenbach, A.M. van den Maagdenberg, J.H. Koelman, M.A. Tijssen

P331 Diaphragmatic myoclonus successfully treated with botulinum toxin
P. Simal, J.C. Martinez-Castrillo, F. Vivancos

P332 Myoclonus associated with long-standing poliomyelitis is due to central reorganization
C. Cordivari, N.J. Toms, S. Catania, V.P. Misra, A.J. Lees, P. Brown

P333 Action myoclonus - Renal failure syndrome; a further case report
L. Vadlamudi, F.L. Ierino, S.F. Berkovic, A.J. Hughes

Spasticity

Poster number 334-343

P334 Two year randomised, double blind, parallel group, placebo controlled, multi-injection cycle trial of treatment with botulinum toxin A for leg spasticity in cerebral palsy
A.P. Moore, R.A. Ade-Hall, C. Tudor-Smith, L.R. Rosenbloom, J. Walsh, K. Mohamed

P335 Short-term electrical stimulation enhances the effectiveness of botulinum toxin in spasticity
E. Frasson, B. Ruzzante, G. Didonè, M. Bottanelli, A. Priori, L. Bertolasi

P336 A way to improve the effectiveness of botulinum toxin in spinal cord injury patients
W.A. Raza, N. Green, H. Francis

P337 Perception of emotional prosody in Parkinson's disease
C. Schroeder, S. Martin, F. Szymanowski, W. Nager, T.F. Muente, R. Dengler

P338 Intrathecal baclofen pump therapy improves functional motor control and quality of life in spastic hemiplegia after stroke
R. Izor, S. Fisher, R. Simpson, K. Johnson, T. Tran, M. Schiess

P339 Ocular myasthenia following botulinum toxin type A injection for limb spasticity
M. Umaiorubahan, V.C. Uthamarayan

P340 Treatment of spasticity with botulinum toxin: A ten-years follow-up study
M. Bottanelli, S. Vicentini, G. Rossato, E. Fincati, N. Rizzuto, L. Bertolasi

P341 The synthetic cannabinoid Nabilone® reduces spasticity-associated pain: A double-blind placebo-controlled cross-over trial
J. Wissel, T. Entner, J. Mueller, C. Brenneis, T. Berger, W. Poewe

P342 A double-blind, randomised, placebo-controlled study to evaluate efficacy and safety of botulinum toxin type b (myobloc/neurobloc) and botulinum toxin type A (dysport) for the treatment of spastic paraparesis
F. Mancini, A. Moglia, M. Allena, G. Sandrini, G. Nappi, C. Pacchetti

P343 Cocontractions related to obstetrical brachial plexus palsy treated with botulinum toxin
L.R. Gonçalves, L. Silveira-Moriyama, C.O. Heise, J.L. Gherpelli, E.R. Barbosa

In early Parkinson's disease (PD)

Initiate MIRAPEX now for long-term benefits

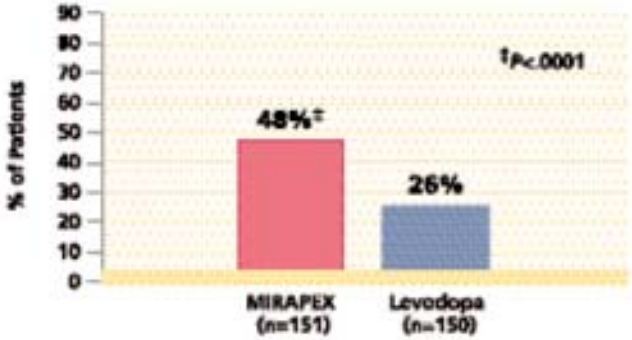
Delays the need for levodopa

At 4 years, there is a 41% probability that patients initiated with MIRAPEX are still on monotherapy[1,2*]

Delays onset of motor complications

After 4 years, 48% of patients initiated on MIRAPEX alone were free of any major motor complication vs 26% of patients initiated on levodopa[2]

Free of any major motor complication at 4 years[2†]



MIRAPEX is indicated for the treatment of the signs and symptoms of idiopathic Parkinson's disease.

Patients have reported falling asleep without perceived warning signs during daily activities, including operation of a motor vehicle, which sometimes resulted in accidents. Hallucinations and postural (orthostatic) hypotension may occur.

The most commonly reported adverse events in early and late disease in clinical trials were dizziness, dyskinesia, EPS, hallucinations, headache, insomnia, somnolence, and nausea.

*The probability is based on a survival analysis of a 48-month maintenance dose, open-label, long-term safety study using the life-table method for 225 patients with early PD (Hoehn and Yahr stages I-III). This study was an extension of an 11-week, double-blind, dose-ranging trial.

At 48 months, 60 patients had taken MIRAPEX continuously without the addition of levodopa. The remaining patients had begun levodopa, discontinued the trial, or had not yet reached the 4-year time point.

†Based on a 4-year, double-blind, randomized, controlled trial of 301 patients with early PD (Hoehn and Yahr stages I-III). Primary outcome was time from randomization to first occurrence of wearing off, dyskinesias, or on-off fluctuations, as measured by the Unified Parkinson's Disease Rating Scale.

References: 1. Barone P, Bressman S. Pramipexole without levodopa as early treatment for Parkinson's disease: a long-term follow-up of 717 patients. Poster presented at: 53rd Annual American Academy of Neurology; [illegible]; Philadelphia, Pa. **2.** Data on file, Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, CT.

Please see Brief Summary of Prescribing Information on adjacent page.

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WARNINGS

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ADVERSE REACTIONS

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DRUG ABUSE AND DEPENDENCE

[illegible]

OVERDOSAGE

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DOSAGE AND ADMINISTRATION

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POSTER SESSION 2

TUESDAY, JUNE 15

Poster Viewing: 8:30 am to 5:00 pm
Authors Present Odd Numbers: 11:30 am to 12:30 pm
Authors Present Even Numbers: 4:00 pm to 5:00 pm

Parkinson's disease 1

Poster numbers 344-694

P344 Dyskinesias predict the onset of motor response fluctuations in patients with Parkinson's disease on L-dopa monotherapy
L. Mazzella, N. Huang, A. Di Rocco, M.D. Yahr

P345 An assessment of the impact of Parkinson's disease on quality of life
W.C. Koller, M.B. Stern, L. Stone, J. Blazer

P346 The Hong Kong Parkinson's Disease Registry: A multi-centre study of clinical and treatment profiles of ethnic Chinese patients using strict diagnostic criteria
J.H. Yeung, on Behalf of the Hong Kong Parkinson's Disease Registry Study Group

P347 Immune-inflammatory changes in the substantia nigra in Parkinson's disease
C.F. Orr, D.B. Rowe, G.M. Halliday

P348 Long-term efficacy and safety of Zydis® selegiline in Parkinson's disease (PD)
M.F. Lew, R. Pahwa, J. Bertoni

P349 Emergency hospital admissions in idiopathic Parkinson's disease
H.J. Woodford, R.W. Walker

P350 Spinal cord inhibitory mechanisms in early onset Parkinson' s disease: Evaluation of Ib inhibition into soleus motor neurons
C.L. Scaglione, G. Rizzo, G. Lopane, M. Marchi, D. Resi, P. Martinelli

P351 Orthostatic hypotension and cognitive impairment in Parkinson's disease: A cross sectional community-based study
L.M. Allcock, S. Tordoff, T. Hildreth, K. Wesnes, R. Kenny, D.J. Burn

P352 Pathoarchitectonic staging of brain destruction related to idiopathic Parkinson's disease
H. Braak, K. Del Tredici, U. Rueb, R. de Vos, E. Steur, E. Braak

P353 Ergot side-effect issues in dopamine agonist treatment of Parkinson's disease
K.A. Grosset, F. Needleman, G. Macphee, D.G. Grosset

P354 Nurr1 gene targeting therapy for Parkinson disease
W. Le, Q. Jiang, W. Xie, S. Hintermann, J. Jankovic

P355 Extensive oxidative stress and microglial activation in substantia nigra following instrastriatal MPP+ injection
H. Miwa, Y. Kubo, S. Morita, I. Nakanishi, T. Kondo

P356 Are panic attacks and freezing episodes related?
A.N. Lieberman, C. Singer, A.N. Neophytides

P357 Sex, gambling and Parkinson disease (PD)
A.N. Lieberman, A.R. Rezai

P358 Statins, Co Q 10, and Parkinson disease (PD): Is there a relationship?
A.N. Lieberman, J. Levine, R. Myerburg, L. Vela

P359 In vivo proton MR spectroscopy study of brain metabolism in Early-Onset Parkinson's disease
C. Tonon, C.L. Scaglione, R. Lodi, S. Iotti, B. Barbiroli, P. Martinelli

P360 Polysomnographic studies in Parkinson's disease
D. Lee, K. Park, S. Koh, J. Han

P361 Abnormal temporal discrimination threshold and its responsiveness to levodopa treatment in Parkinson's disease
M. Lee, H. Kim, C. Lyoo, J. Kim

P362 Parkinson's disease as an asymmetrical disorder: Does it matter which side presents first?
J.H. Yeung, on Behalf of the Hong Kong Parkinson's Disease Registry Study Group

P363 Gender differences in clinical features of Chinese Parkinson's disease patients based on strict diagnostic criteria
J.H. Yeung, on Behalf of the Hong Kong Parkinson's Disease Registry Study Group

P364 Reduction of myocardial MIBG uptake is correlated with cognitive impairment in patients with Parkinson's disease
K. Kashihara, M. Ohno

P365 Non-motor off symptoms in Parkinson's disease
J.W. Kim, W.J. Kim, S.M. Chun

P366 Cochrane systematic review of catechol-O-methyl transferase (COMT) inhibitors for levodopa-induced complications in Parkinson's disease
C.E. Clarke, K.H. Deane, S. Spieker

P367 A clinical and videolaryngoscopic study in PD patients
M. Behari, J.P. Lazarus, K.K. Handa, T. Srivastava, V. Goyal, S. Singh

P368 Voice profile and acoustic signs in Indian Parkinson's disease patients
M. Behari, J.P. Lazarus, K.K. Handa, V. Goyal, S. Singh, T. Srivastava

P369 High dose dopamine agonist treatment
P. Odin, A. Storch, U. Polzer, G. Werner, R. Renner, M. Shing

P370 The prevalence of Parkinson's disease in an area of North Tyneside in the North East of England
R.W. Porter, R. Macfarlane, R. Walker

P371 Stimulating music increases fine motor coordination in patients afflicted with morbus Parkinson
G.J. Bernatzky, P.P. Bernatzky, H.P. Hesse, E. Mueller, M. Grobovschek, G. Ladurner

P372 Degenerative parkinsonian syndromes assessed by HmPaO-SPECT: The utility of factorial discriminant analysis
A. Kreisler, L. Defebvre, P. Lecouffe, A. Duhamel, M. Steinling, A. Destée

P373 Effectiveness of milnacipran in treatment of depression associated with Parkinson's disease
T. Maruyama, T. Hashimoto, Y. Chiba

P374 Double vision in Parkinson's disease
A. Nebe, E. Georg

P375 Gait changes in de novo Parkinson's disease patients: A force / rhythm dichotomy
R. Baltadjieva, N. Giladi, Y. Balash, T. Herman, J.M. Hausdorff

P376 Treadmill walking as an external cue to improve gait rhythm and stability in Parkinson's disease
S. Toledo-Frankel, N. Giladi, L. Gruendlinger, R. Baltadjieva, T. Herman, J.M. Hausdorff

P377 Effects of rhythmic auditory stimulation on gait dynamics in Parkinson's disease
J. Lowenthal, L. Gruedlinger, R. Baltadjieva, T. Herman, J.M. Hausdorff, N. Giladi

P378 Executive function, mental loading and gait variability in Parkinson's disease
G. Yogev, N. Giladi, E.S. Simon, L. Gruendlinger, J.M. Hausdorff

P379 Clinical and electrophysiological study of hand deformity in Parkinson's disease
H. Okano, C. Ooishi, K. Nishiyama, M. Sakuta

P380 Electroconvulsive therapy in Parkinson's disease
R. Faber

P381 Activation in deep brain structures during therapeutic DBS of the STN in patients with PD revealed by functional MRI
K.B. Baker, S. Cooper, B. Kopell, M.D. Phillips, M.J. Lowe, A.R. Rezai

P382 Value of early [^{123}I]β-CIT SPECT imaging in the differential diagnosis of parkinsonian syndromes
D. Stoffers, L. Bosscher, J. Booij, A. Winogrodzka, E.C. Wolters, H.W. Berendse

P383 The effect of botulinum toxin injections to the calf muscles on freezing of gait in Parkinson's disease, double blind placebo controlled study
T. Gurevich, C. Peretz, O. Moore, N. Weizmann, N. Giladi

P384 The association between the level of education and neuropsychiatric manifestations in Parkinson's disease
O.S. Cohen, D. Tanne, E. Vakil, Z. Nitsan, J. Chapman, S. Hassin-Baer

P385 Exercise habits of patients with Parkinson's disease: A survey based study
A. Daley Ryan, A. Kaszmarek, T. Simuni

P386 Parkinsonism with excessive daytime sleepiness: A narcolepsy-like disorder?
C. Baumann, L. Ferini-Strambi, D. Waldvogel, E. Werth, M. Bischof, C.L. Bassetti

P387 Prevalence of Parkinson's disease in the Eolian Archipelago
L. Morgante, M. Zappia, U. Bonuccelli, R. Marconi, Q. Aldo, S. Giovanni

P388 Metric properties of a fatigue impact scale for daily use (D-FIS) in Parkinson's disease
P. Martinez-Martin, M.J. Catalan, A. Ortega-Moreno, J. Benito-Leon, I. Zamarbide, E. Cubo

P389 The SCOPA-Motor Scale for assessment of Parkinson's disease: Consistency and validity
P. Martinez-Martin, J. Benito-Leon, J.A. Burguera, A. Castro, G. Linazasoro, J.C. Martinez-Castrillo

P390 Genetic analysis of DJ-1 in ethnic Chinese and Malay patients with Parkinson's disease
V.R. Chandran, C. Tan, Z. Yi, K. Yew, S. Hui, E.K. Tan

