The *Movement* Disorder Society's

9th International Congress of Parkinson's Disease & Movement Disorders

FINAL PROGRAM

NEW Orleans

LOUISIANA, USA • MARCH 5~8, 2005

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NEW Orleans

LOUISIANA, USA • MARCH 5~8, 2005

The photographs of New Orleans featured in this program were provided by the New Orleans Metropolitan Convention and Visitor's Bureau.

GE Healthcare

Essential Tremor

Early stage
Parkinson's Disease

DaTSCAN™

IOFLUPANE (^{123}I)

The objective adjunct for clinically uncertain parkinsonian syndromes patients[1]

PRESCRIBING INFORMATION. DaTSCAN™ ioflupane (^{123}I)
Please refer to full national Summary of Product Characteristics (SPC) before prescribing. Indications and approvals may vary in different countries. Further information available on request. **PRESENTATION** Vials containing 185 MBq or 370 MBq ioflupane (^{123}I) at reference time. **INDICATIONS** 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 METHOD OF 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. See SPC. **CONTRAINDICATIONS** Pregnancy and in patients with hypersensitivity to iodide or any of the excipients. **WARNINGS AND 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, benzatropine, bupropion, cocaine, mazindol, methylphenidate, phentermine and sertraline. Drugs shown during clinical trials not to interfere with DaTSCAN imaging include amantadine, trihexyphenidyl, 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. **UNDESIRABLE 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. **MARKETING AUTHORISATION HOLDER** Amersham plc, Little Chalfont, Bucks, UK. **CLASSIFICATION FOR SUPPLY** Subject to medical prescription. **MARKETING AUTHORISATION NUMBERS** EU/1/00/135/001 and EU/1/00/135/002. **DATE OF REVISION OF TEXT** 11 October 2004.

Reference: 1. Catafau A and Tolosa E. Mov Disord 2004

DaTSCAN holds a European marketing authorization but is not approved for use by the US Food and Drug Administration

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www.gehealthcare.com

12-2004 JB1219/OS

Welcome Letter

Dear Colleagues,

Welcome to The *Movement* Disorder Society's (MDS) 9th International Congress of Parkinson's Disease and Movement Disorders.

We are pleased to convene this International Congress and encourage you to take every opportunity to participate in the Scientific Program which features world renowned speakers. In the next days, the latest research and perspectives regarding Movement Disorders will be presented and discussed in an open format, offering unique educational opportunities for participants.

The International Congress begins with a series of Kickoff Seminars and then continues with an array of Plenary, Parallel, Poster and Video Sessions, as well as Controversies and Skills Workshops. New to this year's International Congress, the Parallel Sessions and Skills Workshops have been designed to meet the requested need for smaller, more focused sessions. As a result, they will be able to provide greater in-depth coverage of specific topics and allow for additional audience participation.

Please also plan to participate in the Opening Ceremony and Welcome Reception on Saturday evening. The Welcome Reception will celebrate the unique styles, tastes and culture of the city of New Orleans. As a city famous for its French Quarter, jazz music, lively entertainment, distinctive cultures, architecture and the only surviving historic streetcar system in the United States, New Orleans is an ideal setting for the International Congress.

This is our first International Congress to convene in our new annual format, and we are looking forward to the many new and exciting opportunities that this format will allow both this year and in the future.

Thank you for attending.

With best regards,

C. Warren Olanow, MD
President, The *Movement* Disorder Society, 2003-2004
Chair, Congress Scientific Program Committee

Andrew J. Lees, MD FRCP
President, The *Movement* Disorder Society, 2005-2006

Anthony E. Lang, MD FRCPC
Co-chair 2005, Congress Scientific Program Committee

Acknowledgements

The International Congress Oversight Committee of the 9th International Congress of Parkinson's Disease and Movement Disorders wishes to acknowledge and thank the following companies for providing support in the form of educational grants:

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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

Be sure to attend the MDS Business Meeting.

Monday, March 7, 2005 ~ 7:30 a.m. to 8:30 a.m.

Location: Acadia Room, Third Floor, Marriott

Organization

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MDS International Secretariat

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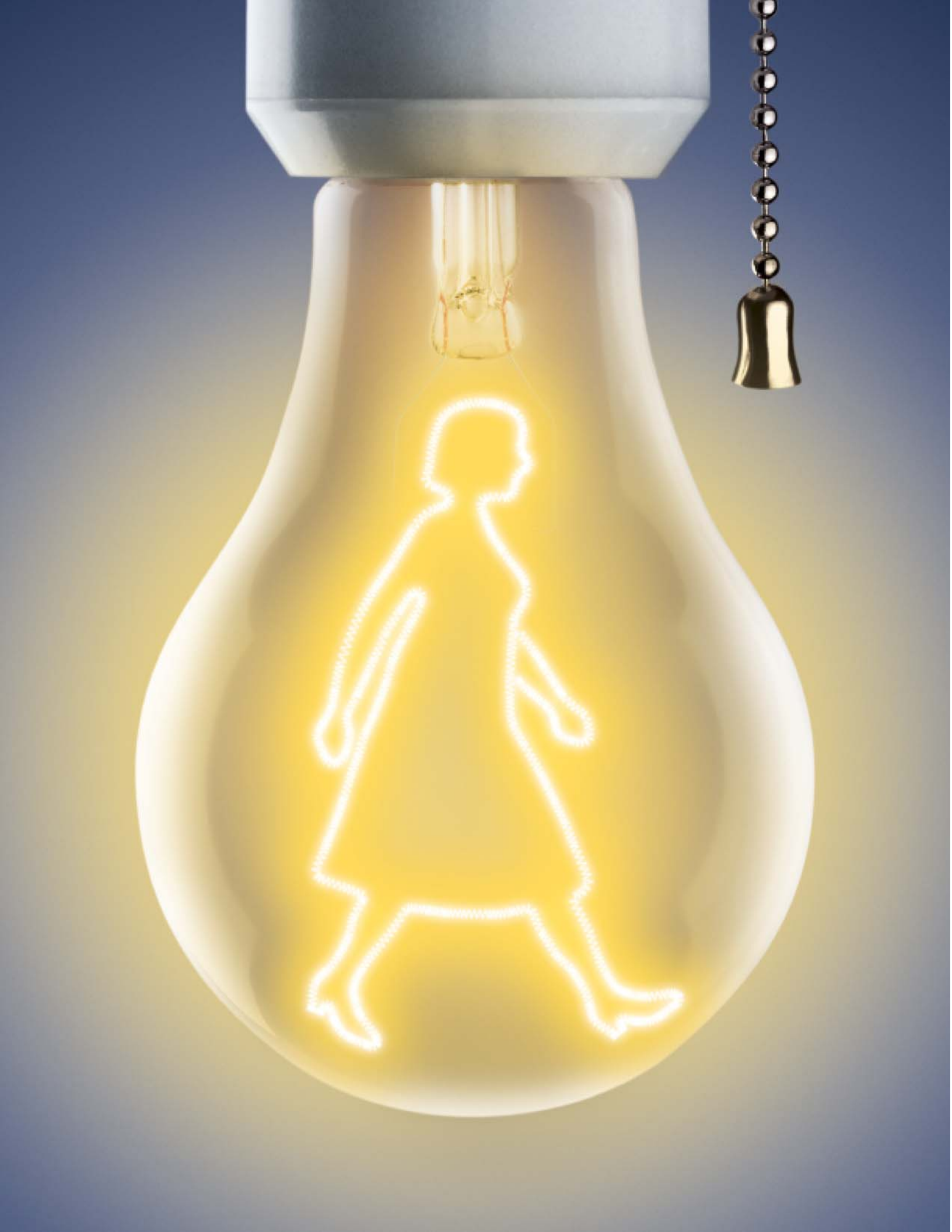

An adjunct therapy to levodopa/carbidopa
for the treatment of the signs and symptoms
of idiopathic Parkinson's disease

KEEP THEM "ON" LONGER

TASMAR improves symptom control with more "ON" time

- Increases "ON" time 1.7–2.9 hours and decreases "OFF" time 1.6–3.2 hours per 16-hour waking day[1-4]
- 71% to 91% improvement in symptom severity and "wearing-off" effect at 3 months[2]
- Reduces levodopa daily dose by up to 29% due to a significant increase of levodopa bioavailability[1]

Liver monitoring is essential for patients taking TASMAR.

REFERENCES: 1. Adler CH, Singer C, O'Brien C, et al. Randomized, placebo-controlled study of tolcapone in patients with fluctuating Parkinson's disease treated with levodopa-carbidopa. *Arch Neurol.* 1998;55:1089-1095. **2.** Rajput AH, Martin W, Saint-Hilaire MH, et al. Tolcapone improves motor function in parkinsonian patients with the "wearing-off" phenomenon: a double-blind, placebo-controlled, multicenter trial. *Neurology.* 1997;49:1066-1071. **3.** Baas H, Beiske AG, Ghika J, et al. Catechol-O-methyltransferase inhibition with tolcapone reduces the "wearing off" phenomenon and levodopa requirements in fluctuating parkinsonian patients. *J Neurol Neurosurg Psychiatry.* 1997;63:421-428. **4.** TASMAR Complete Prescribing Information (US).

USE OF TASMAR REQUIRES WRITTEN INFORMED CONSENT BY THE PATIENT (SEE PATIENT CONSENT SECTION IN THE COMPLETE PRODUCT INFORMATION).

WARNING: Due to the risk of potentially fatal, acute fulminant liver failure, TASMAR should ordinarily be used in patients with Parkinson's disease on levodopa/carbidopa who have symptom fluctuations and are not responding satisfactorily to or who are not appropriate for other adjunctive therapies (see INDICATIONS and DOSAGE AND ADMINISTRATION).

TASMAR should not be initiated in patients with clinical evidence of liver disease or 2 SGPT/ALT or SGOT/AST values >ULN and should be discontinued if substantial clinical benefit is not seen within 3 weeks.

Patients with severe dyskinesia or dystonia should be treated with caution (see PRECAUTIONS: *Rhabdomyolysis*).

Frequent laboratory monitoring is essential (see PRECAUTIONS: *Laboratory Tests* for the recommended schedule). Liver monitoring may not prevent liver failure; however, early detection and immediate drug withdrawal are believed to enhance the likelihood for recovery. Patients should be advised to self-monitor for signs of liver disease. Discontinue TASMAR if hepatic enzymes exceed ULN or patient exhibits signs of liver failure.

TASMAR® (tolcapone) tablets 100mg/200mg

Longer-lasting "ON" time

VALEANT Pharmaceuticals International

TASMAR®
(tolcapone)
TABLETS

PLEASE READ: THIS INFORMATION IS ONLY A BRIEF SUMMARY AND THE PACKAGE INSERT SHOULD BE CONSULTED FOR THE COMPLETE PRESCRIBING INFORMATION.

Before prescribing TASMAR, the physician should be thoroughly familiar with the details of this prescribing information.

TASMAR SHOULD NOT BE USED BY PATIENTS UNTIL THERE HAS BEEN A COMPLETE DISCUSSION OF THE RISKS AND THE PATIENT HAS PROVIDED WRITTEN INFORMED CONSENT (SEE PATIENT CONSENT SECTION).

WARNING:

Because of the risk of potentially fatal, acute fulminant liver failure, TASMAR (tolcapone) should ordinarily be used in patients with Parkinson's disease on l-dopa/carbidopa who are experiencing symptom fluctuations and are not responding satisfactorily to or are not appropriate candidates for other adjunctive therapies (see INDICATIONS and DOSAGE AND ADMINISTRATION sections).

Because of the risk of liver injury and because TASMAR, when it is effective, provides an observable symptomatic benefit, the patient who fails to show substantial clinical benefit within 3 weeks of initiation of treatment, should be withdrawn from TASMAR.

TASMAR therapy should not be initiated if the patient exhibits clinical evidence of liver disease or two SGPT/ALT or SGOT/AST values greater than the upper limit of normal. Patients with severe dyskinesia or dystonia should be treated with caution (see PRECAUTIONS: Rhabdomyolysis).

Patients who develop evidence of hepatocellular injury while on TASMAR and are withdrawn from the drug for any reason may be at increased risk for liver injury if TASMAR is reintroduced. Accordingly, such patients should not ordinarily be considered for retreatment.

Cases of severe hepatocellular injury, including fulminant liver failure resulting in death, have been reported in postmarketing use. As of October 1998, 3 cases of fatal fulminant hepatic failure have been reported from approximately 60,000 patients providing about 40,000 patient years of worldwide use. This incidence may be 10- to 100-fold higher than the background incidence in the general population. Underreporting of cases may lead to significant underestimation of the increased risk associated with the use of TASMAR.

A prescriber who elects to use TASMAR in face of the increased risk of liver injury is strongly advised to monitor patients for evidence of emergent liver injury. Patients should be advised of the need for self-monitoring for both the classical signs of liver disease (eg, clay colored stools, jaundice) and the nonspecific ones (eg, fatigue, loss of appetite, lethargy).

Although a program of frequent laboratory monitoring for evidence of hepatocellular injury is deemed essential, it is not clear that baseline and periodic monitoring of liver enzymes will prevent the occurrence of fulminant liver failure. However, it is generally believed that early detection of drug-induced hepatic injury along with immediate withdrawal of the suspect drug enhances the likelihood for recovery. It is also widely held, without a robust body of evidence, that patients with preexisting hepatic disease are more vulnerable to hepatotoxins. Accordingly, the following liver monitoring program is recommended.

Before starting treatment with TASMAR, the physician should conduct appropriate tests to exclude the presence of liver disease. In patients determined to be appropriate candidates for treatment with TASMAR, serum glutamic-pyruvic transaminase (SGPT/ALT) and serum glutamic-oxaloacetic transaminase (SGOT/AST) levels should be determined at baseline and then every 2 weeks for the first year of therapy, every 4 weeks for the next 6 months, and then every 8 weeks thereafter. If the dose is increased to 200 mg tid (see DOSAGE AND ADMINISTRATION section), liver enzyme monitoring should take place before increasing the dose and then be reinitiated at the frequency above.

TASMAR should be discontinued if SGPT/ALT or SGOT/AST exceeds the upper limit of normal or if clinical signs and symptoms suggest the onset of hepatic failure (persistent nausea, fatigue, lethargy, anorexia, jaundice, dark urine, pruritus, and right upper quadrant tenderness).

INDICATIONS: TASMAR is indicated as an adjunct to levodopa and carbidopa for the treatment of the signs and symptoms of idiopathic Parkinson's disease. Because of the risk of potentially fatal, acute fulminant liver failure, TASMAR (tolcapone) should ordinarily be used in patients with Parkinson's disease on l-dopa/carbidopa who are experiencing symptom fluctuations and are not responding satisfactorily to or are not appropriate candidates for other adjunctive therapies. Because of the risk of liver injury and because TASMAR, when it is effective, provides an observable symptomatic benefit, the patient who fails to show substantial clinical benefit within 3 weeks of initiation of treatment, should be withdrawn from TASMAR.

The effectiveness of TASMAR was demonstrated in randomized controlled trials in patients receiving concomitant levodopa therapy with carbidopa or another aromatic amino acid decarboxylase inhibitor who experienced end of dose wearing-off phenomena as well as in patients who did not experience such phenomena.

CONTRAINDICATIONS: TASMAR tablets are contraindicated in patients with liver disease in patients who were withdrawn from TASMAR because of evidence of TASMAR-induced hepatocellular injury or who have demonstrated hypersensitivity to the drug or its ingredients.

TASMAR is also contraindicated in patients with a history of non-traumatic rhabdomyolysis or hyperpyrexia and confusion possibly related to medication (see PRECAUTIONS: Events Reported With Dopaminergic Therapy).

WARNINGS: (SEE BOXED WARNING) Because of the risk of potentially fatal, acute fulminant liver failure, TASMAR (tolcapone) should ordinarily be used in patients with Parkinson's disease on l-dopa/carbidopa who are experiencing symptom fluctuations and are not responding satisfactorily to or are not appropriate candidates for other adjunctive therapies (see INDICATIONS and DOSAGE AND ADMINISTRATION sections).

Because of the risk of liver injury and because TASMAR, when it is effective, provides an observable symptomatic benefit, the patient who fails to show substantial clinical benefit within 3 weeks of initiation of treatment, should be withdrawn from TASMAR.

TASMAR therapy should not be initiated if the patient exhibits clinical evidence of liver disease or two SGPT/ALT or SGOT/AST values greater than the upper limit of normal. Patients with severe dyskinesia or dystonia should be treated with caution (see PRECAUTIONS: Rhabdomyolysis).

Patients who develop evidence of hepatocellular injury while on TASMAR and are withdrawn from the drug for any reason may be at increased risk for liver injury if TASMAR is reintroduced. Accordingly, such patients should not ordinarily be considered for retreatment.

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 TASMAR and a non-selective MAO inhibitor (eg, phenelzine and tranylcypromine) would result in inhibition of the majority of the pathways responsible for normal catecholamine metabolism. For this reason, patients should ordinarily not be treated concomitantly with TASMAR and a non-selective MAO inhibitor.

Tolcapone can be taken concomitantly with a selective MAO-B inhibitor (eg, selegiline).

PRECAUTIONS: Hypotension/Syncope: Dopaminergic therapy in Parkinson's disease patients has been associated with orthostatic hypotension. Tolcapone enhances levodopa bioavailability and, therefore, may increase the occurrence of orthostatic hypotension. In TASMAR clinical trials, orthostatic hypotension was documented at least once in 8%, 14% and 13% of the patients treated with placebo, 100 mg and 200 mg TASMAR tid, respectively. A total of 2%, 5% and 4% of the patients treated with placebo, 100 mg and 200 mg TASMAR tid, respectively, reported orthostatic symptoms at some time during their treatment and also had at least one episode of orthostatic hypotension documented (however, the episode of orthostatic symptoms itself was invariably not accompanied by vital sign measurements). Patients with orthostasis at baseline were more likely than patients without symptoms to have orthostatic hypotension during the study, irrespective of treatment group. In addition, the effect was greater in tolcapone-treated patients than in placebo-treated patients. Baseline treatment with dopamine agonists or selegiline did not appear to increase the likelihood of experiencing orthostatic hypotension when treated with TASMAR. Approximately 0.7% of the patients treated with TASMAR (5% of patients who were documented to have had at least one episode of orthostatic hypotension) eventually withdrew from treatment due to adverse events presumably related to hypotension.

In controlled Phase 3 trials, approximately 5%, 4% and 3% of tolcapone 200 mg tid, 100 mg tid and placebo patients, respectively, reported at least one episode of syncope. Reports of syncope were generally more frequent in patients in all three 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) compared to patients who did not have any episodes of documented hypotension.

Diarrhea: Typically, diarrhea presents 6 to 12 weeks after tolcapone is started, but it may appear as early as 2 weeks and as late as many months after the initiation of treatment. Clinical trial data suggested that diarrhea associated with tolcapone use may sometimes be associated with anorexia (decreased appetite).

No consistent description of tolcapone-induced diarrhea has been derived from clinical trial data, and the mechanism of action is currently unknown.

It is recommended that all cases of persistent diarrhea should be followed up with an appropriate work-up (including occult blood samples).

Hallucinations: In general, hallucinations present shortly after the initiation of therapy with tolcapone (typically within the first 2 weeks). Clinical trial data suggest that hallucinations associated with tolcapone use may be responsive to levodopa dose reduction. Patients whose hallucinations resolved had a mean levodopa dose reduction of 175 mg to 200 mg (20% to 25%) after the onset of the hallucinations. Hallucinations were commonly accompanied by confusion and to a lesser extent sleep disorder (insomnia) and excessive dreaming.

Dyskinesia: TASMAR may potentiate the dopaminergic side effects of levodopa and may 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 0.0%, 0.3% and 1.0% for placebo, 100 mg and 200 mg TASMAR tid, respectively.

Rhabdomyolysis: Cases of severe rhabdomyolysis, with one case of multiorgan system failure rapidly progressing to death, have been reported. The complicated nature of these cases makes it impossible to determine what role, if any, TASMAR played in their pathogenesis. Severe prolonged motor activity including dyskinesia may account for rhabdomyolysis. Some cases, however, included fever, alteration of consciousness and muscular rigidity. It is possible, therefore, that the rhabdomyolysis may be a result of the syndrome described in Hyperpyrexia and Confusion (see PRECAUTIONS: Events Reported With Dopaminergic Therapy).

Renal Impairment: No dosage adjustment is needed in patients with mild to moderate renal impairment, however, patients with severe renal impairment should be treated with caution (see DOSAGE AND ADMINISTRATION).