P391 Falls in Parkinson's disease: Clinico-statistical analysis
Y. Balash, C. Peretz, G. Leibovich, T. Herman, J.M. Hausdorff, N. Giladi

P392 Nocturnal body movements in Parkinson's disease: Large ranges in Hoehn and Yahr stages II to IV!
N. Kuehnl, M.H. Strothjohann, D.J. Emmans, G.A. Fuchs

P393 Identification and characterization of a novel biomarker that specifically predicts Parkinson's disease
G.M. Pasinetti

P394 Pergolide associated valvulopathy: A Belgian experience
F.L. Evrard, A. Flamez, G. Van Camp, T. Muller, M.J. Dupuis, C. Dilman

P395 The dopamine receptor agonist lisuride attenuates MPP^+-induced dopaminergic neurodegeneration in C. elegans
M. Gerlach, E. Braungart, K. Tovar, M. Hoener, A. Engfer, P. Riederer

P396 Effect of rotigotine on locomotor activity and disability scores in MPTP-treated common marmosets following subcutaneous administration
D.K. Scheller, L. Smith, A. Breidenbach, M. Jackson, S. Rose, P. Jenner

P397 Static and dynamic posturometry: Effect of deep brain stimulation of the subthalamic nucleus on postural stability in Parkinson disease
D. Guehl, P. Dehail, M. de Sèze, E. Cuny, M. Barat, P. Burbaud

P398 Healthcare utilization and expenditures among privately insured patients with Parkinson's disease in the United States
L.S. Orsini, J. Castelli-Haley, S. Kennedy, D.M. Huse

P399 Gait festination in Parkinson: Insights into pathophysiological mechanisms
R. Iansek, J.L. McGinley, F.E. Huxham

P400 Therapy of drug-induced psychosis in PD in the GP and specialists' office in Germany
D. Brandstaedter, A. Schneider, J. Lotze, S. Spieker, G. Ulm, W.H. Oertel

P401 Effects of ropinirole on postural stability in Parkinson's disease: A computerized dynamic posturography study
G.-H. Lee, J.-I. Kim

P402 Effect of zonisamide on parkinsonian tremor: A pilot cross-over study in comparison with trihexyphenidyl
I. Nakanishi, J. Kohmoto, H. Miwa, T. Kondo

P403 Response of tremor to subcutaneous apomorphine in patients with tremor predominant idiopathic Parkinson's disease
M. Hellmann, T. Sabach, E. Melamed, R. Djaldetti

P404 Prevalence studies in Parkinson's disease: A systematic review of prospective data sets
R. Almeida-Freire, J.J. Ferreira, J. Costa, C. Sampaio

P405 Effect of locus coeruleus noradrenaline depletion on levodopa-induced motor fluctuations in hemiparkinsonian rats
C. Marin, E. Aguilar, M. Bonastre, B. Verhoeff

P406 Is drug profile a risk factor to hallucinations emergence in patients with advanced Parkinson's disease?
D. Merims, H. Shabtai, N. Weizman, C. Peretz, A. Korczyn, N. Giladi

P407 Cortical and thalamic cholinergic activity in Lewy body disease in relation to dementia and parkinsonism
I. Ziabreva, C. Ballard, D. Aarsland, J.-P. Larsen, E. Perry

P408 Mitochondrial complex I activity in familial and sporadic Parkinson's disease
H.A. Hanagasi, D. Ayribas, K. Baysal, M. Emre

P409 Effects on cardiovascular regulation of bilateral subthalamic nucleus stimulation in Parkinson patients
P. Cortelli, G. Barletta, P. Guaraldi, L. Romito, G. Pierangeli, A. Albanese

P410 Atypical clinical presentations in patients with parkin gene mutations
E. Munoz, J. Campdelacreu, F. Valldeoriola, M.J. Marti, E. Tolosa

P411 Load force during manual transport in Parkinson's disease
X. Guo, N. Hosseini, T. Zackrisson, B. Eriksson, B. Johnels, G. Steg

P412 Modulation of serotonin receptors in MPTP-lesioned monkeys
C. Chassain, A. Eschalier, F. Durif

P413 Cerebral glutamate (Glu) metabolism in Parkinsons disease (PD) model rat: An *in vivo* dynamic ^{13}C nuclear magnetic spectroscopy (NMS) study
C. Chassain, G. Bielicki, J.-P. Donnat, J.-P. Renou, A. Eschalier, F. Durif

P414 Parkinson's disease sleep scale in Korea
J. Baik, J. Kim, J. Park, Y. Kim

P415 Effect of rate of execution on predictable sequences of saccades in Parkinson's disease
B. Spieth, M.R. MacAskill, T.J. Anderson

P416 Group intervention for improving speech abilities and communication skills in patients with Parkinson's disease
Y. Manor, J. Posen, O. Amir, N. Dori, N. Giladi

P417 The effects of levodopa treatment on speech rate and dysfluency characteristics in patients with Parkinson's disease
Y. Manor, G. Ovadia, H. Zadok, H. Shabtai, R. Ezrati-Vinacour, N. Giladi

P418 Prenatal Lipopolysaccharide (LPS) induces permanent reductions in dopamine neurons: Effect of epigenetic alterations in CNS inflammatory homeostasis as an animal model for Parkinson's disease
P.M. Carvey, Q.A. Chang, Z. Ling

P419 Assessment of autonomic function in patients with a parkinsonian syndrome
D.I. Tiple, S.S. Odobescu, V.I. Vovc, I.I. Moldovanu

P420 Malignant syndrome in Parkinson' disease: Presentation of three cases occurring during the 2003 heat wave
C. Gaig, M. Gomez-Choco, S. Amaro, M. Marti, E. Tolosa

P421 Echogenicity of the substantia nigra in Parkinson's disease – an open and controlled pilot study
P. Ressner, D. Skoloudik, P. Kanovsky

P422 Timed tests detect age-related motor performance decline in normal controls
P.J. Garcia Ruiz, V. Sanchez Bernardos

P423 Abnormal stance strategy is found in PD patients
E. Weissblueth, M. Schwartz, S. Hocherman

P424 Parkinson's disease in the Navajo people: Evalutation of 100 patients
J. Marjama-Lyons, L. Clark, A. Singleton, J. Barr, K. Parko

P425 The effect of pramipexol on the maintenance of wakefulness in Parkinson's disease
H. Kumru, J. Santamaria, F. Valldeoriola, F. Marti, E. Tolosa

POSTER SESSION 2

P426 Are there gender differences in patients attending a Parkinson's disease clinic?
J.C. Sharma, L. Macnamara

P427 Early and late effectiveness of subthalamic nucleus stimulation in Parkinson's disease
D. Guehl, E. Cuny, A. Benazzouz, A. Rougier, F. Tison, P. Burbaud

P428 Lets talk about sex. Preliminary results of a sexual dysfunction study in people with PD
A. Bowron, R. Porter, B. Chandler, R. Walker

P429 Relationship between age and type of psychotic symptoms in Parkinson's disease
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P538 Rasagiline treatment can improve freezing of gait in advanced Parkinson's disease; a prospective randomized, double blind, placebo and entacapone controlled study
*N. Giladi, on Behalf of the European LARGO Study Group***

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P563 A comparative randomised study of rasagiline versus placebo or entacapone as adjunct to levodopa in Parkinson's disease (PD) patients with motor fluctuations (the LARGO Study)
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P568 The incidence and clinical significance of orthostatic hypotension in advanced Parkinson's disease patients treated with subcutaneous apomorphine
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P580 Frequency of orthostatic hypotension as a cause of orthostatic intolerance in Parkinson disease
K.F. Nahm, S. Nouri, M.D. Yahr, H.C. Kaufmann

P581 Fibroblast growth factor 20 polymorphisms and haplotypes strongly influence risk of Parkinson disease
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P582 A longitudinal study of the motor response to levodopa in Parkinson's disease
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P583 Saccadic eye movements in Parkinson's disease with and without dementia
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P584 The PDQ-39 is a sensitive measure of change in quality of life in early Parkinson's disease
N.J. Ives, C. Jenkinson, R. Fitzpatrick, K. Wheatley, C.E. Clarke, PD MED Collaborative Group

P585 Effect of rasagiline on severity of OFF in Parkinson's disease
*F. Stocchi, on Behalf of the LARGO Study Group***

P586 Dopamine agonist therapy in early Parkinson's disease: A systematic review of randomised controlled trials
N.J. Ives, R.L. Stowe, C.E. Clarke, R. Gray, K. Wheatley, L. Shah

P587 Growth hormone response to low-dose apomorphine in patients with restless legs syndrome and Parkinson's disease
S. Happe, K. Helmschmied, T. Tings, W. Wuttke, W. Paulus, C. Trenkwalder

P588 Relationship between nigrostriatal dopaminergic degeneration and urinary symptoms in Parkinson's disease
K. Winge, L. Friberg, L. Werdelin, K.K. Nielsen, H. Stimpel

P589 Rapid improvement in balance of patients with Parkinson's disease through training based on movements guided by rhythmic cues
M.E. Piemonte, E.S. Takata, M.C. Moura, T.T. Capato, M.C. Fornari, E.R. Barbosa

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P590 Rapid gait improvement in patients with Parkinson's disease through training based on movements guided by rhythmic cues
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P591 Assessment into the impact of depression, fatigue, apathy and daytime sleepiness on motor performance, independence in daily life activities and quality of life for patients with Parkinson's disease
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P592 A unilateral toxin-induced mouse model for Parkinsons disease
R. Iancu, P. Mohapel, P. Brundin, G. Paul

P593 Long term effectiveness of a physiotherapeutic scheme, based on a supervised weekly program of home-exercises, for patients with Parkinson's disease in early and advanced stages of evolution: 24-month interim report
M.E. Piemonte, D.M. Almeida, K. Burgi, M.C. Melo, M.M. Morimoto, E.R. Barbosa

P594 Effects of bilateral and unilateral subthalamic nucleus deep brain stimulation on Parkinson disease symptoms
M. Ushe, M. Hong, J.W. Mink, F. Revilla, J.L. Dowling, J.S. Perlmutter

P595 A new bilateral rat model of Parkinson's disease
V. Paillé, L. Lescaudron, P. Brachet, P. Damier

P596 A prospective study of reduced impulse control in patients with idiopathic Parkinson's disease
C.S. Kubu, R.M. Busch, R. Shaw, H.D. Stott, A. Ahmed, A. Rezai

P597 Oral festination in Parkinson's disease
C. Moreau, C. Ozsancak, J.L. Blatt, P. Derambure, A. Destee, L. Defebvre

P598 Parenteral treatment of acute psychosis in Parkinson's disease with ziprasidone
M. Oechsner, A. Korchounov

P599 Levodopa-induced hyperhomocysteinemia and cardiovascular dysfunction in Parkinson's disease
T.A. Zesiewicz, B. Giunta, R.A. Hauser, M. Hoffman, P. Wallach, K.L. Sullivan

P600 Alpha -synuclein pathology does not predict extrapyramidal signs or cognitive impairment
I. Alafuzoff, T. Kauppinen, T. Pirttilä, J. Autere, L. Parkkinen

P601 Prospective study on the use of ropinirole in patients with advanced Parkinson's disease
M. Relja, S. Telarovic

P602 Abnormal cortical oscillatory activity in voluntary muscle relaxation in de novo Parkinson's disease
E.E. Labyt, F.F. Cassim, D.D. Devos, J.-L.J. Bourriez, L.L. Defebvre, P.P. Derambure

P603 Piribedil efficacy in monotherapy (150 to 300 mg/day) in de novo parkinsonian patients: A 6-month planned intermediate ANALYSIS of the 2-year Parkinson-regain study
O. Rascol, O. Gershanik, O. Blin, J. Ferreira, N. Bodjarian, A. Lees

P604 The pharmacological and biological effects of pramipexole on dopamine neuron associated genes: DAT, VMAT-2, and Nurr1
T. Pan, W. Xie, J. Jankovic, W. Le

P605 Istradefylline (KW-6002) as adjunctive therapy in patients with advanced Parkinson's disease: A positive safety profile with supporting efficacy
M.A. Stacy, the US-005 and US-006 Investigator Group

P606 Combined use of NMDA and AMPA antagonists further reduces levodopa-induced dyskinesias in MPTP-lesioned primates
F. Bibbiani, A. Kielaite, T.N. Chase

P607 Onset of action of intermittent subcutaneous apomorphine in the treatment of "off" episodes in patients with advanced Parkinson's disease
K.L. Hull, Jr., L. Gutman, the APO-302 Investigators

P608 Is levodopa-induced dyskinesias risk decreased in parkinsonian patients initially treated with dopamine agonist? A longitudinal study among 425 patients
L. Ouchchane, S. Perrette, N. Saïkali, B. Aublet-Cuvelier, F. Durif

P609 Decrease in UPDRS motor scores following intermittent subcutaneous apomorphine for 6 months in patients with advanced Parkinson's disease
D.M. Trosch, the APO-303 Investigators

P610 Subthalamic nucleus stimulation improves manipulative finger force control in Parkinson's disease
D.A. Nowak, S. Tisch, M. Hariz, P. Limousin, J.C. Rothwell, H.R. Topka

P611 Parkin is a potential modifier of the phenotype of α-synuclein-associated familial Parkinson's disease
K. Markopoulou, D.W. Dickson, R.D. McComb, L. Avery, B.A. Chase

P612 Incidence of PD, depression, and dementia at the primary care level – additional results of a questionnaire survey for the early diagnosis of PD
A. Metz, E. Baum, I. Rissling, G. Hoeglinger, V. Ries, W.H. Oertel

P613 Personality and behavior changes after subthalamic nucleus deep brain stimulation in Parkinson's disease (STN-DBS): A retrospective study
A. Gronchi-Perrin, S. Viollier, J.A. Ghika, P.R. Burkhard, J.-G. Villemure, F.J. Vingerhoets

P614 Apomorphine subcutaneous injection in a patient who has undergone surgical treatment of Parkinson's disease with deep brain stimulation
D.M. Swope

P615 Combining dopamine agonists of different dopamine receptor profiles in advanced Parkinson's disease
F. Sixel-Doering, M. Rausch-Hertel, H. Klinke, C. Trenkwalder

P616 Gender and the Parkinson's disease phenotype
Y. Baba, J.D. Putzke, A.J. Strongosky, M.F. Turk, Z.K. Wszolek, R.J. Uitti

P617 Intraoperative tapping test for clinical monitoring during deep brain stimulation surgery in Parkinson's disease
A. Pesenti, V. Chiesa, F. Tamma, E. Caputo, G. Ardolino, P. Rampini

P618 Asymmetry of overactivity between left and right subthalamic nucleus parallels severity of extremity symptoms in Parkinson's disease patients
C.C. Kao, D.P. Charles, T.L. Davis, J.Y. Fang, J.R. Albea, P.E. Konrad

P619 Alzheimer disease (AD) pathology in patients clinically and pathologically diagnosed with Parkinson disease (PD). Are the symptoms different?
S. Papapetropoulos, J. Gonzalez, A. Lieberman, D.C. Mash

P620 Subthalamic nucleus deep brain stimulation (STN DBS) in idiopathic Parkinson's disease (IPD): Predictive value of intra-operative (IO) improvement of motor function to long-term outcome
H. Bronte-Stewart, R. Rajan Das, G. Fujikami, M. Urbano, M. Koop, G. Heit

P621 How long after the onset of motor symptoms does dementia in Parkinson's disease arise?
E.M. Dunn, N.L. Read, T.A. Hughes, R.H. Mindham, E.G. Spokes

P622 Can we measure the effectiveness of a Parkinson's disease (PD) club at improving PD care?
A. Nasar, P. Dyer, C. Short, A.-M. Hunter, G. Greenwood, L. Wright

P623 Proteasomal inhibition leads to nigral degeneration with ubiquitin positive inclusions in mice
T. Hatano, N. Hattori, Y.C. Kawamura, Y. Imamichi, H. Kaneko, Y. Mizuno

P624 "Off" time reduction from adjunctive use of istradefylline (KW-6002) in levodopa-treated patients with advanced Parkinson's disease
P.A. LeWitt, US-005/US-006 Clinical Investigator Group

P625 *Nocardia asteroides*: A possible environmental cause of Parkinson's disease?
P.A. LeWitt, B.L. Beaman, D.M. Camp, D.A. Loeffler

P626 Risk factors for vascular disorders are reduced in Parkinson's disease: A case-control study on 483 consecutive patients
G. Scigliano, M. Musicco, P. Soliveri, I. Piccolo, F. Girotti

P627 Plasma brain natriuretic peptide (BNP) levels in Parkinson's disease
Y.K. Nakao, A. Satoh, K. Iwanaga, I. Tomita, M. Seto, M. Tsujihata

P628 Sensory dysfunction in idiopathic Parkinson's disease
H.-W. Shin, Y.H. Sohn, S.Y. Kang

P629 Patient home diary: A reproducible and reliable tool to assess motor complications in Parkinson's disease
M. Faighel, J.-M. NGuyen, P. Damier

P630 The side effect profile of cabergoline, an ergot agonist; a clinical follow up study in Parkinson's disease and restless legs syndrome
F. Stegie, P. Metcalf, V. Dhawan, A. Williams, A. Forbes, K.R. Chaudhuri

P631 Safinamide add-on to levo dopa and dopamine agonist treatment in Parkinson disease. An open escalating dose study
F. Stocchi, L. Vacca, P. Grassini, G. Battaglia, M. Altibrandi, S.A. Ruggieri

P632 Neuroprotective role of nicotine, cigarette smoke and cigarette extracts against MPTP-induced neutoxicity
A. Hild, V. Marchand, B. Dumery, E.C. Hirsch

P633 Internalization of D1 but not D2 dopamine receptors after l-dopa treatment and absence of internalisation after ropinirole treatment
M.-P. Muriel, G. Orieux, E.C. Hirsch

P634 Neurosurgery for Parkinson's disease at an early stage: Impact on quality of life and social adjustment (preliminary results at 12-months)
D. Maltête, J.-L. Houeto, M. Schüpbach, M.-L. Welter, P. Cornu, Y. Agid

P635 Genetics of α-synucleinopathies
M. Farrer, P. Pals, S. Lincoln, D. Dickson, A. Hope, C. Van Broeckhoven

P636 Articulatory kinematics and speech dysfunction in patients with Parkinson's disease
S.A. Venkatesan, V.V. Venkatachalam, L.R. Ranganathan, Y.S. Subramanian

P637 Biochemical analysis of missense mutations in ceruloplasmin (CP) in Parkinson's disease (PD)
H. Hochstrasser, U. Walter, J. Spiegel, S. Behnke, I. Csoti, P. Bauer

P638 Neuroprotection of dopaminergic neurons by electroconvulsive shock in an animal model of Parkinson's disease
G.A. de Erausquin, A. Anastasia, R. Reynoso, H. López Morra, D.H. Mascó

P639 Consumption of milk and calcium in mid-life and the future risk of Parkinson's disease
G.W. Ross, M. Park, H. Petrovitch, L.R. White, C.M. Tanner, R.D. Abbott

P640 Fas and Bcl-2 expression in T leucocytes of patients with Parkinson's disease
S. Bostantjopoulou, Z. Katsarou, O. Hatzizisi, G. Kyriazis

P641 Systematic RNAi screening for effectors of α-synuclein folding and degradation in *C. elegans*
K.A. Caldwell, S.B. Fulghum, S. Cao, G.A. Caldwell

P642 Systemic exposure to proteasome inhibitors causes progressive parkinsonism in rats
K.S. McNaught, C.W. Olanow

P643 Efficacy and safety of α-dihydroergocryptine in the treatment of early and advanced Parkinson's disease: An open-label study
R. Peng, D. Zhou, X.H. Lai, G.G. Yuan

P644 Depression in a group of Puerto Rican patients with Parkinson's disease
I.L. Pita, C. Serrano, V. Wojna

P645 Analysis of dynamics of gait in Parkinson's disease
J.S. Yoon, S.B. Koh, S.H. Lee, K.W. Park, D.H. Lee

P646 Cardiac sympathetic nerve denervation might occur in the early stage of Parkinson's disease
S. Orimo, T. Amino, T. Kojo, A. Takahashi, T. Uchihara, K. Wakabayashi, H. Takahashi

P647 Preclinical prediction of the best contact in STN stimulated PD patients
A.L. Benabid, B.A. Wallace, S. Chabardes, A. Batir, V. Fraix, P. Pollak

P648 Electrophysiological evaluation of autonomic function in Parkinson disease
M. Umaiorubahan, V.C. Uthamarayan

P649 Glucocerebrosidase gene mutations in Ashkenazi Jewish patients with Parkinson disease
J. Aharon-Peretz, H. Rosenbaum, R. Gershoni-Baruch

P650 Analysis of gait pattern in Parkinson's disease: Relationship to clinical features
S.B. Koh, J.S. Yoon, S.H. Lee, K.W. Park, D.H. Lee

P651 Usefulness of statistical image analysis (SPM/eZIS) based on brain perfusion SPECT in patients with Parkinson's disease–relation with excessive daytime sleepiness–
N. Sasaki, H. Watanabe, F. Maki, H. Sugihara, M. Kawakami, Y. Takahashi

P652 Effects of STN deep brain stimulation on gait stability in adanced Parkinson's disease: Disparity between UPDRS scores and gait stability
J.M. Hausdorff, D. Tarsy, L. Scollins, S. O'Herron, J. Solomont, L. Gruendlinger

P653 Prevalence of Parkinson's disease in Central Russia based on population survey
O.S. Levin, M.A. Lobov, L.V. Dokadina, V.N. Shtok

P654 Gait disorder and cognitive impairment in patients with Parkinson's disease
N.A. Unizshenko, O.S. Levin, D.Y. Olyunin

P655 Emotion and decision making in Parkinson's disease: Effects of levodopa and subthalamic nucleus (STN) stimulation
I. Benatru, N. Camille, S. Thobois, P. Mertens, A. Sirigu, E. Broussolle

P656 Neuropsychological study in patients with Parkinson's disease and long-term exposure to hydrocarbon-solvents
D. De Gaspari, M. Canesi, C. Siri, G. Pezzoli

P657 Levodopa associated homocysteine increase and sural axonal neurodegeneration
T. Muller, K. Renger, W. Kuhn

P658 Family caregivers of Parkinson's disease patients deserve professionals attention and support: A report from one year of experience with caregivers' clinic
O. Moore, N. Giladi

P659 Predictors of long-term efficacy pramipexole therapy in patients with Parkinson's disease
I.G. Smolentseva, O.S. Levin, B. Tserensodnom, N.V. Fedorova, L.V. Dokadina

P660 Dissociation of emotional and voluntary facial movements in Parkinson's disease
H. Topka, C. Droll, D. Wildgruber, J. Dichgans

P661 Statistical issues in analysis of a large simple neuroprotection trial (LST) in Parkinson's disease (PD)
B.C. Tilley, Y. Palesch, P. Huang, K. Kieburtz, B. Ravina, C.G. Goetz, C. Kamp, J. Elm, P. Guimaraes, K. Shannon, F. Wooten, C. Tanner

P662 The zona incerta may be a better target than the subthalamic nucleus for deep brain stimulation for Parkinson's disease
P. Plaha, Y.B. Shlomo, N.K. Patel, D.O. Brien, K.O. Sullivan, S.S. Gill

P663 Dyskinesias induced by subthalamotomy unresponsive to NMDA inhibitor amantadine
M. Merello

P664 High frequency STN stimulation activates associative projection cortices of the basal ganglia and improves memory function in patients with advanced Parkinson's disease
E. Kalbe, R. Hilker, L. Burghaus, K. Herholz, J. Kessler, V. Sturm

P665 Homocysteine levels and MTHFR C67T genotype in patients with Parkinson's disease with and without levodopa therapy
E. Dzoljic, I. Novakovic, D. Mirkovic, Z. Todorovic, M. Prostran, V. Kostic

P666 History of obsessive compulsive behavior, younger age of symptoms onset and treatment with dopamine agonists are risk factors for the development of addiction-like behavior in patients with Parkinson's disease
N. Giladi, N. Weitzman, C. Peretz, H. Shabtai, S. Schreiber

P667 Plasma concentrations of fluoxetine and motor signs in patients with Parkinson's disease
E. Dzoljic, I. Kovacevic, M. Pokrajac, M. Mijajlovic, M. Milosevic, V. Kostic

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P668 Kinematical evaluation of movement after partial and total interruption of basal ganglion output in Parkinson's disease
M. Merello, J. Balej, C. Aveleyra, R. Leiguarda

P669 Quality of life (QoL) in patients with Parkinson's disease (PD)
A.B. Guekht, E.S. Chikina, K.S. Glushkov, A.A. Shpak, E.I. Gusev

P670 A simple method to assess oxidative stress in subjects with Parkinson's disease
R. Marconi, S. Carapelli, L. Morgante, A. Epifanio, N. Vanacore, G. Meco

P671 L-DOPA effects on laryngeal dysfunction in Parkinson's disease: An acoustic and aerodynamic study
F. Viallet, L. Jankowski, A. Purson, B. Teston

P672 Neoplastic and preneoplastic skin lesions in Parkinson's disease: A cross-sectional clinical survey in PD patients and age-matched controls
J.J. Ferreira, J.M. Silva, R. Freire, J. Pignatelli, L.C. Guedes, A. Feijó

P673 Validity of a modified Parkinson's disease screening questionnaire in India: Effects of literacy of participants and medical training of screeners and implications for screening efforts in developing countries
R. Rattihalli, N. Sarangmath, G. Gopalkrishna, S.K. Doddaballapur, E.D. Louis, U.B. Muthane

P674 Somnolence and sleep attacks in a small sample of Parkinson's disease patients
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P675 The placebo effect in Parkinson's disease patients: Is this an acute effect?
F. Fregni, F. Maia, P.S. Boggio, F. Bermpohl, A. Pascual-Leone, E.R. Barbosa

P676 Comparison of the effects of a self-supervised home exercise program and a physiotherapist-supervised exercise program on gait parameters in a Parkinson's disease population
V. Lun, N. Pullan, C. Adams, B. Ramage, J. Ronsky, O. Suchowersky

P677 Bladder dysfunction in patients with Parkinson's disease
M. Kiljako, P. Taba, U. Krikmann, E. Olt

P678 Gender differences in Parkinson's disease
C.A. Haaxma, M.W. Horstink

P679 Amyloid peptides and Tau proteins in cerebrospinal fluid of Parkinson patients
S. Haegele, I. Zerr, T. Vogt

P680 Repetitive TMS is as effective as fluoxetine in the treatment of depression in Parkinson's disease patients
F. Fregni, C.M. Santos, M.A. Marcolin, A. Pascual-Leone, L. Silveira-Moriyama, E.R. Barbosa

P681 The relationship between two measures of postural stability: Pull test and functional reach in subjects with and without Parkinson's disease
C. Marras, M.J. Korell, F. Kamel, J.A. Hoppin, D.M. Umbach, C.M. Tanner

P682 Depression, anxiety and cognitive disorders in patients with Parkinson's disease
O. Ozturk, F. Ozer, L. Hanoglu, H. Meral

P683 Learned irrelevance revisited: The cognitive basis of attentional set-shifting impairments in Parkinson's disease
A.E. Slabosz, S.J. Lewis, A.M. Owen

P684 Rapid-rate repetitive transcranial magnetic stimulation and cognitive function in Parkinson's disease patients - A safety study
F. Fregni, P.S. Boggio, M.T. Silva, M.A. Marcolin, A. Pascual-Leone, E.R. Barbosa

P685 Quantitative digitography scores correlate with unified Parkinsons disease rating scale motor scores and are improved by both medication and bilateral subthalamic deep brain stimulation
A. Taylor Tavares, G.S. Jefferis, G. Fujikami, T. Courtney, B. Hill, H. Bronte-Stewart