Renal Toxicity: When rats were dosed daily for 1 or 2 years (exposures 6 times the human exposure or greater) there was a high incidence of proximal tubule cell damage consisting of degeneration, single cell necrosis, hyperplasia, karyocytomegaly and atypical nuclei. 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 it has been speculated that these toxicities may occur as the result of a species-specific mechanism, experiments which would confirm that theory have not been conducted.

Hepatic Impairment: Because of the risk of liver injury, TASMAR therapy should not be initiated in any patient with liver disease. For similar reasons, treatment should not be initiated in patients who have two SGPT/ALT or SGOT/AST values greater than the upper limit of normal (see BOXED WARNING) or any other evidence of hepatocellular dysfunction.

Hematuria: The rates of hematuria in placebo-controlled trials were approximately 2%, 4% and 5% in placebo, 100 mg and 200 mg TASMAR tid, respectively. The etiology of the increase with TASMAR has not always been explained (for example, by urinary tract infection or coumadin therapy). In placebo-controlled trials in the United States (N=593) rates of microscopically confirmed hematuria were approximately 3%, 2% and 2% in placebo, 100 mg and 200 mg TASMAR tid, respectively.

Events Reported With Dopaminergic Therapy: The events listed below are 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. While cases of Hyperpyrexia and Confusion have been reported in association with tolcapone withdrawal (see paragraph below), the expected incidence of fibrotic complications is so low that even if tolcapone caused these complications at rates similar to those attributable to other dopaminergic therapies, it is unlikely that even a single example would have been detected in a cohort of the size exposed to tolcapone.

Hyperpyrexia and Confusion: In clinical trials, four cases of a symptom complex resembling the neuroleptic malignant syndrome characterized by elevated temperature, muscular rigidity and altered consciousness, similar to that reported in association with the rapid dose reduction or withdrawal of other dopaminergic drugs, have been reported in association with the abrupt withdrawal or lowering of the dose of tolcapone. In 3 of these cases, CPK was elevated as well. One patient died, and the other 3 patients recovered over periods of approximately 2, 4 and 6 weeks. Rare cases of this symptom complex have been reported during marketed use. These cases are of a complicated nature including the concomitant administration of several medications affecting brain monoaminergic (ie, MAO-I, tricyclic and selective serotonin reuptake inhibitors) and anticholinergic systems. It is difficult, therefore, to determine what role, if any, TASMAR played in the pathogenesis. It may therefore be prudent to be particularly cautious if several concomitant medications of these types are used.

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 drugs (eg, tolcapone) that increase dopaminergic activity can cause them is unknown.

Three cases of pleural effusion, one with pulmonary fibrosis, occurred during clinical trials. These patients were also on concomitant dopamine agonists (pergolide or bromocriptine) and had a prior history of cardiac disease or pulmonary pathology (non-malignant lung lesion).

Information for Patients: Patients should be instructed to take TASMAR only as prescribed.

TASMAR should not be used by patients until there has been a complete discussion of the risks and the patient has provided written informed consent (see PATIENT CONSENT section).

Patients should be informed of the clinical signs and symptoms that suggest the onset of hepatic injury (persistent nausea, fatigue, lethargy, anorexia, jaundice, dark urine, pruritus, and right upper quadrant tenderness) (see WARNINGS). If symptoms of hepatic failure occur, patients should be advised to contact their physician immediately.

Patients should be informed that hallucinations can occur.

Patients should be informed of the need to have regular blood tests to monitor liver enzymes.

Patients should be advised that they may develop postural (orthostatic) hypotension with or without symptoms such as dizziness, nausea, syncope, and sometimes sweating. Hypotension may occur more frequently during initial therapy. 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 TASMAR.

Patients should be advised that they should neither drive a car nor operate other complex machinery until they have gained sufficient experience on TASMAR 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 TASMAR.

Patients should be informed that nausea may occur, especially at the initiation of treatment with TASMAR.

Patients should be advised of the possibility of an increase in dyskinesia and/or dystonia.

Although TASMAR has not been shown to be teratogenic in animals, it is always given in conjunction with levodopa/carbidopa, which is known to cause visceral and skeletal malformations in the rabbit. Accordingly, patients should be advised to notify their physicians if they become pregnant or intend to become pregnant during therapy (see PRECAUTIONS: Pregnancy).

Tolcapone is excreted into maternal milk in rats. Because of the possibility that tolcapone may be excreted into human maternal milk, patients should be advised to notify their physicians if they intend to breastfeed or are breastfeeding an infant.

Laboratory Tests: **Although a program of frequent laboratory monitoring for evidence of hepatocellular injury is deemed essential, it is not clear that baseline and periodic monitoring of liver enzymes will prevent the occurrence of fulminant liver failure. However, it is generally believed that early detection of drug-induced hepatic injury along with immediate withdrawal of the suspect drug enhances the likelihood for recovery. It is also widely held, without a robust body of evidence, that patients with preexisting hepatic disease are more vulnerable to hepatotoxins. Accordingly, the following liver monitoring program is recommended.**

Before starting treatment with TASMAR, the physician should conduct appropriate tests to exclude the presence of liver disease. In patients determined to be appropriate candidates for treatment with TASMAR, serum glutamic-pyruvic transaminase (SGPT/ALT) and serum glutamic-oxaloacetic transaminase (SGOT/AST) levels should be determined at baseline and then every 2 weeks for the first year of therapy, every 4 weeks for the next 6 months and then every 8 weeks thereafter.

If the dose is increased to 200 mg tid (see DOSAGE AND ADMINISTRATION section), liver enzyme monitoring should take place before increasing the dose and then be reinitiated at the frequency above.

TASMAR should be discontinued if SGPT/ALT or SGOT/AST exceeds the upper limit of normal or if clinical signs and symptoms suggest the onset of hepatic failure (persistent nausea, fatigue, lethargy, anorexia, jaundice, dark urine, pruritus, and right upper quadrant tenderness).

Special Populations: TASMAR therapy should not be initiated if the patient exhibits clinical evidence of active liver disease or two SGPT/ALT or SGOT/AST values greater than the upper limit of normal. Patients with severe dyskinesia or dystonia should be treated with caution (see PRECAUTIONS: Rhabdomyolysis). Patients with severe renal impairment should be treated with caution (see INDICATIONS, DOSAGE AND ADMINISTRATION, BOXED WARNING and WARNINGS).

Drug Interactions: *Protein Binding:* Although tolcapone is highly protein bound, in vitro studies have shown that tolcapone at a concentration of 50 µg/mL did not displace other highly protein-bound drugs from their binding sites at therapeutic concentrations. The experiments included warfarin (0.5 to 7.2 µg/mL), phenytoin (4.0 to 38.7 µg/mL), tolbutamide (24.5 to 96.1 µg/mL) and digitoxin (9.0 to 27.0 µg/mL).

Drugs Metabolized by Catechol-O-Methyltransferase (COMT): Tolcapone may influence the pharmacokinetics of drugs metabolized by COMT. However, no effects were seen on the pharmacokinetics of the COMT substrate carbidopa. The effect of tolcapone on the pharmacokinetics of other drugs of this class such as alpha-methyldopa, dobutamine, isoproterenol, and isoxsuprine has not been evaluated. A dose reduction of such compounds should be considered when they are coadministered with tolcapone.

Effect of Tolcapone on the Metabolism of Other Drugs: In vitro experiments have been performed to assess the potential of tolcapone to interact with isoenzymes of cytochrome P450 (CYP). No relevant interactions with substrates for CYP 2A6 (coumadin), CYP 1A2 (caffeine), CYP 3A4 (midazolam, terfenadine, cyclosporine), CYP 2C19 (S-mephenytoin) and CYP 2D6 (desipramine) were observed in vitro. The absence of an interaction with desipramine, a drug metabolized by cytochrome P450 2D6, was also confirmed in an in vivo study where tolcapone did not change the pharmacokinetics of desipramine.

Due to its affinity to cytochrome P450 2C9 in vitro, tolcapone may interfere with drugs, whose clearance is dependent on this metabolic pathway such as tolbutamide and warfarin. However, in an in vivo interaction study, tolcapone did not change the pharmacokinetics of tolbutamide. Therefore, clinically relevant interactions involving cytochrome P450 2C9 appear unlikely. Similarly, tolcapone did not affect the pharmacokinetics of desipramine, a drug metabolized by cytochrome P450 2D6, indicating that interactions with drugs metabolized by that enzyme are unlikely. Since clinical information is limited regarding the combination of warfarin and tolcapone, coagulation parameters should be monitored when these two drugs are coadministered.

Drugs That Increase Catecholamines: Tolcapone did not influence the effect of ephedrine, an indirect sympathomimetic, on hemodynamic parameters or plasma catecholamine levels, either at rest or during exercise. Since tolcapone did not alter the tolerability of ephedrine, these drugs can be coadministered.

When TASMAR was given together with levodopa/carbidopa and desipramine, there was no significant change in blood pressure, pulse rate and plasma concentrations of desipramine. Overall, the frequency of adverse events increased slightly. These adverse events were predictable based on the known adverse reactions to each of the three drugs individually. Therefore, caution should be exercised when desipramine is administered to Parkinson's disease patients being treated with TASMAR and levodopa/carbidopa.

In clinical trials, patients receiving TASMAR/levodopa preparations reported a similar adverse event profile independent of whether or not they were also concomitantly administered selegiline (a selective MAO-B inhibitor).

Pregnancy: Pregnancy Category C. Tolcapone, when administered alone during organogenesis, was not teratogenic at doses of up to 300 mg/kg/day in rats or up to 400 mg/kg/day in rabbits (5.7 times and 15 times the recommended daily clinical dose of 600 mg, on a mg/m² basis, respectively). In rabbits, however, an increased rate of abortion occurred at a dose of 100 mg/kg/day

(3.7 times the daily clinical dose on a mg/m² basis) or greater. Evidence of maternal toxicity (decreased weight gain, death) was observed at 300 mg/m² in rats and 400 mg/kg in rabbits. When tolcapone was administered to female rats during the last part of gestation and throughout lactation, decreased litter size and impaired growth and learning performance in female pups were observed at a dose of 250/150 mg/kg/day (dose reduced from 250 to 150 mg/kg/day during late gestation due to high rate of maternal mortality; equivalent to 4.8/2.9 times the clinical dose on a mg/m² basis).

Tolcapone is always given concomitantly with levodopa/carbidopa, which is known to cause visceral and skeletal malformations in rabbits. The combination of tolcapone (100 mg/kg/day) with levodopa/carbidopa (60/20 mg/kg/day) produced an increased incidence of fetal malformations (primarily external and skeletal digit defects) compared to levodopa/carbidopa alone when pregnant rabbits were treated throughout organogenesis. Plasma exposures to tolcapone (based on AUC) were 0.5 times the expected human exposure, and plasma exposures to levodopa were 6 times higher than those in humans under therapeutic conditions. In a combination embryo-fetal development study in rats, fetal body weights were reduced by the combination of tolcapone (10, 30 and 50 mg/kg/day) and levodopa/carbidopa (120/30 mg/kg/day) and by levodopa/carbidopa alone. Tolcapone exposures were 0.5 times expected human exposure or greater; levodopa exposures were 21 times the expected human exposure or greater. The high dose of 50 mg/kg/day of tolcapone given alone was not associated with reduced fetal body weight (plasma exposures of 1.4 times the expected human exposure).

There is no experience from clinical studies regarding the use of TASMAR in pregnant women. Therefore, TASMAR should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Women: In animal studies, tolcapone was excreted into maternal rat milk.

It is not known whether tolcapone is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when tolcapone is administered to a nursing woman.

Pediatric Use: There is no identified potential use of tolcapone in pediatric patients.

ADVERSE REACTIONS: Cases of severe hepatocellular injury, including fulminant liver failure resulting in death, have been reported in postmarketing use. As of October 1998, 3 cases of fatal fulminant hepatic failure have been reported from approximately 60,000 patients providing about 40,000 patient years of worldwide use. This incidence may be 10- to 100-fold higher than the background incidence in the general population.

The imprecision of the estimated increase is due to uncertainties about the base rate and the actual number of cases occurring in association with TASMAR. The incidence of idiopathic potentially fatal fulminant hepatic failure (ie, not due to viral hepatitis or alcohol) is low. One estimate, based upon transplant registry data, is approximately 1/1,000,000 patients per year in the United States. Whether this estimate is an appropriate basis for estimating the increased risk of liver failure among TASMAR users is uncertain. TASMAR users, for example, differ in age and general health status from candidates for liver transplantation. Similarly, underreporting of cases may lead to significant underestimation of the increased risk associated with the use of TASMAR.

During the premarketing development of tolcapone, two distinct patient populations were studied, patients with end-of-dose wearing-off phenomena and patients with stable responses to levodopa therapy. All patients received concomitant treatment with levodopa preparations, however, and were similar in other clinical aspects. Adverse events are, therefore, shown for these two populations combined.

The most commonly observed adverse events (>5%) in the double-blind, placebo-controlled trials (N=682) associated with the use of TASMAR (not seen at an equivalent frequency among the placebo-treated patients) were dyskinesia, nausea, sleep disorder, dystonia, dreaming, excessive, anorexia, cramps muscle, orthostatic complaints, somnolence, diarrhea, confusion, dizziness, headache, hallucination, vomiting, constipation, fatigue, upper respiratory tract infection, falling, sweating increased, urinary tract infection, xerostomia, abdominal pain, urine discoloration.

Effects of Gender and Age on Adverse Reactions: Experience in clinical trials have suggested that patients greater than 75 years of age may be more likely to develop hallucinations than patients less than 75 years of age, while patients over 75 may be less likely to develop dystonia. Females may be more likely to develop somnolence than males.

Other Adverse Events Observed During All Trials in Patients With Parkinson's Disease: TASMAR has been administered in 1536 patients with Parkinson's disease 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 adverse events were grouped into a smaller number of standardized categories using COSTART dictionary terminology. These categories are used in the listing below.

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 TASMAR.

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, infrequent adverse events are defined as those occurring in between 1/100 and 1/1000 patients; and rare adverse events are defined as those occurring in fewer than 1/1000 patients.

Nervous System — *frequent:* depression, hypesthesia, tremor, speech disorder, vertigo, emotional lability; *infrequent:* neuralgia, amnesia, extrapyramidal syndrome, hostility, libido increased, manic reaction, nervousness, paranoid reaction, cerebral ischemia, cerebrovascular accident, delusions, libido decreased, neuropathy, spathy, choreoathetosis, myoclonus, psychosis, thinking abnormal, twitching; *rare:* antisocial reaction, delirium, encephalopathy, hemiplegia, meningitis.

Digestive System — *frequent:* tooth disorder; *infrequent:* dysphagia, gastrointestinal hemorrhage, gastroenteritis, mouth ulceration, increased salivation, abnormal stools, esophagitis, cholelithiasis, colitis, tongue disorder, rectal disorder; *rare:* cholecystitis, duodenal ulcer, gastrointestinal carcinoma, stomach atony.

Body as a Whole — *frequent:* flank pain, accidental injury, abdominal pain, infection; *infrequent:* hernia, pain, allergic reaction, cellulitis, infection fungal, viral infection, carcinoma, chills, infection bacterial, neoplasm, abscess, face edema; *rare:* death.

Cardiovascular System — *frequent:* palpitation; *infrequent:* hypertension, vasodilation, angina pectoris, heart failure, atrial fibrillation, tachycardia, migraine, aortic stenosis, arrhythmia, arteritis, postural hypotension, bradycardia, cerebral hemorrhage, coronary artery disorder, heart arrest, myocardial infarct, myocardial ischemia, pulmonary embolus; *rare:* arteriosclerosis, cardiovascular disorder, pericardial effusion, thrombosis.

Musculoskeletal System — *frequent:* myalgia; *infrequent:* tenosynovitis, arthritis, joint disorder.

Urogenital System — *frequent:* urinary incontinence, impotence; *infrequent:* prostatic disorder, dysuria, nocturia, polyuria, urinary retention, urinary tract disorder, hematuria, kidney calculus, prostatic carcinoma, breast neoplasm, oliguria, uterine atony, uterine disorder, vaginitis; *rare:* bladder calculus, ovarian carcinoma, uterine hemorrhage.

Respiratory System — *frequent:* bronchitis, pharyngitis; *infrequent:* cough increased, rhinitis, asthma, epistaxis, hyperventilation, laryngitis, hiccup; *rare:* apnea, hypoxia, lung edema.

Skin and Appendages — *frequent:* rash; *infrequent:* herpes zoster, pruritus, seborrhea, skin discoloration, eczema, erythema multiforme, skin disorder, furunculosis, herpes simplex, urticaria.

Special Senses — *frequent:* tinnitus; *infrequent:* diplopia, ear pain, eye hemorrhage, eye pain, lacrimation disorder, otitis media, parosmia; *rare:* glaucoma.

Metabolic and Nutritional — *infrequent:* edema, hypercholesteremia, thirst, dehydration.

Hemic and Lymphatic System — *infrequent:* anemia; *rare:* leukemia, thrombocytopenia.

Endocrine System — *infrequent:* diabetes mellitus.

Unclassified — *infrequent:* surgical procedure.

DRUG ABUSE AND DEPENDENCE: Tolcapone is not a controlled substance.

Studies conducted in rats and monkeys did not reveal any potential for physical or psychological dependence. Although clinical trials have not revealed any evidence of the potential for abuse, tolerance or physical dependence, systematic studies in humans designed to evaluate these effects have not been performed.

OVERDOSAGE: The highest dose of tolcapone administered to humans was 800 mg tid, with and without levodopa/carbidopa coadministration. This was in a 1-week study in elderly, healthy volunteers. The peak plasma concentrations of tolcapone at this dose were on average 30 µg/mL (compared to 3 µg/mL and 6 µg/mL with 100 mg and 200 mg tolcapone, respectively). Nausea, vomiting and dizziness were observed, particularly in combination with levodopa/carbidopa.

The threshold for the lethal plasma concentration for tolcapone based on animal data is >100 µg/mL. Respiratory difficulties were observed in rats at high oral (gavage) and intravenous doses and in dogs with rapid (bolus) injected intravenous doses.

Management of Overdose: Hospitalization is advised. General supportive care is indicated. Based on the physicochemical properties of the compound, hemodialysis is unlikely to be of benefit.

DOSAGE AND ADMINISTRATION: Because of the risk of potentially fatal, acute fulminant liver failure, TASMAR (tolcapone) should ordinarily be used in patients with Parkinson's disease on l-dopa/carbidopa who are experiencing symptom fluctuations and are not responding satisfactorily to or are not appropriate candidates for other adjunctive therapies (see INDICATIONS and DOSAGE AND ADMINISTRATION sections).

Because of the risk of liver injury and because TASMAR when it is effective provides an observable symptomatic benefit, the patient who fails to show substantial clinical benefit within 3 weeks of initiation of treatment, should be withdrawn from TASMAR.

TASMAR therapy should not be initiated if the patient exhibits clinical evidence of liver disease or two SGPT/ALT or SGOT/AST values greater than the upper limit of normal. Patients with severe dyskinesia or dystonia should be treated with caution (see PRECAUTIONS: Rhabdomyolysis).

Patients who develop evidence of hepatocellular injury while on TASMAR and are withdrawn from the drug for any reason may be at increased risk for liver injury if TASMAR is reintroduced. Accordingly such patients should not ordinarily be considered for retreatment.

Treatment with TASMAR should always be initiated at a dose of 100 mg tid, always as an adjunct to levodopa/carbidopa therapy. The recommended daily dose of TASMAR is also 100 mg tid. In clinical trials, elevations in ALT occurred more frequently at the dose of 200 mg tid. While it is unknown whether the risk of acute fulminant liver failure is increased at the 200-mg dose, it would be prudent to use 200 mg only if the anticipated incremental clinical benefit is justified (see BOXED WARNING, WARNINGS, PRECAUTIONS: Laboratory Tests). If a patient fails to show the expected incremental benefit on the 200-mg dose after a total of 3 weeks of treatment (regardless of dose), TASMAR should be discontinued.

In clinical trials, the first dose of the day of TASMAR was always taken together with the first dose of the day of levodopa/carbidopa, and the subsequent doses of TASMAR were given approximately 6 and 12 hours later.

In clinical trials, the majority of patients required a decrease in their daily levodopa dose if their daily dose of levodopa was >600 mg or if patients had moderate or severe dyskinesias before beginning treatment.

To optimize an individual patient's response, reductions in daily levodopa dose may be necessary in clinical trials; the average reduction in daily levodopa dose was about 30% in those patients requiring a levodopa dose reduction. (Greater than 70% of patients with levodopa doses above 800 mg daily required such a reduction.)

TASMAR can be combined with both the immediate and sustained release formulations of levodopa/carbidopa.

TASMAR may be taken with or without food.

Patients With Impaired Hepatic Function: TASMAR therapy should not be initiated if any patient with liver disease or two SGPT/ALT or SGOT/AST values greater than the upper limit of normal. (See BOXED WARNING and WARNINGS).