P686 Repetitive transcranial magnetic stimulation: May it be a potential treatment for speech dysfunction in Parkinson's disease patients?
A.E. Dias, F. Fregni, S.P. Rigonatti, A. Pascual-Leone, M.A. Marcolin, E.R. Barbosa

P687 Protective role of heat shock protein70 during lactacystin-induced cell death both in rat and PC12 cells
T.-B. Ahn, B.S. Jeon

P688 NEMO binding domain inhibits the formation of cytoplasmic inclusions: Phosphorylted IkBa as its target protein is a novel component of Lewy bodies
K. Noda, T. Kitami, W.P. Gai, P.H. Jensen, N. Hattori, Y. Mizuno

P689 Analysis of progenitor cells in the striatum and midbrain by MPTP administration
H. Ohizumi, H. Mochizuki, M. Yamaguchi, Y. Mizuno

P690 Parkinsonism and Parkinson's disease in the elderly: A community-based survey in Brazil
M.T. Barbosa, F. Cardoso, P. Caramelli, D.P. Maia, M.C. Cunningham, M.F. Lima e Costa

P691 Gene therapy for Parkinson's disease by rAAV-parkin
H. Mochizuki, M. Yamada, Y. Mizuno

P692 Pathologic study on the relationship between Parkinson disease with dementia and dementia with Lewy bodies
H. Mori, L. Guo, M. Takanashi, M. Itaya, Y. Mizuno

P693 A comparison of demented patients with pathologically confirmed Parkinson disease (PD) and Alzheimer disease (AD) pathology and non-demented patients also with confirmed Parkinson disease and Alzheimer disease
S. Papapetropoulos, J. Gonzalez, A. Lieberman, D.C. Mash

P694 Rimantadine as an alternative to amantadine in Parkinson patients with severe leg edema secondary to amantadine: Case reports
C. Singer, S. Papapetropoulos, M.A. Gonzalez, A. Lieberman, E. Roberts

Prescribing Information DaTSCAN™ ioflupane(^{123}I)
Refer to full SPC before prescribing **Presentation:** Vials containing 185 MBq or 370 MBq ioflupane (123I) at reference time. **Uses:** Detecting loss of functional dopaminergic neuron terminals in the striatum of patients with clinically uncertain Parkinsonian Syndromes in order to help differentiate Essential Tremor from Parkinsonian Syndromes related to idiopathic Parkinson's Disease (PD), Multiple System Atrophy (MSA), Progressive Supranuclear Palsy (PSP). DaTSCAN is unable to discriminate between PD, MSA and PSP. **Dosage and Administration:** DaTSCAN is a 5% (v/v) ethanolic solution for intravenous injection and should be used without dilution. Clinical efficiency has been demonstrated across the range of 111-185 MBq; do not use outside this range. Appropriate thyroid blocking treatment must be given prior to and post injection of DaTSCAN. SPECT imaging should take place 3-6 hours after injection of DaTSCAN. DaTSCAN is not recommended for use in children or adolescents. For use in patients referred by physicians experienced in the management of movement disorders. To minimise the potential for pain at the injection site during administration, a slow intravenous injection (not less than 15 - 20 seconds) via an arm vein is recommended. See SPC. **Contraindications:** Pregnancy and in patients with hypersensitivity to iodide or any of the excipients. **Precautions:** Radiopharmaceuticals should only be used by qualified personnel with appropriate government authorisation and should be prepared using aseptic and radiological precautions. DaTSCAN is not recommended in moderate to severe renal or hepatic impairment. **Interactions:** Consider current medication. Medicines that bind to the dopamine transporter may interfere with diagnosis; these include amphetamine, benzotropine, buproprion, cocaine, mazindol, methylphenidate, phentermine and sertraline. Drugs shown during clinical trials not to interfere with DaTSCAN imaging include amantadine, benzhexol, budipine, levodopa, metoprolol, primidone, propranolol and selegiline. Dopamine agonists and antagonists acting on the postsynaptic dopamine receptors are not expected to interfere with DaTSCAN imaging and can therefore be continued if desired. **Pregnancy and Lactation:** Contraindicated in pregnancy. Information should be sought about pregnancy from women of child bearing potential. A woman who has missed her period should be assumed to be pregnant. If administration to a breast feeding woman is necessary, substitute formula feeding for breast feeding. **Side Effects:** No serious adverse effects have been reported. Common side effects include headache, vertigo and increased appetite and formication. Exposure to ionising radiation is linked with cancer induction and a potential for hereditary defects and must be kept as low as reasonably achievable. Intense pain on injection has been reported uncommonly following administration into small veins. **Dosimetry:** Effective dose from 185 MBq is 4.35 mSv. **Overdose:** Encourage frequent micturition and defecation. **Legal category:** Subject to medical prescription (POM). Consult full SPC before prescribing. Further information available on request. **Marketing Authorisation numbers:** EU/1/00/135/001 and EU/1/00/135/002. **Date of Preparation:** July 2003. Amersham, Amersham Health and DaTSCAN are trademarks of Amersham plc. Amersham plc, Amersham Place, Little Chalfont, Buckinghamshire, England HP7 9NA.
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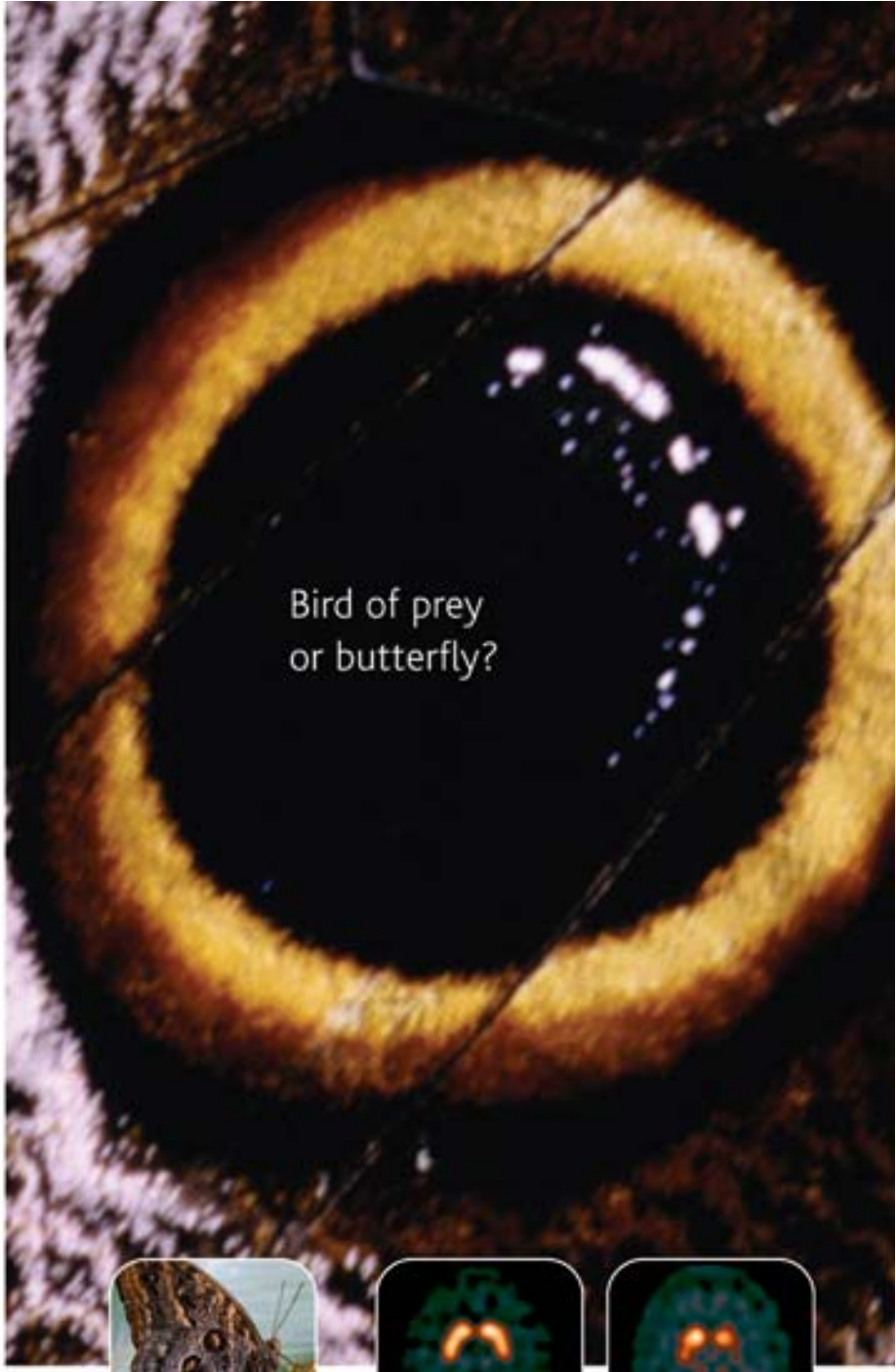

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POSTER SESSION 3

WEDNESDAY, JUNE 16

Poster Viewing: 8:30 am to 5:00 pm
Authors Present Odd Numbers: 11:30 am to 12:30 pm
Authors Present Even Numbers: 4:00 pm to 5:00 pm

Parkinson's disease 2

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P695 A registry-based, case control investigation of Parkinson's disease with and without cognitive impairment
S. Papapetropoulos, J. Ellul, P. Polychronopoulos, E. Chroni, T. Papapetropoulos

P696 Compliance among clinical trial participants in Parkinson's disease: Can it be predicted?
J.K. Dobson, R.L. Rodnitzky, E.Y. Uc

P697 Unilateral orthostatic tremor as an initial manifestation of Parkinson's disease and a possible predictor for good prognosis
S.Y. Kang, J.-S. Kim, Y.H. Sohn

P698 Examination of valosin containing protein (VCP) in Parkinson's disease (PD)
M. Iijima, T. Kitami, S. Hori, A. Kakizuka, N. Hattori, Y. Mizuno

P699 Analysis of cerebrospinal fluid in dementia with Lewy Bodies and Parkinson's disease dementia
B. Mollenhauer, C. Trenkwalder, F. Sixel-Doering, M. Bibl, M. Canelo, J. Schindehuette

P700 Modulation of GABAergic interneuronal activity in the motor cortex of parkinsonian rats
C. Capper-Loup, J.-M. Burgunder, A. Kaelin-Lang

P701 Homocystein serum levels and MTHFR C67T genotype in patients with Parkinson's disease, with and without levodopa therapy
E. Dzoljic, I. Novakovic, D. Mirkovic, Z. Todorovic, M. Prostran, V. Kostic

P702 Longtem ambulatory gait monitoring in Parkinson's disease: Validation of a new wireless measurement system
H. Russmann, A. Salarian, K. Aminian, J. Villemure, P.R. Burkhard, F.J. Vingerhoets

P703 Intranigral blockade of nociceptin/orphanin FQ transmission attenuates haloperidol-induced catalepsy in rats
M. Marti, F. Mela, F. Martina, G. Remo, B. Clementina, M. Michele

P704 What are factors associated with dementia in Parkinson's disease?
T. Sato

P705 Acute Akinesia in Parkinson's disease: A concealed syndrome revisited
M. Onofrj, A. Thomas, D. Iacono, A. Luciano, K. Armellino

P706 Suicidal thoughts in patients with Parkinson's disease treated by deep brain stimulation of the subthalamic nuclei: Our experience and meta-analysis of the literature
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P707 COX-2 is a transcriptional target gene of JNK and induces neurodegeneration in the 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine mouse model of Parkinson disease
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P999 PSP look alike in a patient with a dorsorostral midbrain lesion sparing dopaminergic nigrostriatal projection - Is axial rigidity independent of dopamine deficiency?
J. Lewerenz, B. Zurowski, A. Munchau

P1000 Corticobasal degeneration-like presentation with SCA8 mutation
Y. Baba, Z.K. Wszolek, M. Farrer, R.J. Uitti

P1001 Putaminal atrophy in multiple system atrophy
H.Y. Shin, Y.H. Sohn, J.H. Yang, H.S. Kim, M.S. Lee, J.-S. Kim

P1002 Dopa-responsive hemiparkinsonism due to midbrain Virchow-Robin spaces
M. Krause, S. Hähnel, H.-M. Meinck

P1003 The effects of repetitive transcranial magnetic stimulation (rTMS) on frozen gait in the patients with parkinsonism
M. Tamaki, Y. Sawada, Y. Ichikawa, K. Arasaki, K. Sudo

P1004 Comparison of DWI and [123-I]-IBZM-SPECT for the differentiation of patients with the Parkinson variant of multiple system atrophy from PD
A. Diem, K. Seppi, M.F. Schocke, E. Donnemiller, G.K. Wenning, W. Poewe

P1005 Dopa-responsive hemiparkinsonism secondary to a contralateral ischemic lesion of the nigrostriatal dopaminergic pathway
G. Fénelon, C. Guidoux, E. Itti, P. Remy, P. Cesaro

P1006 A patient with PSP-like brain and spinal cord NFT-tau pathology presenting as young onset spastic paraplegia
T. Scaravilli, S. Papapetropoulos, H. Morris, N.P. Quinn, F. Scaravilli, K.P. Bhatia

P1007 Early presentation of gait disorder is indicative of non –Alzheimer's dementia
L.M. Allan, D.J. Burn, C.G. Ballard, R. Kenny

P1008 Psychogenic parkinsonism: Clinical features of a large case series
J.C. Morgan, P. Mir, R.K. Mahapatra, K.P. Bhatia, K.D. Sethi

POSTER SESSION 3

P1009 New insights in the environmental origins of neurodegenerative disorders: Effects of prenatal stress on the sensitivity to post-natal exposure to pesticides in the rat
C.C. Vanbesien-Mailliot, M.-C. Chartier-Harlin, O. Viltart, M.-L. Caillet-Boudin, A. Pierce, D. Vieau

P1010 Dysregulation of chaperone proteins in Dementia with Lewy Bodies
I. Cantuti-Castelvetri, M. Ingelsson, J. Klucken, K. Ramasamy, B.T. Hyman, D.G. Standaert