Patients With Impaired Renal Function: No dose adjustment of TASMAR is recommended for patients with mild to moderate renal impairment. However, patients with severe renal impairment should be treated with caution. The safety of tolcapone has not been examined in subjects who had creatinine clearance less than 25 mL/min.

Withdrawing Patients From TASMAR: As with any dopaminergic drug, withdrawal or abrupt reduction in the TASMAR dose may lead to emergence of signs and symptoms of Parkinson's disease or Hyperpyrexia and Confusion, a syndrome complex resembling the neuroleptic malignant syndrome (see PRECAUTIONS: Events Reported With Dopaminergic Therapy). If a decision is made to discontinue treatment with TASMAR, then it is recommended to closely monitor the patient and adjust 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 TASMAR has not been systematically evaluated. As the duration of COMT inhibition with TASMAR is generally 5 to 6 hours on average, decreasing the frequency of dosage to twice or once a day may not in itself prevent withdrawal effects.

Manufactured for
VALEANT
Pharmaceuticals International
3300 Hyland Ave.
Costa Mesa, CA 92626

Manufactured by
Hoffman- La Roche
Nutley, NJ 07110

PATIENT CONSENT

TASMAR SHOULD NOT BE USED BY PATIENTS UNTIL THERE HAS BEEN A COMPLETE DISCUSSION OF THE RISKS AND WRITTEN INFORMED CONSENT HAS BEEN OBTAINED.

IMPORTANT INFORMATION AND WARNING

Reports of potentially life-threatening cases of severe hepatocellular injury, including fulminant liver failure resulting in death, have been reported in association with use of TASMAR.

PATIENT CONSENT

My, _____, treatment with TASMAR has been personally described to me by Dr._____.

The following points of information, among others, have been specifically discussed and made clear and I have had the opportunity to ask any questions concerning this information:

1 . I ,

_____

(patient's name)

understand that TASMAR is used to treat certain types of patients with Parkinson's disease and my physician has told me that I am this type of patient.

Initials: _____

2. I understand that there is a serious risk that I could develop severe liver failure, which may be potentially fatal, by using TASMAR.

Initials: _____

3. I understand that there are no laboratory tests that will predict if I am at an increased risk for fatal liver failure.

Initials: _____

4. I understand that I should have the recommended blood work before my treatment with TASMAR is begun or continued and every 2 weeks for the first year, then every 4 weeks for the next 6 months, and then every 8 weeks thereafter while taking TASMAR. I understand that although this blood work may help detect if I develop liver failure it may do so only after significant, irreversible and potentially fatal damage has already occurred.

Initials: _____

5. I understand that I must immediately report any unusual symptoms to Dr._____ and be especially aware of persistent nausea, fatigue, lethargy, decreased appetite, jaundice (yellowing of skin or the whites of the eyes), dark urine, itchiness or right-sided abdominal pain.

Initials: _____

I now authorize Dr. _____ to begin my treatment with TASMAR; OR, if my treatment has already begun with TASMAR, to continue such treatment.

Patient/Caretaker_____

Address_____

_____

Telephone_____

PHYSICIAN STATEMENT:

I have fully explained to the patient, _____, the nature and purpose of the treatment with TASMAR (tolcapone) and the potential risks associated with that treatment. I have asked the patient if he/she has any questions regarding this treatment or the risks and have answered those questions to the best of my ability. I also acknowledge that I have read and understand the prescribing information listed above.

_____

Physician Date

NOTE TO PHYSICIAN: It is strongly recommended that you retain a signed copy of the informed consent with the patient's medical records.

SUPPLY OF PATIENT CONSENT FORMS:

A supply of "Patient Consent" forms as printed above, is available, free of charge, from your local Valeant representative, or may be obtained by calling 1-800-321-4576. Permission to use the above Patient Consent by photocopy reproduction is also hereby granted by Valeant Pharmaceuticals International.

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MDS Exhibit and Information Booth

Location: Lobby Area, Third Floor, Marriott

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 Booth located in the third floor lobby area of the New Orleans Marriott during the following hours:

Friday, March 4 3:00 p.m. to 8:30 p.m.
Saturday, March 5 8:00 a.m. to 5:00 p.m.
Sunday, March 6 8:00 a.m. to 5:00 p.m.
Monday, March 7 8:00 a.m. to 5:00 p.m.
Tuesday, March 8 8:00 a.m. to 5:00 p.m.

International Congress Registration and Venue

Badges

All International Congress attendees should have received 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
Black = Staff

Dates

March 5-8, 2005

Hotel Information

New Orleans Marriott

555 Canal Street
New Orleans, LA 70130 USA
Telephone: +1 504-581-1000
Fax: +1 504-523-6755
Internet: www.neworleansmarriott.com

The New Orleans Marriott serves as the headquarters hotel for the 9th International Congress. It is located on the edge of the historic French Quarter and within walking distance to the French Market, Riverwalk shopping, Bourbon Street and the Aquarium of the Americas. This 41-floor hotel has a health club and sauna, an on-site restaurant, laundry valet, full business center, concierge service, 24-hour room service and more.

Sheraton New Orleans

500 Canal Street
New Orleans, LA 70130 USA
Telephone: +1 504-525-2500
Fax: +1 504-561-0178
Internet: www.sheratonneworleans.com

The Sheraton New Orleans serves as a second venue for the 9th International Congress, located approximately one block south of the New Orleans Marriott. This hotel borders the historic French Quarter and is just steps away from several attractions such as the trendy Warehouse Arts District, Aquarium of the Americas, Harrah's Casino, a streetcar ride to the beautiful Garden District and many world renowned restaurants. The Sheraton recently completed a $25 million renovation that includes Sweet Sleeper mattresses and bedding and leather chairs. Other amenities include a fitness center with 24-hour access and spa amenities, concierge service, business center, 24-hour room service and more.

Holiday Inn French Quarter

124 Royal Street
New Orleans, LA 70130 USA
Telephone: +1 504-529-7211
Fax: +1 504-566-1127
Internet: www.holidayinnfrenchquarter.com

The Holiday Inn French Quarter is providing guestrooms for International Congress delegates. This hotel is located one block and a half north of the New Orleans Marriott, is only one block from the legendary Bourbon Street, and minutes from world famous shopping, restaurants and nightspots. This hotel has 347 beautifully decorated guestrooms, plus spacious King Leisure rooms featuring a sitting area and sofa sleeper. Amenities include an indoor heated pool, sun deck, and a complimentary exercise facility.

Language

The official language of the International Congress is English.

Registration Desk

Location: Lobby Area, Second Floor, Marriott

Name badges, session tickets, special event tickets and International Congress registration bags can be collected at the International Congress Registration Desk located in the Preservation Hall Lobby of the New Orleans Marriott.

Registration Desk Hours

Friday, March 4	3:00 p.m. to 8:30 p.m.
Saturday, March 5	7:00 a.m. to 8:30 p.m.
Sunday, March 6	7:00 a.m. to 7:30 p.m.
Monday, March 7	7:00 a.m. to 6:00 p.m.
Tuesday, March 8	7:00 a.m. to 9:00 p.m.

Venues

New Orleans Marriott

555 Canal Street
New Orleans, LA 70130 USA
Scientific Sessions, Registration, Exhibits, Posters, Internet Café

Sheraton New Orleans

500 Canal Street
New Orleans, LA 70130 USA
Scientific Sessions

The New Orleans Marriott and the Sheraton New Orleans are located across the street from each other on Canal Street.

International Congress Information

Abstract Volume

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

Abstracts-On-Disk™

All abstracts published in the supplement to the MDS Journal are available by Abstracts-On-Disk™, which has been sponsored by MDS and supported by an unrestricted educational grant from Medtronic Neurological. To obtain a copy, please visit the Medtronic Booth #300 and exchange the voucher located in your registration bag.

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 9th International Congress of Parkinson's Disease and Movement Disorders includes clinicians, researchers, post-doctoral fellows, medical residents and medical school students with an interest in the current research and approaches for the diagnosis and treatment of Movement Disorders.

Availability of CME Credit

The *Movement* Disorder Society is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

The Scientific Program of the 9th International Congress of Parkinson's Disease and Movement Disorders 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 38.25 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 their participation in 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 second floor of the New Orleans Marriott or placed in one of the drop boxes located throughout the Marriott.

Participants can find CME Request Forms for the International Congress in their International Congress registration bags. International Congress registration bags should have been collected upon registering at the Registration Desk on the second floor of the New Orleans Marriott. Additional CME Request Forms can be obtained 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) of 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.

International Congress Information

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.

Evaluations

Please take time to complete the evaluation form 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, the evaluation drop boxes, the MDS Registration Desk or the CME Desk.

Exhibition

Location: Preservation Hall and LaGalerie, Second Floor, Marriott

Please allow adequate time in your daily schedule to visit the exhibits located in Preservation Hall and La Galerie of the New Orleans Marriott. 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:

Saturday, March 5	9:15 p.m. to 11:00 p.m.
Sunday, March 6	8:00 a.m. to 5:00 p.m.
Monday, March 7	8:00 a.m. to 5:00 p.m.
Tuesday, March 8	8:00 a.m. to 5:00 p.m.

Internet Café

Location: Lobby Area, Third Floor, Marriott

Internet access is available to meeting attendees on the third floor of the New Orleans Marriott. Please limit your Internet use to 15 minutes to allow other attendees use of this service.

MDS Exhibit and Information Booth

Location: Lobby Area, Third Floor, Marriott

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, educational workshops 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 Booth located in the third floor lobby area of the New Orleans Marriott during the following hours:

Friday, March 4	3:00 p.m. to 8:30 p.m.
Saturday, March 5	8:00 a.m. to 5:00 p.m.
Sunday, March 6	8:00 a.m. to 5:00 p.m.
Monday, March 7	8:00 a.m. to 5:00 p.m.
Tuesday, March 8	8:00 a.m. to 5:00 p.m.

MDS History Exhibit

Location: Lobby Area, Third Floor, Marriott

Funded through an unrestricted grant from Cephalon.

Continuing a tradition established by MDS, a history exhibit is on display throughout the duration of the International Congress. This year's exhibit honors the 250th anniversary of James Parkinson's birth, focusing on Parkinson himself and the early history of Parkinson's disease. Original books, manuscripts, letters, photographs, medical artifacts and instruments related to the early history of Parkinson's disease are displayed in glass cases.

The MDS membership has been the primary source for these original artifacts; other items have been loaned from libraries and private collections. Archival films documenting early clinical demonstrations of Parkinson's disease and related disorders, as well as several celebrated neurologists examining Movement Disorder patients, accompany the exhibit.

The MDS History Exhibit will be displayed in the third floor lobby area of the New Orleans Marriott. The hours are as follows:

Friday, March 4	3:00 p.m. to 8:30 p.m.
Saturday, March 5	7:00 a.m. to 5:00 p.m.
Sunday, March 6	7:00 a.m. to 5:00 p.m.
Monday, March 7	7:00 a.m. to 5:00 p.m.
Tuesday, March 8	7:00 a.m. to 5:00 p.m.

No Cameras

Cameras are not permitted in any 9th International Congress educational session or in the poster areas.

International Congress Information

Opening Ceremony and Welcome Reception

Location: Acadia Room, Mardi Gras Ballroom, Preservation Hall, La Galerie, Second and Third Floor, Marriott

The Opening Ceremony will take place on Saturday, March 5 at 8:30 p.m in the Acadia Room. A Welcome Reception will follow immediately after the Opening Ceremony. These events are open to all delegates and registered guests.

Optional Tours Desk

Location: Lobby Area, Second Floor, Marriott

Tours have been arranged by Visit New Orleans.

Please visit the Tours Desk in the Registration Area on the second floor of the New Orleans Marriott to collect your tickets. Additional tour tickets may be purchased at the desk, based on availability.

Press Room

Location: Audubon Room, Fifth Floor, Marriott

Members of the working media receive waived registration fees for the 9th International Congress. Journalists and writers should report to the Press Room with their credentials to register for the International Congress and must wear their name badge for admittance into MDS sessions. The Press Room will be open during the following hours:

Saturday, March 5	8:00 a.m. to 5:00 p.m.
Sunday, March 6	8:00 a.m. to 5:00 p.m.
Monday, March 7	8:00 a.m. to 5:00 p.m.
Tuesday, March 8	8:00 a.m. to 5:00 p.m.

Scientific Sessions

The 2005 Scientific Program incorporates Kickoff Seminars, Plenary and Parallel Sessions, Skills Workshops, Video Sessions and Poster Sessions.

Although the ever popular Kickoff and Plenary Sessions follow a style similar to the 2002 Miami and 2004 Rome International Congresses, Parallel Sessions and Skills Workshops have been newly designed to meet the need for smaller, more focused sessions. These sessions are offered to an audience size of 50-200 participants resulting in greater opportunity for audience participation.

Tickets are required for admission into all Parallel Sessions, Video Sessions and Skills Workshops. There is no additional fee for tickets to these sessions. Please check the On-Site Registration Desk for availability of these tickets.

Platform Presentations

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

Abstract Poster Sessions

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

Poster Session 1
Location: Mardi Gras Ballroom, Third Floor, Marriott
Sunday, March 6
Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:45 p.m. to 2:30 p.m.
Abstracts: 1-187

Poster Session 2
Location: Mardi Gras Ballroom, Third Floor, Marriott
Monday, March 7
Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:30 p.m. to 2:15 p.m.
Abstracts: 188-383

Poster Session 3
Location: Mardi Gras Ballroom, Third Floor, Marriott
Tuesday, March 8
Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:45 p.m. to 2:30 p.m.
Abstracts: 384-592

Speaker Ready Room

Location: Bonaparte Room, Fourth Floor, Marriott

All speakers must check-in to the Speaker Ready Room with presentation materials on the day prior to their scheduled presentation. Equipment is available for faculty to review their presentations. Audiovisual personnel will be available for assistance. The Speaker Ready Room hours are as follows:

Friday, March 4	5:00 p.m. to 8:00 p.m.
Saturday, March 5	7:30 a.m. to 7:00 p.m.
Sunday, March 6	7:30 a.m. to 7:00 p.m.
Monday, March 7	7:30 a.m. to 7:00 p.m.
Tuesday, March 8	7:30 a.m. to 7:00 p.m.

Program-at-a-Glance

	Saturday	Sunday	Monday	Tuesday	
6:30 a.m.	Committee Meetings				6:30 a.m.
7:00 a.m.					7:00 a.m.
		Committee Meetings	MDS Business Meeting	Committee Meetings	
8:00 a.m.	Kickoff Seminars				8:00 a.m.
		Plenary Session	Plenary Session	Plenary Session	
9:00 a.m.					9:00 a.m.
		Marsden Lecture		Fahn Lecture	
10:00 a.m.					10:00 a.m.
		Parallel Sessions		Parallel Sessions	
11:00 a.m.					11:00 a.m.
12:00 p.m.					12:00 p.m.
			Poster Session		
		Poster Session		Poster Session	
1:00 p.m.					1:00 p.m.
2:00 p.m.					2:00 p.m.
			Plenary Session		
		Platform Presentations/ Junior Awards		Highlights Session	
3:00 p.m.					3:00 p.m.
4:00 p.m.		Parallel Sessions		Parallel Sessions	4:00 p.m.
5:00 p.m.					5:00 p.m.
			Skills Workshops		
6:00 p.m.					6:00 p.m.
				Plenary Session	
		Video Sessions			
7:00 p.m.					7:00 p.m.
8:00 p.m.					8:00 p.m.
	Opening Ceremony and Welcome Reception				
9:00 p.m.					9:00 p.m.

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.

GlaxoSmithKline

ORANGE CARD®
from GlaxoSmithKline
1-888-ORANGE-6

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 on 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 0.8 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/m^2 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: P_{450} *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_{min} 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 analyses revealed that estrogens (mainly ethinylestradiol: intake 0.6–3 mg over 4-month to 23-year period) reduced the oral clearance of ropinirole by 36% 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/m^2 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/m^2 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/m^2 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 (L5178Y 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/m^2 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/m^2 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/m^2 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/m^2 basis, respectively). The combined administration of ropinirole (10 mg/kg/day, 8 times the maximum recommended human dose on a mg/m^2 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/m^2 basis). In a perinatal-postnatal study in rats, 10 mg/kg/day (4 times the maximum recommended human dose on a mg/m^2 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. 5%), 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 (2.4%), vomiting (2.4%), confusion (2.4%), nausea (1.9%), hallucinations (1.9%), anxiety (1.9%), and increased sweating (1.4%). 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%),

REQUIP® (ropinirole hydrochloride) Tablets

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%), constipation (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* – hypoglycemia, 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, balanoposthitis, 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, hyperkeratosis, 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 boldfaced 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 10 patients ingesting more than 24 mg/day. The largest overdose reported in premarketing clinical trials was 435 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 87% 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.

GlaxoSmithKline
GlaxoSmithKline
Research Triangle Park, NC 27709

References: 1. Weiner WJ, Shulman LM, Lang AE. Advanced Parkinson's disease. In: *Parkinson's Disease: A Complete Guide for Patients and Families*. Baltimore, Md: The Johns Hopkins University Press; 2001:58-72. **2.** Rascol O, Brooks DJ, Korczyn AD, De Deyn PP, Clarke CE, Lang AE, for the 056 Study Group. A five-year study of the incidence of dyskinesia in patients with early Parkinson's disease who were treated with ropinirole or levodopa. *N Engl J Med*. 2000;342:1484-1491.

Scientific Program ~ Saturday

Saturday, March 5

Kickoff Seminars

Admission to these sessions is by delegate name badge. No ticket is required for admission to Kickoff Sessions.

8:00 a.m. to 9:00 a.m.

1001 Novel delivery of MAO-B inhibition

Location: Carondelet Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Valeant Pharmaceuticals International.

Chair: Kapil D. Sethi
Augusta, GA, USA

Update on clinical studies
Cheryl Waters
New York, NY, USA

Rationale for development
Peter A. LeWitt
Southfield, MI, USA

Panel discussion

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the use of major inhibitors in Parkinson's disease; 2. Explain the use of novel methods of delivery of MAO-B in Parkinson's disease patients, this includes sublingual and patch formulations; 3. Differentiate oral compounds from novel MAO-B delivery.

9:00 a.m. to 11:00 a.m.

1002 Dopamine agonists in the treatment of Parkinson's disease and restless legs syndrome

Location: Acadia Room, Third Floor, Marriott

Supported through an unrestricted educational grant from GlaxoSmithKline.

Chairs: Anthony E. Lang
Toronto, Canada
José A. Obeso
Pamplona, Spain

RLS: epidemiology and disease burden
Cynthia L. Comella
Chicago, IL, USA

RLS: treatment
Diego Garcia Borreguero
Madrid, Spain

PD: agonists and motor complications
Charles H. Adler
Scottsdale, AZ, USA

PD: agonists and neuroprotection
David J. Brooks
Isleworth, United Kingdom

Panel discussion

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the epidemiology, symptomatology and disease burden of restless legs syndrome; 2. Indicate the various treatment options for restless legs syndrome and their outcomes; 3. Recognize the influence of dopamine agonists on the development and management of motor complications as well as their potential effect on disease progression.

11:00 a.m. to 12:00 p.m.

1401 Rescue therapy for Parkinson's disease

Location: Carondelet Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Mylan Bertek Pharmaceuticals Inc.

Chair: Andrew J. Lees
London, United Kingdom

On-off episodes
Peter A. LeWitt
Southfield, MI, USA

Role of apomorphine in treating off episodes
Mark A. Stacy
Durham, NC, USA

Panel discussion

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the phenomena which are included in the rubric of motor fluctuations; 2. Discuss the role of apomorphine as a rescue therapy in Parkinson's disease; 3. Define the indications for the use of subcutaneous apomophine in Parkinson dosing.

12:00 p.m. to 1:30 p.m.

1402 MAO-B inhibitors: back to the future

Location: Acadia Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Lundbeck, Teva Pharmaceutical Industries Ltd, Teva Neuroscience and Eisai.

Chairs: C. Warren Olanow
New York, NY, USA
Anthony H.V. Schapira
London, United Kingdom

Propargylamines
Anthony H.V. Schapira
London, United Kingdom

New clinical data
Werner Poewe
Innsbruck, Austria

Disease modification approaches
Karl D. Kieburtz
Rochester, NY, USA

Panel discussion

Opening Ceremony and Welcome Reception

Location: Acadia Room, Mardi Gras Ballroom, Preservation Hall, La Galerie, Second and Third Floor, Marriott

The Opening Ceremony will take place on Saturday, March 5 at 8:30 p.m. in the Acadia Room on the Third Floor of the Marriott New Orleans. A Welcome Reception will follow immediately after the Opening Ceremony and will take place in the Mardi Gras Ballroom on the Third Floor and the Exhibit Halls on the Second Floor. These events are open to all delegates and registered guests.