P1011 Warning signs ('red flags') in multiple system atrophy (MSA): A preliminary cross-sectional analysis of 79 European MSA-P patients
F. Geser, M. Stampfer-Kountchev, K. Seppi, J.-P. Ndayisaba, G. Wenning, W. Poewe, on behalf of the European MSA-Study Group (EMSA-SG)

P1012 The European MSA-Study Group (EMSA-SG) natural history study of multiple system atrophy (MSA) – an analysis of baseline data
F. Geser, M. Stampfer-Kountchev, K. Seppi, J.-P. Ndayisaba, W. Poewe, G.K. Wenning, on behalf of the European MSA-Study Group (EMSA-SG)

P1013 The clinical presentation of multiple system atrophy (MSA) in Europe: An interim analysis of the EMSA-SG (European MSA-Study Group) Registry
F. Geser, M. Stampfer-Kountchev, K. Seppi, J.-P. Ndayisaba, G. Wenning, W. Poewe, on behalf of the European MSA-Study Group (EMSA-SG)

P1014 Dopa-responsive parkinsonism after acute subdural hematoma
A. Maertens de Noordhout, F. Daenen, V. Bex

P1015 Profile and severity of parkinsonian features in atypical parkinsonian disorders (APD)
G. Meco, N. Vanacore, V. Bonifati, U. Bonuccelli, G. De Michele, M. De Mari

P1016 Progressive Supranuclear Palsy in the Netherlands
L. Donker Kaat, A. Boon, P. Heutink, J.v. Swieten


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POSTER SESSION 4

THURSDAY, JUNE 17

Poster Viewing: 8:30 am to 4:30 pm
Authors Present Odd Numbers: 12:00 pm to 1:00 pm
Authors Present Even Numbers: 1:00 pm to 2:00 pm

Genetics

Poster numbers 1017-1072

P1017 Association of vitamin D receptor gene polymorphism and Parkinson's disease in Koreans
J.-S. Kim, C. Song, I. Yoon, Y.-I. Kim, S.-B. Ko, K.-S. Lee

P1018 A CD14 monocyte receptor polymorphism and genetic susceptibility to Parkinson's disease
J.-J. Lin, K.-C. Yueh

P1019 Parkin mutations in early-onset Parkinson's disease among South Indians
A. Kishore, R. H.M., A. Anand

P1020 Allele 2 of the Ser9Gly polymorphism in the dopamine D3 receptor gene is responsible of the most common familial form of the essential tremor desease
G. Lucotte, B. Funalot, P. Sokoloff

P1021 Association of a dopamine D2 receptor gene polymorphism with Parkinson disease
M. Ezquerra, J. Campdelacreu, E. Munnoz, M. Marti, F. Valldoriola, E. Tolosa

P1022 A new mutation in the tau gene in a family with PSP-like syndrome and Parkinson's disease
E. Gasparoli, G. Rossi, T. Scaravilli, A. Albanese, F. Tagliavini, F. Bracco

P1023 A patient with dopa-responsive dystonia and juvenile Parkinson's disease
L.E. Hjermind, L.G. Johannsen, R.A. Wevers, N. Blau, L. Friberg, L. Regeur

P1024 Homozygous deletion of the exon 4 in the *Parkin* in a Brazilian and Turkish families due to independent events
J. Clarimon, A. Singleton, J. Johnson, N.W. Wood, O. Dugu, A.J. Lees

P1025 MDR1 haplotype analysis in Parkinson's disease
E. Tan, K. Tang, C. Tan, Y. Zhao, S. Chong, C. Lee

P1026 Motor behaviour of parkin knock out mice
A. Serrano, E. Gallego, M. Mena, M. Sanchez, J. Benavides, J. Garcia-de-Yebenes

P1027 The new mutation, E46K, of -synuclein causes Parkinson, insomnia and Lewy body dementia
J. Alegre, J.J. Zarranz, J. Hoenicka, J.C. Gomez Esteban, J. G de Yebenes, E. Lezcano

P1028 Analysis of CRHR1 and NSF genes in progressive supranuclear palsy
J. Campdelacreu, M. Ezquerra, E. Muñoz, M.J. Marti, F. Valldeoriola, E. Tolosa

P1029 The welfare of research participants in the Prospective Huntington at risk observational study (PHAROS)
E.P. Kayson, Huntington Study Group/PHAROS Investigators

P1030 A genomic screen for a novel essential tremor locus
A.E. Ashley-Koch, L. Zhang, J.M. Stajich, S. West, M.A. Pericak-Vance, J.R. Gilbert

P1031 Screening of a large cohort of patients with atypical parkinsonism for repeat expansions in the FMR1 gene
C. Kamm, K. Buerk, T. Illig, E. Wichmann, European Multiple System Atrophy (EMSA) Study Group, T. Gasser

P1032 Combined effect of interleukin-6 and estrogen receptor beta gene polymorphisms on the risk for Parkinson's disease
A. Håkansson, L. Westberg, H. Niazi Shahabi, S. Nilsson, E. Eriksson, H. Nissbrandt

P1033 alpha-synuclein locus duplication causes familial Parkinson's disease
M.-C. Chartier-Harlin, J. Kachergus, C. Roumier, P. Amouyel, M. Farrer, A. Destee

P1034 PET and neuropsychological features in a case of spinocerebellar ataxia type 17(SCA17)
J.E. Nielsen, T. Petersen, A. Noerremoelle, A. Gjedde, L. Ehlers, L. Hasholt

P1035 Prenatal diagnosis of autosomal dominant hereditary spastic paraplegia (SPG4) using direct mutation detection
J.E. Nielsen, P. Koefoed, S. Kjaergaard, L. Neerup-Jensen, A. Noerremoelle, L. Hasholt

P1036 The Val66Met polymorphism of the brain derived neurotrophic factor (BDNF) - A shared genetic risk factor for obsessive-compulsive behaviour and Gilles de la Tourette syndrome?
S. Klaffke, I. Koenig, A. Ziegler, J. Hebebrand, O. Bandmann

P1037 Restless legs syndrome (RLS) in a large family (Family LA) with *Parkin*-associated Parkinson's disease (PD)
S. Maniak, K. Kabakci, I. Pichler, P.L. Kramer, P.P. Pramstaller, C. Klein

P1038 PARK11 is not linked with Parkinson's disease in European families
J. Prestel, M. Sharma, A. Zimprich, B. Mueller-Myhsok, T. Gasser, the European Consortium of Genetics in PD (GSPD)

P1039 Definition of the tau gene haplotype block that is associated with progressive supranuclear palsy
R. de Silva, A.M. Pittman, A.J. Myers, N.W. Wood, J. Hardy, A.J. Lees

P1040 An English family with a dominantly inherited PSP-like illness, genetic anticipation and cortical blindness
D.J. Nicholl, S. Nightingale, C. Hawkins, P. Heutink

P1041 Genetic polymorphisms of the dopamine transporter gene and hallucinations in Parkinson's disease
J.G. Goldman, C.G. Goetz, E. Berry-Kravis, S.E. Leurgans, C. DeSai, L. Zhou

P1042 Identification of PARK6, a novel mitochondrial protein causing Parkinson's disease
E. Valente, P.M. Abou-Sleiman, V. Caputo, M.M. Muqit, G. Auburger, A. Bentivoglio

P1043 Clinical and molecular genetic analysis of hereditary dopa-responsive syndromes in Serbian population
G. Djuric, P.A. Slominsky, M. Svetel, S.N. Illarioshkin, V. Kostic, E.D. Markova

P1044 Genome-wide microsatellite association studies for sporadic Parkinson's disease by using the pooled DNA method
T. Toda, W. Satake, I. Mizuta, M. Yamamoto, N. Hattori, M. Murata

P1045 A novel mutation causing exon skipping within the epsilon-sarcoglycan gene causes myoclonus-dystonia syndrome with prominent psychiatric features
A. Misbahuddin, M.R. Placzek, G. Lennox, T.T. Warner

P1046 Clinical and genetic study of parkin disease in a large sample set
A.M. Bertoli-Avella, B.A. Oostra, G. Meco, P. Heutink, V. Bonifati, the Italian Parkinson Genetics Network

P1047 Mutational analysis of divalent metal transporter 1 in restless legs syndrome
J. Winkelmann, P. Lichtner, D.P. Auer, A. Pastore, T.M. Strom, T. Meitinger

P1048 Evidence for further genetic locus heterogeneity in restless legs syndrome
J. Winkelmann, P. Lichtner, C. Trenkwalder, T. Meitinger, T.M. Strom, B. Muller-Myhsok

P1049 Effect of l-dopa and subthalamic surgery on proprioception in Parkinson's disease patients
M. Merello, J. Balej, C. Aveleyra, R. Leiguarda

P1050 Prevalence of UCHL1, DJ1 and NR4A2 gene mutations in young onset PD (YOPD) patients
I. Rissling, O. Bandmann, C. Hoft, R. Burmester, W.H. Oertel, C. Moller

P1051 A subject with a homozygous exon 4 parkin deletion whose parkinsonism dramatically improves following smoking
O. Dogu, A. Crawley, J. Werner, K. Gwinn-Hardy, G. Lopez

P1052 Parkin mutations in autosomal recessive early-onset parkinsonism (AR-EP)
N. Hattori, H. Yoshino, Y. Imamichi, Y. Miuzno

P1053 Adult onset hereditary dystonia
K.P. Frei, M. Pathak, P. Pham, D.D. Truong

P1054 The role for Fragile X premutation in essential tremor and spinocerebellar ataxia: Findings from two cohorts of Italian patients
E. Di Maria, M. Grasso, G. Abbruzzese, P. Mandich, S. Ratto, R. Sciolla

P1055 PARK6-linked autosomal recessive early-onset parkinsonism in European and Asian populations
Y. Hatano, T. Shimazaki, K. Sato, V. Bonifati, N. Hattori, Y. Mizuno

P1056 Chromosome 1 association map to identify gene(s) associated with age-at-onset and risk for Parkinson disease
S.A. Oliveira, L. Yi-Ju, Q. Xuejun, P.-V.A. Margaret, V.M. Jeffery

P1057 Familial clustering of restless legs syndrome in a population isolate in South Tyrol (Northern Italy)
I. Pichler, S. Maniak, F.D. Vogl, G. Casari, K. Christine, P.P. Pramstaller

P1058 Molecular study of Park2 gene in 266 patients affected by early-onset Parkinson disease
D. Ghezzi, F. Invernizzi, E. Marelli, M. Zeviani, B. Garavaglia

P1059 Fronto-temporal dementia syndrome with parkinsonism in a Colombian family with E280A presenilin-1 mutation
N. Duarte, J. Lozano, M. Pena, W. Fernandez, G. Arboleda, H. Arboleda

P1060 Analysis of polymorphisms in APOE, ACE, alpha-synuclein and Tau genes and screening of mutations in the Parkin gene in Parkinson's disease in Colombia
B. Benitez, D. Forero, C. Alvarez, G. Arango, W. Fernandez, H. Arboleda

P1061 Clinical and genealogical study of a large Brazilian family with early-onset Parkinson's disease
H. Chien, M. Costa, V. Bonifati, E. Barbosa

P1062 Analysis of polyglutamine-coding repeats in the TATA-binding protein in different neurodegenerative diseases
Y.-R. Wu, C.-M. Chen, G.-J. Lee-Chen, H.-C. Fung

P1063 Mapping a gene for Parkinson's disease in Norway
M. Toft, L. Skipper, M. Hulihan, J. Aasly, M. Farrer

P1064 A consanguineous Turkish family with early-onset Parkinson's disease and an exon 4 *Parkin* deletion
O. Dogu, J. Johnson, D.G. Hernandez, M. Hanson, J. Hardy, H. Apaydin

P1065 Blinded diagnoses confirm elevated frequency in relatives of restless legs syndrome (RLS) probands in a case-control family study of RLS
W.A. Hening, W. Mystinna, A.P. Richard, L. Suzanne, E.J. Earley

P1066 The role of the epsilon-sarcoglycan gene (SGCE) in Gilles de la Tourette patients
F. Asmus, S. Schoenian, P. Lichtner, B. Mueller-Myhsok, O. Bandmann, T. Gasser

P1067 Identification of candidate genes for Parkinson disease (PD) using genetic linkage and gene expression in the substantia nigra
M.A. Hauser, M. Noureddine, C.M. Hulette, L. Yi-Ju, S. Clemens, J.M. Vance

P1068 Apolipoprotein E gene and Parkinson's disease in Russian population
T.B. Zagorovskaya, I.A. Ivanova-Smolenskaya, S.N. Illarioshkin, E.D. Markova

P1069 Slow acetylation genotype to influence the course of Parkinson's disease?
G. Duda, G. Opala, T. Wilczok, B. Jasinska-Myga, J. Samelska, M. Bialecka

P1070 The contribution of PARK2 to Parkinson's disease, a population genetic based study
P.M. Abou-sleiman, D.G. Healy, K.K. Ahmadi, D.B. Goldstein, N.W. Wood

P1071 A single cycle of iron chelation therapy can stop progression of aceruloplasminemia for a sustained period of time
M. Pandolfo, I. Haemers, S. Goldman

P1072 Glutathione S-transferase P1 and Z1 alleles influence onset age in Parkinson's disease caused by the α-synuclein A53T mutation
L.I. Golbe, G. Di Iorio, K.M. Markopoulou, A. Athanassiadou, S. Papapetropoulos, R.L. Watts

Neuroimaging

Poster numbers 1073-1130

P1073 Reversibilty of D2 dopamine receptor downregulation after dopaminergic treatment withdrawal and subthalamic nucleus stimulation in Parkinson's disease: A [^{11}C]-Raclopride PET study
S. Thobois, P. Pollak, F. Vingerhoets, P. Mertens, A.-L. Benabid, E. Broussolle

P1074 Perfsuion-weighted imaging with Tc-SPECT or perfusion-weighted MRI in combination with a spinal tap test is helpful in the diagnosis of patients with gait disorders caused by normal pressure hydrocephalus
F. Hertel, C. Walter, M. Schmitt, M. Moersdorf, M. Bettag, W. Jammers