Scientific Program ~ Saturday

2:00 p.m. to 3:00 p.m.

1601 Dopamine agonists: new considerations

Location: Carondelet Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Boehringer Ingelheim.

Chairs: Yoshikuni Mizuno
Tokyo, Japan
Matthew B. Stern
Philadelphia, PA, USA

Non-motor features of Parkinson's disease and possible role of dopamine agonists
Matthew B. Stern
Philadelphia, PA, USA

Long-term studies of dopamine agonists and disease modification
Kenneth Marek
New Haven, CT, USA

Panel discussion

Session Objectives: At the conclusion of this session, participants should be able to: 1. Recognize important non-motor features of Parkinson's disease; 2. Discuss current concepts in modifying the long-term progression of Parkinson's disease; 3. Recognize the indications and role of Dopamine agonists in the treatment of Parkinson's disease.

3:00 p.m. to 5:00 p.m.

1602 Levodopa: a new look at an old drug

Location: Acadia Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Novartis/Orion Pharma.

Chairs: Peter Jenner
London, United Kingdom
Fabrizio Stocchi
Roma, Italy

Levodopa: is it toxic?
Stanley Fahn
New York, NY, USA

Levodopa: how does it cause motor complications?
Speaker to be announced

A rationale for the early use of carbidopa/levodopa/entacapone
Fabrizio Stocchi
Roma, Italy

Panel discussion

Session Objectives: At the conclusion of this session, participants should be able to: 1. Understand the debate over whether levodopa is potentially neurotoxic to remaining dopaminergic neurones in Parkinson's disease; 2. Recognize the range of motor complications caused by levodopa and the mechanism responsible for their development; 3. Recognize the therapeutic usefulness of levodopa in the treatment of Parkinson's disease.

5:00 p.m. to 6:00 p.m.

1901 Tolcapone: a non-jaundiced view

Location: Carondelet Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Valeant Pharmaceuticals International.

Chair: Yves Agid
Paris, France

Tolcapone: an update
Ray Watts
Birmingham, AL, USA

Safety considerations
Speaker to be announced

Panel discussion

6:00 p.m. to 8:00 p.m.

1902 Restless legs syndrome: a kickoff

Location: Acadia Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Boehringer Ingelheim.

Chairs: David Rye
Atlanta, GA, USA
Claudia M. Trenkwalder
Kassel, Germany

Etiology and pathogenesis
Richard Allen
Arnold, MD, USA

Clinical features
William Ondo
Houston, TX, USA

Therapeutics
Wayne A. Hening
New York, NY, USA

Panel discussion

Session Objectives: At the conclusion of this session, participants should be able to: 1. Define the clinical diagnostic criteria of restless legs syndrome and recognize them in a patient interview; 2. Discuss the differential diagnosis of patients with restless legs syndrome and explain the major pathophysiological hypothesis of the syndrome. 3. Indicate the need for treatment in RLS and discuss the advantages and problems of pharmacological therapy in RLS patients.

7:00 p.m. to 8:00 p.m.

1903 Botulinum toxins for Movement Disorders 2005

Location: Carondelet Room, Third Floor, Marriott

Supported through an unrestricted educational grant from Allergan.

Chairs: Alfredo Berardelli
Roma, Italy
Cynthia L. Comella
Chicago, IL, USA

Basic pharmacology and immunology
Joseph Jankovic
Houston, TX, USA

Clinical aspects of botulinum toxin
Dirk Dressler
Rostock, Germany

Panel discussion

Scientific Program ~ Sunday

Sunday, March 6, 2005

Plenary Sessions

Admission to these sessions is by delegate name badge. No ticket is required for admission to Plenary Sessions.

8:30 a.m. to 9:30 a.m.

2101 Parkinson's disease

Location: Acadia Room, Third Floor, Marriott

Chair: Anthony E. Lang
Toronto, Canada

8:30 a.m. **Etiology and pathogenesis in Parkinson's disease**
Yoshikuni Mizuno
Tokyo, Japan

9:00 a.m. **Medical therapeutics in Parkinson's disease**
Anthony E. Lang
Toronto, Canada

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the current concepts of etiology of Parkinson's disease including recent genetic discoveries; 2. Explain the current concepts of pathogenesis of cell death in Parkinson's disease; 3. Describe the latest developments in the field of medical therapeutics in Parkinson's disease.

9:30 a.m. to 10:00 a.m.

2102 C. David Marsden Lecture

Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet Room, Third Floor, Marriott

Alim L. Benabid
Grenoble, France

Evaluations

Please take time to complete the evaluation form 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, the evaluation drop boxes, the MDS Registration Desk or the CME Desk.

Parallel Sessions

A ticket is required for admission to these smaller, interactive sessions. Attendance for Parallel Sessions is limited. There are no additional fees for tickets. Delegates that do not have tickets to these sessions, but would like to attend, are asked to check at the On-Site Registration Desk for ticket availability.

10:30 a.m. to 12:45 p.m.

2201 Early untreated Parkinson's disease

Location: Carondelet Room, Third Floor, Marriott

Chair: Karl D. Kieburtz
Rochester, NY, USA

10:30 a.m. **Clinical features (distinguishing PD from atypical parkinsonism)**
Eduardo Tolosa
Barcelona, Spain

11:00 a.m. **Role of neuroimaging in early Parkinson's disease (SWEDD issue)**
Kenneth Marek
New Haven, CT, USA

11:30 a.m. **Early treatment for Parkinson's disease**
Karl D. Kieburtz
Rochester, NY, USA

12:00 p.m. **Discussion**

At the conclusion of this session, participants should be able to: 1. Identify the main clinical features of Parkinson's disease; 2. Recognize early Parkinson's disease from similar disorders; 3. Identify appropriate treatment for early Parkinson's disease.

2202 Tourette syndrome

Location: Balcony K, Fourth Floor, Marriott

Chair: David G. Lichter
Clarence, NY, USA

10:30 a.m. **Clinical overview - including epidemiology, phenomenology, diagnosis**
Anette Schrag
London, United Kingdom

11:00 a.m. **Etiology/pathogenesis/ pathophysiology**
Harvey S. Singer
Baltimore, MD, USA

11:30 a.m. **Therapy - current and experimental**
David G. Lichter
Clarence, NY, USA

12:00 p.m. **Discussion**

At the conclusion of this session, participants should be able to: 1. Describe the epidemiology, phenomenology and diagnostic criteria for Tourette's syndrome; 2. Discuss current concepts of etiology, pathogenesis and pathophysiology of Tourette's syndrome; 3. Describe current therapy and experimental treatments for Tourette's syndrome, including deep brain stimulation.

Scientific Program ~ Sunday

2203 FTD

Location: Balcony L, Fourth Floor, Marriott

Chair: Zbigniew K. Wszolek
Jacksonville, FL, USA

10:30 a.m. **Clinical features, classification and imaging**
Zbigniew K. Wszolek
Jacksonville, FL, USA

11:00 a.m. **Neuropathology and pathogenesis**
Ian Mackenzie
Vancouver, Canada

11:30 a.m. **FTDP17/Molecular biology of tau**
Peter Heutink
Amsterdam, Netherlands

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the clinical features, current classifications and neuroimaging characteristics of fronto-temporal dementias (FTD); 2. Describe the current veins of pathogenesis of FTD and list the neuropathological findings seen in this class of disorders; 3. Recognize the molecular genetics of fronto-temporal dementia and parkinsonism linked to chromosome 17 (FTD P-17), and understand the molecular biology of tau.

2204 Surgery for Movement Disorders

Location: Napoleon A, Third Floor, Sheraton

Chair: Pierre Pollak
Grenoble, France

10:30 a.m. **Surgery for Parkinson's disease**
Jens Volkmann
Kiel, Germany

11:00 a.m. **Surgery for dystonia**
Marie Vidailhet
Paris, France

11:30 a.m. **Surgery for Movement Disorders and future directions**
Pierre Pollak
Grenoble, France

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Define eligible patients with Movement Disorders for surgical therapy and list the various surgical techniques available; 2. Discuss the results of reported clinical trials related to the surgical treatments of Parkinson's disease, dystonia or other Movement Disorders; 3. Discuss the future of developing experimental surgical therapies for Movement Disorders.

2205 The role of neuroimaging in Movement Disorders

Location: Napoleon B, Third Floor, Sheraton

Chair: David J. Brooks
London, United Kingdom

10:30 a.m. **PET/SPECT in Movement Disorders**
David Eidelberg
Manhasset, NY, USA

11:00 a.m. **MR/MRS in Movement Disorders**
Klaus Seppi
Innsbruck, Austria

11:30 a.m. **Practical application of neuroimaging in Movement Disorders**
David J. Brooks
London, United Kingdom

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the possible applications of MRI, PET and SPECT; 2. Discuss the role of imaging in the differential diagnosis of Parkinson's disease; 3. Discuss the role of imaging in the management of Parkinson's disease.

2206 Sleep disorders in Parkinson's disease

Location: Napoleon C, Third Floor, Sheraton

Chair: Claudia M. Trenkwalder
Kassel, Germany

10:30 a.m. **Functional anatomy and physiology of sleep**
David Rye
Atlanta, GA, USA

11:00 a.m. **REM behavior disorder**
Claudia M. Trenkwalder
Kassel, Germany

11:30 a.m. **Excessive daytime sleepiness**
Cynthia L. Comella
Chicago, IL, USA

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the major principles of sleep physiology and explain its pathomechanism in Parkinsonian sleep; 2. Recognize the key features of REM sleep behavior disorder in Parkinson syndromes and explain the major pathophysiological hypothesis; 3. Define excessive daytime sleepiness and discuss the various reasons for daytime sleepiness in Parkinson's disease patients.

Scientific Program ~ Sunday

2207 Magnetic stimulation and Movement Disorders
Location: Napoleon D, Third Floor, Sheraton
Chair: John C. Rothwell
London, United Kingdom

10:30 a.m. **How TMS works and insights it provides into Movement Disorders**
Robert Chen
Toronto, Canada

11:00 a.m. **TMS and plasticity**
Angelo Quartarone
Messina, Italy

11:30 a.m. **TMS as a therapy in Movement Disorders**
John C. Rothwell
London, United Kingdom

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Explain the mechanisms of action of transcranial magnetic stimulation and be aware of the various techniques that can be applied in patients with Movement Disorders; 2. Explain how TMS can be used to address questions of functional plasticity in the motor system and understand how abnormalities in this system contribute to symptoms of dystonia; 3. Discuss the potential of TMS methods as therapeutic options to treat Movement Disorders.