P1075 Asymmetric loss of dopamine transporters in tremor-predominant hemi-Parkinson's disease
Y.-M. Shon, J.-S. Kim, Y.-A. Chung, K.-S. Lee

P1076 Striatal dopamine D2 receptor function measured by in vivo [^{123}I]-IBZM pinhole SPECT correlates with ex-vivo quantification of striatal medium-sized spiny neurons
S.W. Scholz, E. Donnemiller, R. Moncayo, W. Poewe, G.K. Wenning, C. Scherfler

P1077 Voxel based analysis of [^{123}I] bð-CIT SPECT distinguishes idiopathic Parkinson's disease from multiple system atrophy
C. Scherfler, K. Seppi, E. Donnemiller, W. Poewe, G.K. Wenning

P1078 Neural correlates of motor timing in Parkinson's disease and the effect of apomorphine studied with PET
M. Jahanshahi, C. Jones, J. Zijlmans, R. Katzenschalger, C. Frith, N. Quinn

P1079 Repetitive involuntary limb movements in patients with brainstem lesions involving pontine tegmentum: Evidence for pontine inhibitory region in human
P. Lee, J. Lee, D. Shin, K. Huh

P1080 Superior Cerebellar Peduncle (SCP) volume measurement on MRI differentiates progressive supranuclear palsy (PSP) from multiple system atrophy (MSA), Parkinson's disease (PD) and controls
D.C. Paviour, S.L. Price, J. Stevens, A.J. Lees, N.C. Fox

P1081 Cerebral glucose metabolism and dopamine transporter PET in SCA
U. Wüllner, M. Reimold, K. Bürk, M. Abele, M. Minnerop, H.-J. Machulla

P1082 Comparison of FP-CIT SPECT scans with F-Dopa PET scans in a similar group of patients with de novo and advanced stages of Parkinson's disease
S.A. Eshuis, P. Jager, R.P. Maguire, K.L. Leenders

P1083 Dopamine transporter imaging (b-CIT-SPECT) in disulfiram-induced striatal damage
F. Tison, F. Macia, E. Bertandeau, M. Guyot, E. Bussy, M. Allard

P1084 Changes in cortical motor activity induced by levodopa administration in cerebellar multiple system atrophy: A positron emission tomography study
C. Brefel-Courbon, P. Payoux, J. Azulay, F. Durif, F. Tison, O. Rascol

P1085 A PET study on the role of sigma-receptors in dopa-induced dyskinesia in patients with advanced Parkinson disease
T. Nimura, T. Ando, K. Yamaguchi, R. Shirane, M. Itoh, T. Tominaga

P1086 Chorea associated with non-ketosic hyperglycaemia and hiperintensity in basal ganglia on T1-weighted MRI. Presentation of a case with bilateral symptoms and images
V. Puente, C. Oliveras, I. Volmer, J. Jimenez, N. Segura

P1087 Brainstem [1] H-MRS in Parkinson patients with and without REM sleep behavioural disorders
L. Hanoglu, H. Meral, F. Ozer, A. Dincer

POSTER SESSION 4

P1088 Brainstem atrophy quantification by magnetic resonance imaging (MRI) is powerful to differentiate idiopathic Parkinson's disease (IPD), multiple system atrophy (MSA) and progressive supranuclear palsy (PSP)
Y. Rolland, B. Bruneau, M. Verin, C. Payan, G. Bensimon, the NNIPPS Study Group

P1089 Progression of early Parkinson's disease, an [^{123}I] FP-CIT study
V.L. Marshall, W.H. Oertel, D.M. Hadley, O. Pogarell, A. Gerstner, D.G. Grosset

P1090 Brain energy metabolism in Huntington's disease
M. Wieler, C. Hanstock, W. Martin

P1091 3-Dimensional MR CISS and TOF MRA evaluation of rostral ventro-lateral medulla compression and hypertension in Hemifacial Spasm
L. Chan, E. Tan, W. Lim

P1092 Magnetic resonance spectroscopy in patients with spinocerebellar ataxia type 2 and type 3
J.-Y. Li, C.-S. Liu, P.-H. Lai, P.-C. Chen

P1093 Diffusion Tensor Imaging demonstrates differential involvement of brain stem structures in Progressive Supranuclear Palsy but not idiopathic Parkinson's disease
C.R. Blain, G.J. Barker, J.M. Jarosz, S.C. Williams, P.N. Leigh

P1094 Regional cerebral blood flow during active music therapy in patients with Parkinson's disease
F. Yoshii, N. Namai, K. Hatakeyama, T. Shimizu, H. Takahashi, Y. Shinohara

P1095 Differences between idiopathic Parkinson's disease and dementia with Lewy bodies detected with [^{11}C]dihydrotetrabenazine and positron emission tomography
S. Gilman, K.A. Frey, R.A. Koeppe, R.L. Albin, L. Junck, M. Heumann

P1096 Positron emission tomographic measurement of acetylcholinesterase activity in frontotemporal dementia and parkinsonism linked to chromosome 17
S. Hirano, H. Shinotoh, T. Kobayashi, Y. Tsuboi, Z.K. Wszolek, A. Aotsuka

P1097 Neuroimaging of neuronal circuits involved in generation of tics in patients with Tourette's syndrome
A. Lerner, E. Boudreau, A. Bagic, T. Hanakawa, D. Murphy, M. Hallett

P1098 Assessment of the nigrostriatal pathway after STN-DBS in advanced Parkinson's disease: A follow-up FDOPA-PET study
A.T. Portman, T. van Laar, R.P. Maguire, M.J. Staal, J. Pruim, K.L. Leenders

P1099 Striatal uptake measured with FDOPA PET and cognition in advanced PD
M. van Beilen, A.T. Portman, R.P. Maguire, J. Pruim, M. Koning, K.L. Leenders

P1100 Comparison of magnetic resonance imaging in subtypes of multiple system atrophy
E. Lee, H. Cho, S. Kim, W. Lee

P1101 Attention to action increases activation of the supplementary motor area in Parkinson's disease
R. Cunnington, L. Carabott, G. Egan, R. Iansek

P1102 Quantifying striatal atrophy in Huntington's disease patients
G. Douaud, M.-J. Ribeiro, R. Maroy, A.-C. Bachoud-Lévi, P. Hantraye, P. Remy

P1103 Differential diagnosis of parkinsonian disorders - The diagnostic value of FDG PET
T. Eckert, A. Barnes, S. Frucht, V. Dhawan, A. Feigin, D. Eidelberg

P1104 Three dimensional streotactic surface projection SPECT analysis in Parkinson' disease
Y. Osaki, Y. Morita, M. Fukumoto, N. Akagi, S. Yoshida, Y. Doi

P1105 VBM as a tool to identify regions which may aid differential diagnosis of PSP, PD and MSA using MRI
S.L. Price, D.C. Paviour, R.I. Scahill, J.M. Stevens, A.J. Lees, N.C. Fox

P1106 Nucleus lentiformis hyperechogenicity correlates with severity of neurological symptoms in Wilson's disease
U. Walter, K. Krolikowski, B. Tarnacka, R. Benecke, A. Czlonkowska, D. Dressler

P1107 Bilateral neurostimulation systems used for deep brain stimulation: *In vitro* study of MRI-related heating at 1.5 tesla and implications for imaging during surgery in Parkinson's disease
R. Bhidayasiri, J.M. Bronstein, S.S. Sinha, S. Ahn, E.J. Benhke, M.S. Cohen

P1108 Reward, recognition and motor response processing – an fMRI study
M. Keitz, R. Maguire, R. Kortekaas, J. den Boer, K. Leenders

P1109 Anterior cingulate, insula and parietal operculum commonly activated during motor and vocal tics in patients with Tourette syndrome: An event-related functional MRI study
S. Bohlhalter, A. Goldfine, S. Matteson, G. Garraux, K. Kansaku, M. Hallett

P1110 Body posture and motor imagery in Parkinson's disease
R.C. Helmich, F.P. De Lange, B.R. Bloem, I. Toni

P1111 Substantia nigra hyperechogenicity in asymptomatic and symptomatic parkin mutation carriers - An early predictor of disease manifestation?
U. Walter, C. Klein, R. Hilker, R. Benecke, P.P. Pramstaller, D. Dressler

P1112 Brain metabolic and clinical response to galantamine in dementia with Lewy bodies (DLB): A preliminary report
D.G. Lichter, K. Marton, E.M. Bednarczyk, L. Wray, D. Wack, L.A. Hershey

P1113 Voxel-based relaxometry (VBR): Application to multiple system atrophy (MSA)
T. Klockgether, M. Minnerop, S. Karsten

P1114 Developing the AMADEUS consortium, a multi-center imaging network for PD: A ^{57}Co striatal phantom to reduce the variance of quantitative ^{123}I dopamine transporter imaging
J.P. Seibyl, I.G. Zubal, P. Barone, W. Oertel, W. Poewe, E. Tolosa

P1115 Huntington's disease and globus pallidus stimulation: A PET study
A.P. Strafella, E. Moro, A.M. Lozano, Y.-Y.W. Poon, A. Dagher, A.E. Lang

P1116 Aging effect on the rate of progression in Parkinson's disease: A four year longitudinal PET study
W. Au, M. Schulzer, V. Sossi, T.J. Ruth, D.B. Calne, A.J. Stoessl

P1117 In vivo detection of neuropathological changes in different parkinsonian syndromes by voxel-based magnetization transfer imaging
T. Peschel, J. Kaufmann, C. Schrader, R. Dengler, H.-J. Heinze, T. Eckert

P1118 Comparison of PET tracers for dopamine terminals in Parkinson's disease: Evidence for aging effects in ^{11}C-MP and regulatory changes in ^{18}F-DOPA
A.R. Troiano, M. Schulzer, V. Sossi, T.J. Ruth, D.B. Calne, A.J. Stoessl

P1119 Subthalamic stimulation in Parkinson's disease modulates motor cortex
B. Haslinger, K. Kalteis, F. Alesch, H. Boecker, A.O. Ceballos-Baumann

P1120 The aging process of dopamine tansporter is faster in younger healthy subjects before 45 years of age
Y.-H. Weng, C.-S. Lu, T.-C. Yen

P1121 Supra- and infratentorial atrophy in the cerebellar variant of multiple system atrophy: A VBM study
C. Brenneis, K. Egger, K. Seppi, M. Schocke, G.K. Wenning, W. Poewe

P1122 Discrimination of multiple system atrophy from Parkinson disease using coronal section of T2* weighted MRI
T. Oikawa, K. Hisanaga, R. Fukatsu, H. Mochizuki, Y. Iwasaki, Y. Itoyama

P1123 Clinical and metabolic brain changes in tremor predominant Parkinson's disease patients treated with Vim DBS
M. Trošt, E.S. Simon, V. Dhawan, J. Okulski, H. Fodstad, D. Eidelberg

P1124 The effect of L-DOPA on the functional anatomy of bimanual movements in Parkinson's disease
E. Kraft, W. Loichinger, M. Diepers, W. Becker, J. Schwarz, A. Storch

P1125 PET imaging reveals profound loss of dopamine transporters in Lesch-Nyhan disease and its variants
H.A. Jinnah, J.C. Harris, D.J. Schretlen, R.J. Torres, J.G. Puig, D.F. Wong

P1126 Are any abnormalities in basal ganglia in fronotemporal dementia? A preliminary study through single photon emission computed tomography (SPECT) with ioflupane (I123-FP-CIT)
J. Papatriantafyllou, N. Sifakis, P. Zikos, V. Kontoyanni, T. Visviki, C. Karageorgiou

P1127 Changes of glucose metabolism in patients with Parkinson's disease with disease duration
T. Eckert, A. Barnes, S. Frucht, V. Dhawan, M.F. Gordon, D. Eidelberg

P1128 Evaluating the diagnostic accuracy of [123-I]β-CIT and SPECT in parkinsonian syndrome
D.L. Jennings, J.P. Seibyl, R. Tabamo, K. Marek

P1129 [123-I]β-CIT and SPECT as a diagnostic tool in lower body parkinsonism
D.L. Jennings, J.P. Seibyl, R. Tabamo, K. Marek

P1130 MRI and deep brain stimulation: Safety-related considerations
K.B. Baker, J.A. Nyenhuis, J. Tkach, A. Sharan, F.G. Shellock, A.R. Rezai

Neuropharmacology

Poster numbers 1131-1166

P1131 Effects of riluzole as "rescue therapy" in a MPTP + 3-NP mouse model of striatonigral degeneration: Experimental rationale for its use in multiple system atrophy
E. Diguet, P.-O. Fernagut, C. Scherfler, G. Wenning, F. Tison

P1132 Dopamine and adenosine receptor interaction as basis for the treatment of Parkinson's disease
M. Morelli, A.R. Carta, P. Annalisa, T. Elisabetta, S. Nicola

P1133 Long term effects of Helicobacter pylori eradication on L-dopa absorption in Parkinson's disease patients
L. Brusa, A. Pietroiusti, M. Pierantozzi, S. Galati, E. Fedele, P. Stanzione

P1134 Deleterious effects of minocycline in animal models of Parkinson's disease and Huntington's disease
E. Diguet, P.-O. Fernagut, X. Wei, Y. Du, E. Bezard, F. Tison

P1135 Entacapone increases and extends striatal dopamine release following L-DOPA/benserazide treatment in the rat
M. Gerlach, M. van den Buuse, C. Blaha, D. Bremen, P. Riederer

P1136 Plasma homocysteine levels in pergolide treated Parkinson's disease patients
S. Ozkan, O. Colak, C. Kutlu, M. Ertan, O. Alatas

P1137 Levodopa raises pain threshold in Parkinson's disease: A clinical and positron emission tomography study
C. Brefel-Courbon, P. Payoux, C. Thalamas, M. Galitzky, J. Montastruc, O. Rascol

P1138 Short and long term effect of low doses of botulinum toxin in 100 Tunisian patients over an 8-year period
N. Gouider-Khouja, G. El Euch, I. Turki, S. Chebel, F. Hentati

P1139 Desipramine increases L-DOPA-derived extracellular dopamine in the striatum of 6-hydroxydopamine-lesioned rats
A. Arai, K. Kannari, H. Shen, M. Baba, M. Matsunaga

P1140 Cabergoline, a dopamine agonist, prevent levodopa-induced abnormal increase of lipid peroxidation mainly due to increase of glutathione content and inhibition of caspase activities in 6-OHDA-lesioned mice
K.-I. Tanaka, N. Ogawa

P1141 Metabotropic glutamate 5 (mGlu5) receptor antagonist-induced locomotion requires adenosine A_{2A} and dopamine D_2 receptors and is potentiated by an A_{2A} antagonist
A. Kachroo, D.K. Grandy, J.-F. Chen, L. Orlando, M.A. Schwarzschild

P1142 The dopamine stabiliser ACR16 prevents L-dopa-induced sensitisation in the 6-OHDA-lesioned rat
H. Ponten, A. Carlsson, J. Kullingsjo, C. Sonesson, N. Waters, J. Tedroff

P1143 The diagnosis and management of pergolide-induced fibrosis
P. Agarwal, S. Fahn, S.J. Frucht

P1144 Effects of gap junction blockade in the MPTP-lesioned primate and rodent models of L-DOPA-induced dyskinesia
J. Lee, J. Gomez-Ramirez, T. Johnston, P. Carlen, A.E. Lang, J.M. Brotchie

P1145 Treatment of restless legs syndrome with subcutaneous apomorphine in a patient with short bowl syndrome
T. Tings, G. Stiens, W. Paulus, C. Trenkwalder, S. Happe

P1146 Modulation of histamine H3 receptor-mediated transmission in the MPTP-lesioned non-human primate and rodent models of L-DOPA-induced dyskinesia
J. Gomez-Ramirez, J. Lee, T. Johnston, S.H. Fox, J.M. Brotchie

P1147 Effect of zolpidem on parkinsonian symptoms in patients with advanced Parkinson's disease
G.A. Tagaris, V. Sakkou, P. Zikos, A. Sarafianos, P. Vrentas, C.E. Karageorgiou

P1148 A simple *in vivo* assay in the rodent for identifying novel drug therapies for L-DOPA-induced dyskinesia
T. Johnston, J. Lee, J. Gomez-Ramirez, S.H. Fox, J.M. Brotchie

P1149 Effects of *de novo* treatment with the alpha-2 adrenergic receptor antagonist, idazoxan, on the development of L-DOPA-induced motor complications
T. Johnston, J.M. Brotchie

P1150 Open-label, dose escalation, safety study of Botulinum Toxin Type B (MYOBLOC™) in patients with Hemifacial Spasm
R.M. Trosch, C.H. Adler

P1151 Levodopa for chronic neck pain: A cross-over double blind, placebo controlled study
M. Marziniak, V. Guralnik, U. Dillmann, G. Becker

P1152 Subcellular re-distribution of the synapse-associated proteins, PSD95 and SAP97 in animal models of Parkinson's disease and L-DOPA-induced dyskinesia
J.E. Nash, J. Gomez-Ramirez, G.L. Collingridge, C.C. Garner, J.M. Brotchie

P1153 The effects of NMDA receptor antagonism in a rat model of tardive dyskinesia
C. Tsironis, D. Kiortsis, A. Evangelou, S. Konitsiotis

P1154 Tetrabenazine treatment in hyperkinetic movement disorders
D. Paleacu, N. Giladi, O. Moore, M. Stein, S. Honigman, S. Badarny

P1155 Three cases of peripheral edema caused by prolonged use of ropinirole
D. Apetauerova, J. Srinivasan, P. Gross

P1156 A high throughput screening assay for identifying potential neuroprotective agents in Parkinson's disease
J.E. Nash, S.Y. Lee, S. Kalia, A.M. Lozano

P1157 Continous stimulation: Supplementation of levodopa/carbidopa with entacapone reduces movement fluctuations
T. Muller, J. Welnic, N. Meisel, S. Muhlack, D. Woitalla, D. Bremen

P1158 Cabergoline stimulates synthesis and secretion of neurotrophic factors by primary cultured neurons or astrocytes
S.A. Kuno, K. Ohta, A. Fujinami, A. Sakakimoto, Y. Kitaura, M. Ohta

P1159 Effect of (–)-BPAP on the expression of neurotrophins and their receptors in mesencephalic slice cultures
S. Shimazu, K. Takahata, C. Hirami, K. Hayashi, F. Yoneda, A. Akaike

P1160 Double blind evaluation of symptomatic effect at the end of a 12 week treatment with safinamide, a new neuroprotectant
M. Onofrj, A. Thomas, P. Sala, G. Nordera, G. Fabbrini, R. Fariello

P1161 Changes in neuroendocrine response to L-DOPA during long term treatment in restless legs syndrome augmentation
D. Garcia-Borreguero, R. Egatz, O. Larrosa, C. Serrano

P1162 Aripiprazole in the treatment of movement disorders
M.S. Eisa, B. Jabbari

POSTER SESSION 4

P1163 Endocannabinoid levels are altered in parkinsonism and L-DOPA-induced dyskinesia in the MPTP-lesioned macaque
M. Van Der Stelt, S.H. Fox, V. Di Marzo, J.M. Brotchie

P1164 Involvement of both $delta_1$ and $delta_2$ opioid receptors in the anti-parkinsonian actions of the delta opioid receptor agonist SNC80 in the reserpine-treated rat
P. Hallett, J.M. Brotchie

P1165 A double-blind, randomized, parallel group design comparison of botulinum toxin, type A (Botox) and botulinum toxin, type B (Myobloc) on systemic and ocular autonomic symptoms and physiology in patients with cervical dystonia
R. Tintner, R.L. Gross, U.F. Winzer, K.A. Smalky, J. Jankovic

P1166 Anti-parkinsonian effects of delta opioid receptor stimulation are accompanied by dystonia in MPTP-lesioned non-human primates previously treated with L-DOPA
T. Johnston, S.H. Fox, J. Gomez-Ramirez, J. Lee, J.M. Brotchie

Non-motor Aspects of Movement Disorders

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P1167 Aripiprazole for drug-induced psychosis in Parkinson's disease: Preliminary experience
H.H. Fernandez, M.E. Trieschmann, J.H. Friedman

P1168 Discontinuation of antipsychotic drugs in stable Parkinson patients with a history of drug-induced psychosis
H.H. Fernandez, M.E. Trieschmann, M.S. Okun

P1169 Personality in essential tremor: Additional evidence of non-motor manifestations of the disease?
A. Chatterjee, E.C. Jurewicz, L.M. Applegate, E.D. Louis

P1170 Psychomotor speed, flexibility and verbal memory in Huntington's disease
J. Roth, M. Preiss, J. Klempir, H. Brozova, O. Ulomanova, E. Ruzicka

P1171 Familial risk factors for hallucinations in Parkinson's disease
R. Inzelberg, D. Paleacu, E. Schechtman, R.L. Carasso, S.C. Blumen, P. Nisipeanu

P1172 Cognitive function in multiple system atrophy of the cerebellar type (MSA-C)
K. Bürk, C. Globas, I. Daum

P1173 Quetiapine for sleep disorders in Parkinson's disease
C.C. Juri, P.C. Chana, T.J. Parrao, J.N. Tapia, C.R. Kunstmann, D.S. Alburquenque

P1174 Working memory deficits correlate with frontal atrophy in progressive supranuclear palsy
N.J. Cordato, C. Pantelis, D. Caine, D. Velakoulis, S.J. Wood, J.G. Morris

P1175 Visuospatial attention in Huntington's disease
J. Fielding, N. Georgiou_Karistianis, L. Millist, A. Churchyard, E. Chiu, O. White

P1176 Parkinson's disease dementia in a community-based autopsy sample of dementia
J.B. Leverenz, D.W. Tsuang, E.B. Larson, T. Montine, M. Kraybill, D. Nochlin

P1177 Effects of levodopa and STN stimulation on decision making in Parkinson's disease
A. Funkiewiez, C. Ardouin, P. Krack, B. Dubois, A.-L. Benabid, P. Pollak

P1178 The effect of voice therapy on feedback control in parkinsonian speech
J.F. Houde, S. Nagarajan, T. Heinks, C.M. Fox, L.O. Ramig, W.J. Marks

P1179 An observational study of pattern and occurence of non-motor symptoms in Parkinson's disease
A. Gulati, A. Forbes, F. Stegie, C. Clough, R.K. Chaudhuri

P1180 The role of cerebrovascular risk factors for dementia in PD
K. Haugarvoll, D. AArsland, J.P. Larsen

P1181 The Parkinson Fatigue Scale (PFS-16): The development of a new disease-specific instrument for use in research and clinical practice
R.G. Brown, A. Dittner, L. Findley, S.C. Wessley

P1182 Vigilance states in a parkinsonian model, the MPTP mice
C. Monaca, C. Laloux, R. Bordet, L. Defebvre, A. Destee, P. Derambure

P1183 Anticipatory responses in rapid stimulus streams as a measure of motor impulsivity in Tourette syndrome
F. Richer, M. Thibeault, S. Marti, G. Rouleau, S. Chouinard

P1184 Decreased cognitive control during deep brain stimulation of the STN
T. Hershey, F.J. Revilla, A. Wernle, P. Schneider-Gibson, J.L. Dowling, J.S. Perlmutter

P1185 Validation of the Quality of Life in Essential Tremor Questionnaire (QUEST)
A.I. Tröster, R. Pahwa, C.M. Tanner, K.E. Lyons

P1186 Emotional memory in Parkinson's disease
T.D. Hälbig, F. Wodarz, U.A. Kopp, G. Ebersbach, A. Kupsch

P1187 A clinical observational study of the pattern and occurrence of non-motor symptoms in Parkinson's disease ranging from early to advanced disease
A. Gulati, F. Alison, S. Frauke, K. Linda, C. Chris, K.R. Chaudhuri

P1188 Radiotherapy as treatment of hypersalivation in Parkinson's disease and MSA; long-term follow-up data on the efficacy and safety
T. van Laar, M.A. Heesters, K.L. Leenders

P1189 Modification of respiratory function parameters in patients with severe Parkinson's disease
M. De Pandis, F. Stefanelli, G. De Simone, C. Iannella, I. Meoli, F. Stochhi

P1190 Cognitive function in adult patients with Sydenham's chorea: Preliminary results
R.G. Beato, F.E. Cardoso

P1191 Obsessive-compulsive behavior and hyperactivity and attention deficit disorder in Sydenham Chorea
D.P. Maia, F.E. Cardoso, M.C. Cunningham, A.L. Teixeira

P1192 Affective disturbances and quality of life in patients with essential tremor
P.V. Makedonsky, O.S. Levin

P1193 Defective imitation of limb gestures in Lewy body dementia: A useful early clinical sign
C. Aveleyra, G. Russo, F. Manes, M. Merello, R. Leiguarda

P1194 Comparing semantic, letter and action fluency in Huntington's disease: A preliminary study
M.J. Azambuja, M.S. Haddad, M. Radanovic, E.R. Barbosa, L.L. Mansur

P1195 Depression predicts the pattern of cognitive impairment in early Parkinson's disease
E. Stefanova, A. Potrebic, L. Ziropadja, A. Maksimovic, N. Dragasevic, V. Kostic

P1196 The result of dopaminergic degeneration and the effects of medication on bladder function in Parkinson's disease
K. Winge, L. Friberg, L. Werdelin, K.K. Nielsen, H. Stimpel

P1197 Osmolarity depended effect of water on blood pressure
A. Lipp, J. Jordan, G. Arnold

P1198 Does bilateral high-frequency stimulation of the subthalamic nucleus aggravate apathy in Parkinson's disease?
V. Czernecki, B. Pillon, J.-L. Houeto, M.-L. Welter, V. Mesnages, Y. Agid

P1199 Cognitive initial symptoms and disease progression in cortico-basal degeneration
T. Sgaramella, L. Bartolomei, V. Toso

P1200 The frontal syndrome in Parkinson's disease: Its prevalence and direct impact on activities of daily living
M.-A. Bedard, F. Paquet, S. Chouinard, P. Blanchet, V. Soland, J. Filion

P1201 To ascertain the effect of levodopa on mood in parkinsonism
S.A. Molloy, J.T. O'Brien, I.G. McKeith, D.J. Burn

P1202 Neuropsychiatric symptoms in Parkinson's disease patients presenting for subthalamic stimulation
V. Voon, J.A. Saint-Cyr, A.M. Lozano, E. Moro, A.E. Lang

P1204 Impairment of linguistic rule application in basal ganglia disease
M.M. Teichmann, E.E. Dupoux, A.-C.A. Bachoud-Lévi

P1205 Hallucinations induced by antidepressants in PD patients
E. Fabrizio, C. Pauletti, C. Aurilia, M. Colasanti, G. Fabbrini, G. Meco

P1206 Parkinson's disease and pesticide exposure: Does a selective cognitive profile exist?
M. Gasparini, G. Caldora, E. Fabrizio, S. Di Rezze, N. Vanacore, G. Meco

P1207 Psychosis in the course of Parkinson's disease and the treatment response to atypical antipsychotics
T. Sobow, M. Gorczowski, I. Kloszewska

P1208 Severe "off anxiety" improves with deep brain stimulation of the subthalamic nucleus
D.E. Hardesty, S.L. Ryan, C. Henchcliffe, P. Piboolnurak, L. Winfield, B. Ford

P1209 Long-term effect of continuous positive air pressure (CPAP) in the treatment of nocturnal stridor in multiple system atrophy (MSA)
A. Iranzo, J. Santamaria, E. Tolosa, I. Vilaseca, F. Valldeoriola, M. Marti

Other Clinical

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P1210 Osteoporosis in Parkinson's disease
B. Wood, J. Hunter, R. Walker

P1211 Cognitive assessment of a representative community population with Parkinson's disease using the Cambridge Cognitive Assessment-Revised (CAMCOG-R)
R.J. Athey, R.W. Porter, R. Walker

P1212 Deep brain stimulation of the ventral caudate nucleus is effective in obsessive-compulsive disorder and major depression
B. Aouizerate, E. Cuny, C. Martin-Guehl, D. Guehl, H. Amieva, P. Burbaud

P1213 Cognitive function testing in ADHD children: Potential utility of a novel, web-based battery
R. Barak, Y. Leitner, E.S. Simon, N. Giladi, J.M. Hausdorff

P1214 Gait variability in older adults with age-associated cognitive decline
J.M. Hausdorff, M. Mordechovitch, N. Giladi

P1215 Attention: Regulation of stride-to-stride variability of gait may requires attention
Y. Leitner, R. Barak, N. Giladi, L. Gruendlinger, J.M. Hausdorff

P1216 Screening for falls, stroke or dementia risk factors in a self- referral, middle age population: Is it worth the effort?
N. Giladi, M. Mordechovitch, J.M. Hausdorff, H. Shabtai, Y. Balash, L. Gruendlinger

P1217 Deficits in executive function in idiopathic elderly fallers: Association with fall risk
S. Springer, N. Giladi, E.S. Simon, J.M. Hausdorff

P1218 Restless legs syndrome with L thyroxine: Clinical, biochemical and polysomnographic correlation
P. Ratnagopal, E. Tan, S. Ho, L. Koh

P1219 Retinal nerve fiber layer thinning in Parkinson's disease
R. Inzelberg, A. Ramirez, P. Nisipeanu, R.L. Carasso, A. Ophir

P1220 Daytime somnolence in ADHD patients treated with the dopamine receptor agonist ropinirole
M. Gerlach, A. Claus, S. Peter, W. Christoph, A. Warnke

P1221 Psychogenic or non-psychogenic dysarthrophonia?
D. Haubenberger, M. Vigl, I. Busslinger, D.-M. Denk, E. Fertl, E. Auff

P1222 Restless legs syndrome refractory to therapy: Succesfull treatment with continuous intrathecal morphine application
J. Haan, A. Koulousakis, D. Lenartz, V. Sturm

P1223 Does actigraphy provide good PLM counts? A validation study with polysomnography
B. Hogl, V. Gschliesser, B. Frauscher, E. Brandauer, H. Ulmer, W. Poewe

P1224 Incidence of vascular hemiballism in the population of Belgrade
V.S. Kostic, T. Pekmezovic, M.V. Svetel, A. Ristic, R. Raicevic, N. Ivanovic

P1225 Parkinsonism and exposure to neuroleptic drugs in residents of an Italian nursing home
G. Riboldazzi, D. Calandrella, A. Citterio, C. Mascetti, G. Bono, E. Martignoni

P1226 Static disturbances in different variants of cerebrovascular pathology
L.L. Kononova, N.J. Anan'eva, O.A. Balunov

P1227 Efficacy of graduated-dose levetiracetam in treating restless legs syndrome and associated hypersomnia: A pilot study
D.M. Lacey

P1228 Optic ataxia due to impaired visuomotor transformation
T. Haid, M. Kofler, E. Pucks-Faes, A. Mayr, E. Quirbach, S. Felber

P1229 A new approach to improve the reliability and validity of RLS diagnoses: The restless legs syndrome diagnostic index (RLS-DI)
H. Benes

P1230 Prevalence and severity of restless legs syndrome in France - The "INSTANT Study"
F. Tison, E. Lainey, D. Leger, A. Crochard, S. Bouee, A. El Hasnaoui

P1231 Treatment of facial synkinesis and hyperlacrimation following facial nerve palsy with botulinum toxin
T. Abe, C. Tanaka, K. Sako, M. Mizobuchi, A. Nihira, T. Matsushita

P1232 The long-term management of RLS with ropinirole: Maintained efficacy over 36 weeks
J. Haan, D. Volc, J. Montplaisir

P1233 Restless arms syndrome
L. Queiroz, F.C. Freitas

P1234 Post-infectious autoimmune neuropsychiatric disorder associated with streptococcus (PANDAS), is it a self-limited disease? A six-year follow-up
G. Fabiani, H. Teive

P1235 Symptoms miming of Lewy body disease (LBD) in young patient after heroin intoxication
D. Kountouris, K. Koutsoubelis

P1236 Pramipexole induces a rapid and substantial improvement of idiopathic restless legs syndrome: Results of a large randomized double-blind placebo-controlled dose-finding study
K. Hirvonen, M. Partinen, A. Alakuijala, L. Jama, J. Terttunen

P1237 An inbred-strain of mice as an animal model of restless legs syndrome (RLS)
B.C. Jones, E.J. Chesler, R.W. Williams, J.L. Beard, R.P. Allen, C.J. Earley

P1238 Safety and efficacy of tetrabenazine in childhood hyperkinetic movement disorders
K. Vuong, C.B. Hunter, N. Mejia, J. Jankovic

P1239 Periodic limb movements during sleep or restless legs syndrome in patients on hemodialysis
Y. Oka, S. Koike, Y. Inoue, K. Yamamoto, H. Kadotani, A. Ikeda

P1240 Painful arm and moving fingers: Clinical features of six cases
S. Lee, W. Yoon, E. Jeong, W. Lee

P1241 Antidepressants and Periodic Leg Movement Disorder
G.N. Rizzo

P1242 The adverse-event profile of ropinirole in the treatment of RLS
P. Montagna, P. Tidswell, B. Yee

P1243 The evidence base for ropinirole in RLS: Results from an extensive clinical trial programme
L. Ferini-Strambi, J. Montplaisir, T. Dreykluft

P1244 Alternating hemiplegia of childhood: A family with possible recessive inheritance
A. de Falco, V. Scarano, M. Buongiorno, E. Marano, G. De Michele, A. Filla

P1245 A family with atypical parkinsonism and diffuse leukoencephalopathy with spheroids
Y. Baba, R.J. Uitti, D.W. Dickson, Z.K. Wszolek, T. Bird, B. Ghetti

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P1246 L-Dopa therapy working only on a late phase of disease in a case of neurodegeneration with brain iron accumulation (NBIA)
L. Bartolomei, G. Billo, V. Toso

P1247 Efficacy of cabergoline for the treatment of sensori-motor symptoms and sleep disturbances in restless legs syndrome: A placebo-controlled, 5-week, double-blind, randomized, multicenter, polysomnographic study
W.H. Oertel, H. Benes, S. Happe, R. Kohnen, M. Leroux, K. Stiasny-Kolster

P1248 Chorea and epilepsia partialis continua as initial signs in probable Creutzfeldt-Jacob disease
B. Donmez, I. Oztura, R. Cakmur

P1249 Frontotemporal degeneration with astrocyte-predominant tauopathy and motor neuron disease mimicking corticobasal degeneration: An autopsy case
K. Iwanaga, I. Tomita, A. Satoh, M. Tsujihata, Y. Piao, H. Takahashi

P1250 A case with painless moving toes syndrome
S.-J. Kwon, J.-M. Kim, K. Cho, B. Jeon

P1251 Lessons learned from the long-term cabergoline safety trial in restless legs syndrome patients
H. Benes, M. Leroux, R. Kohnen

P1252 Restless legs syndrome: A case-series study of 97 patients
V. Kiriakakis, Y. Kapsalakis, P. Vrentas, N. Tsiftsis

P1253 Paroxysmal unilateral spasm of the jaw with facial atrophy. A rare manifestation of trigeminal cranial neuropathy
N. Galvez-Jimenez, A. Podichetty, M. Hargreave

P1254 Defining clinical relevance of treatment outcome in studies with restless legs syndrome patients: Example from the cabergoline dose-finding trial
R. Kohnen, H. Benes, M. Leroux, K. Stiasny-Kolster, W.H. Oertel

P1255 Long-term effects of pramipexole in the treatment of restless legs syndrome
M. Fantini, A. Desautels, M. Michaud, D. Petit, J. Montplaisir

P1256 Reversible extra-pontine and central pontine myelinolysis presenting with extrapyramidal features
B. Ho, D. Apetauerova, J. Arle, J. Russell

P1257 Anti-histamine and daytime benzodiazepine effects on restless legs syndrome (RLS) symptoms
R.P. Allen, S. Lesage, C.J. Earley

P1258 Camptocormia or bent spine? A syndrome deserving a closer look
W.R. Schabitz, H.H. Goebel, H.M. Meinck

P1259 REM ehavioral manifestations in the female patients with REM sleep behavior disorder preceding parkinsonism
H.-I. Ma, Y.-J. Kim, M.-K. Chu, K.-H. Yu, B.-C. Lee

P1260 Hemifacial spasm: A clinical and epidemiological study
C. Colosimo, M. Chianese, G. Fabbrini, G. Defazio, M. Prencipe, A. Berardelli

P1261 Ropinirole reduces periodic leg movements in sleep in patients with RLS
R.P. Allen, P.C. Mistry, M. Kelly

P1262 The long-term safety and tolerability of ropinirole in RLS
T. Dreykluft, J. Karrasch, J. Smuts, D. Volc, R. Grunstein, J. Montplaisir

P1263 Long term prognosis of psychogenic movement disorders
M. Thomas, P. Banuelos, K.D. Vuong, J. Jankovic

P1264 Obesity in severe neurological diseases: Treatment with botulinum toxin
M. Porta, G.R. Maggioni, F. Lella

P1265 Clinical, neuropathologic, and molecular genetic studies on Gerstmann-Sträussler-Scheinker disease (F198S-V129)
K. Yamaguchi, B. Ghetti, P. Piccardo, F. Epperson, L. Miravalle, M.R. Farlow

P1266 A case of restless legs syndrome secondary to ischemic stroke in the claustrum-caudate complex
G. Sechi, P. Galistu, V. Agnetti, I. Pirastru, B. Murgia, K.S. Paulus

P1267 Clinical heterogeneity of movement disorders associated with antiphospholipid syndrome
N.-K. Chew, D. Martino, P. Mir, N. Bajaj, M. Edwards, K.P. Bhatia

P1268 Circadian rhythm of restless legs syndrome symptoms: Relationships with salivary melatonin, core body temperature, and subjective vigilance
M. Michaud, M. Dumont, B. Selmaoui, J. Paquet, M.L. Fantini, J. Montplaisir

P1269 Nocturnal quiescegenic dyskinesia: A new sleep-related movement disorder
S. Lesage, R. Allen, C. Earley

P1270 Manganese, movement disorders, and welding
J.Y. Fang, T.L. Davis

P1271 Polysomnographic features of REM sleep behavior disorder
M. Fantini, J.-F. Gagnon, S. Rompré, L. Ferini Strambi, J. Montplaisir

P1272 Periodic leg movements in REM sleep behavior disorder: Further observations
M. Fantini, M. Michaud, J.-F. Gagnon, L. Ferini Strambi, J. Montplaisir

P1273 Dystonia secondary to tetrahydrobiopterin deficiency treated with amantadine hydrochloride in childhood-recognition of the movement disorder
B.L. Lavenstein, C. Greene, K. Rosenbaum

P1274 Diagnostic pallidotomy for primary CNS lymphoma (case report)
F.I. Khan, R. Young

P1275 Different botulinum neurotoxin serotypes injection on the parotid glands in human: A comparison study
R. Eleopra, G. Scanelli, A. De Vito, V. Tugnoli, R. Quatrale, O. Rossetto

P1276 *Mindstreams* computerized cognitive tests identify MCI and mild dementia even in the presence of depression
E.S. Simon, D.M. Zucker, H. Chertkow, T. Dwolatzky, H. Crystal, G.M. Doniger

P1277 Clinical models of continuous dopaminergic stimulation
F. Stocchi, G. Battaglia, L. Vacca, P. Grassini, N. Modugno, W.C. Olanow

Tics

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P1278 Levetiracetam in treatment of tics in Tourette syndrome
Y.M. Awaad, A.M. Michon, S.G. Minarik

P1279 Video assessment of rTMS for Tourette syndrome
A.H. Snijders, B.R. Bloem, M. Orth, M.R. Trimble, M.M. Robertson, A. Munchau

P1280 Aripiprazole is beneficial in refractory tics
P. Agarwal, R. Kumar, V. Segro, L.C. Seeberger

P1281 Tetrabenazine, monoamine depleter, effective in the treatment of Tourette syndrome
C.B. Hunter, K. Vuong, N. Mejia, J. Jankovic

P1282 Dopamine transporter imaging in Tourette syndrome: Evaluation by NeuroSPECT of Trodat 1-Tc99m
I. Mena, M. Miranda, M. Hernandez, M. Fruns

P1283 Bilinear transmission of Tourette's syndrome in a semi-isolated population
V. Scarano, G. Volpe, T. Tucci, P. Mancini, V. Brescia Morra, G. De Michele

P1284 Geriatric tics are often linked to an exacerbation of early life symptoms by an external trigger
J. Filion, F. Richer, V.L. Soland, P.J. Blanchet, S. Chouinard

P1285 Soluble adhesion molecules in Tourette's syndrome
D. Martino, A.J. Church, R.C. Dale, M. Orth, M.M. Robertson, G. Giovannoni

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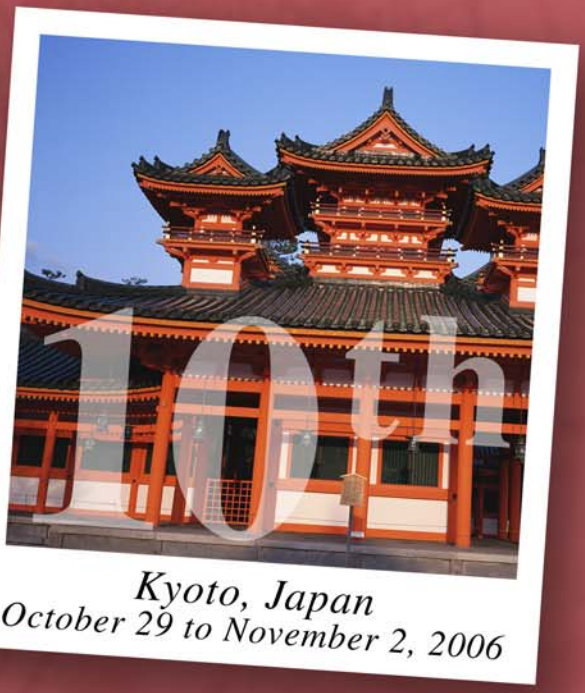

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