Abstract Sessions

Admission to these sessions is by delegate name badge. No ticket is required for admission to Poster Sessions or Platform Presentations.

12:45 p.m. to 2:30 p.m.
2501 Poster Session 1
Location: Mardi Gras Ballroom, Third Floor, Marriott
Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:45 p.m. to 2:30 p.m.
Abstracts: 1-187

2:30 p.m. to 4:00 p.m.
2502 Platform Presentations/Junior Awards
Location: Acadia Room, Third Floor, Marriott
Please refer to the Platform Presentation flyer in your registration bag for a listing of Platform Presentation Abstracts.

2503 Platform Presentations/Junior Awards
Location: Carondelet Room, Third Floor, Marriott
Please refer to the Platform Presentation flyer in your registration bag for a listing of Platform Presentation Abstracts.

Parallel Sessions

A ticket is required for admission to these smaller, interactive sessions. Attendance for Parallel Sessions is limited. There are no additional fees for tickets. Delegates that do not have tickets to these sessions, but would like to attend, are asked to check at the On-Site Registration Desk for ticket availability.

4:00 p.m. to 6:30 p.m.
2601 Motor complications in Parkinson's disease
Location: Carondelet Room, Third Floor, Marriott
Chair: Andrew J. Lees
London, United Kingdom

4:00 p.m. **Clinical and epidemiological features of motor complications**
Andrew J. Lees
London, United Kingdom

4:30 p.m. **Pathogenesis of motor complications**
José A. Obeso
Pamplona, Spain

5:00 p.m. **Medical and surgical management**
Paul Krack
Grenoble, France
Andrew J. Lees
London, United Kingdom

5:45 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Recognize and categorize motor fluctuations and dyskinesia types; 2. Discuss the epidemiology and risk factors; 3. Discuss medical treatment options.

2602 Pediatric Movement Disorders
Location: Napoleon D, Third Floor, Sheraton
Chair: Jonathan Mink
Rochester, NY, USA

4:00 p.m. **Movement Disorders unique to childhood**
Jonathan Mink
Rochester, NY, USA

4:30 p.m. **Movement Disorders and cerebral palsy**
Terence Sanger
Stanford, CA, USA

5:00 p.m. **Lesch-Nyhan and the effects of early dopamine loss**
Hyder A. Jinnah
Baltimore, MD, USA

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Recognize Movement Disorders that are unique to children; 2. Discuss Movement Disorders commonly seen in children with cerebral palsy and understand factors that contribute to the different manifestations in children as compared to adults; 3. Discuss the different effects of dopamine deficiency in children vs. adults and to understand mechanisms underlying Lesch-Nyhan disease.

Scientific Program ~ Sunday

2603 Essential Tremor

Location: Balcony L, Fourth Floor, Marriott

Chair: Günther Deuschl
Kiel, Germany

4:00 p.m. **Clinical overview - including epidemiology, diagnosis and imaging**
Elan D. Louis
New York, NY, USA

4:30 p.m. **Etiology/pathogenesis**
Günther Deuschl
Kiel, Germany

5:00 p.m. **Therapy - medical and surgical**
Rajesh Pahwa
Kansas City, KS, USA

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the clinical diagnosis and differential diagnosis; 2. Discuss the epidemiology and pathophysiology of essential tremor; 3. Describe current and experimental treatment of essential tremor.

2604 Gene and cell based therapies

Location: Napoleon A, Third Floor, Sheraton

Chair: Olle Lindvall
Lund, Sweden

4:00 p.m. **Update on transplantation - fetal nigral and other dopaminergic cell types**
Patrik Brundin
Lund, Sweden

4:30 p.m. **Update on stem cells**
Olle Lindvall
Lund, Sweden

5:00 p.m. **Update on gene therapy**
Jeffrey H. Kordower
Chicago, IL, USA

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe which conclusions can be drawn from clinical trials with fetal nigral and other dopamineregic cell types; 2. Discuss what will be needed from stem cell-based therapies in order to work better than current fetal cell-based approaches; 3. Describe how various gene therapeutic approaches might be applied to Parkinson's disease patients in order to counter act symptom progression and induce functional recovery.

2605 Pathology and pathogenesis of Parkinson's disease

Location: Napoleon B, Third Floor, Sheraton

Chair: Etienne C. Hirsch
Paris, France

4:00 p.m. **New concepts in pathology of Parkinson's disease**
Glenda M. Halliday
Randwick, Australia

4:30 p.m. **Pathogenesis**
Anthony H.V. Schapira
London, United Kingdom

5:00 p.m. **Mechanisms of cell death**
Etienne C. Hirsch
Paris, France

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the etiology of Parkinson's disease including genetic and environmental causes of the disease; 2. Describe the distribution of the lesion in Parkinson's disease including dopaminergic and non-dopaminergic neurons; 3. Describe the molecular and cellular changes associated with the neuronal death in Parkinson's disease including altered protein processing, mitochondrial dysfunction, oxidative stress and inflammatory processes.

2606 Balance and gait in Parkinson's disease

Location: Napoleon C, Third Floor, Sheraton

Chair: Nir Giladi
Tel Aviv, Israel

4:00 p.m. **Clinical aspects**
Nir Giladi
Tel Aviv, Israel

4:30 p.m. **Physiology and pathophysiology of balance and gait**
Fay B. Horak
Beaverton, OR, USA

5:00 p.m. **Treatment**
Robert Iansek
Cheltenham, Australia

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the different gait disorders, their clinical characteristics and the relations to postural control; 2. Discuss the different assessment tools for locomotion gait disorders and disturbances of postural control; 3. Indicate the possible therapeutic interventional options for the improvement of posture and gait.

Evaluations

Please take time to complete the evaluation form 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, the evaluation drop boxes, the MDS Registration Desk or the CME Desk.

Scientific Program ~ Sunday

2607 Drug induced Movement Disorders
Location: Balcony K, Fourth Floor, Marriott
Chair: Kapil D. Sethi
Augusta, GA, USA

4:00 p.m. **How atypical antipsychotics spare motor side effects-the critical role of the D2 receptor**
Shitij Kapur
Toronto, Canada

4:30 p.m. **Status of tardive dyskinesias in 2005**
Stewart A. Factor
Albany, NY, USA

5:00 p.m. **Other drug induced Movement Disorders (neuroleptic and nonneuroleptic)**
Kapil D. Sethi
Augusta, GA, USA

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify Movement Disorders due to dopaminergic blocking agents; 2. Identify Movement Disorders due to drugs like anti-convulsants and anti-depressants; 3. Effectively manage patients with drug induced Movement Disorders.

Video Sessions

A ticket is required for admission to these smaller, interactive sessions. Attendance for Video Sessions is limited. There are no additional fees for tickets. Delegates that do not have tickets to these sessions, but would like to attend, are asked to check at the On-Site Registration Desk for ticket availability.

6:45 p.m. to 8:15 p.m.

2801 DBS case studies (Session A)
Location: Carondelet Room, Third Floor, Marriott
Boulos-Paul Bejjani
Byblos, Lebanon
Paul Krack
Grenoble, France

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the most appropriate DBS target for different Movement Disorders; 2. Explain the limitations of the technique (Which signs are resistant? What are the problems in follow-up?); 3. Identify the best candidates for surgery.

2802 DBS case studies (Session B)
Location: Balcony K, Fourth Floor, Marriott
Jean-luc Houeto
Poitiers, France
Pierre Pollak
Grenoble, France

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify eligible patients for DBS therapy; 2. Discuss what we can expect from DBS in the surgical treatment of tremors, Parkinson's disease and dystonia; 3. List and understand the problems that can arise with DBS and how to approach solving them.

2803 Psychogenic Movement Disorders
Location: Napoleon C, Third Floor, Sheraton
John G.L. Morris
Sydney, Australia
Anthony E. Lang
Toronto, Canada

Session Objectives: 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.

2804 Paroxysmal Movement Disorders
Location: Napoleon A, Third Floor, Sheraton
Kailash P. Bhatia
London, United Kingdom
Kapil D. Sethi
Augusta, GA, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Recognize primary paroxysmal dyskinesias; 2. Differentiate primary from secondary paroxysmal dyskinesias; 3. Describe management of paroxysmal dyskinesias.

2805 Motor disorders and sleep
Location: Napoleon B, Third Floor, Sheraton
Claudia M. Trenkwalder
Kassel, Germany
Birgit Högl
Innsbruck, Austria

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify the most common motor phenomenons in sleep i.e. sleep talking, sleep walking, bruxism, and tremor, observed in Parkinson's disease and various Movement Disorders; 2. Describe the phenomenology of REM sleep behavior disorder in Parkinson syndromes and explain the major pathophysiological hypothesis; 3. Recognize the typical motor pattern of the restless legs syndrome with periodic limb movements in sleep and wakefulness.

2806 Myoclonus/startle/other jerks
Location: Balcony L, Fourth Floor, Marriott
Philip D. Thompson
North Terrace, Adelaide, Australia
Hiroshi Shibasaki
Bethesda, MD, USA

2807 Dystonia
Location: Napoleon D, Third Floor, Sheraton
Marie Vidailhet
Paris, France
Alberto Albanese
Milano, Italy

Session Objectives: At the conclusion of this session, participants should be able to: 1. Recognize the fundamental clinical features of the Movement Disorder and apply skilled methodology in the clinical evaluation of patients with dystonia; 2. Recognize the different forms of dystonia based on current classification criteria, particularly distinguishing primary and secondary cases; 3. Identify the appropriate laboratory examinations to perform in each case and rank them by appropriateness.

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Scientific Program ~ Monday

Monday, March 7

Plenary Session

Admission to this session is by delegate name badge. No ticket is required for admission to Plenary Sessions.

8:30 a.m. to 12:30 p.m.
3101 Hot topics
Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet Room, Third Floor, Marriott
Chairs: M. Flint Beal
New York, NY, USA
Eldad Melamed
Petah Tiqva, Israel

8:30 a.m. **Update on basal ganglia - organization and physiology**
Thomas Wichmann
Atlanta, GA, USA

9:00 a.m. **Genetic and environmental factors in the etiology of Parkinson's disease**
Christine Klein
Luebeck, Germany

9:30 a.m. **Protein dysfunction and neurodegeneration**
C. Warren Olanow
New York, NY, USA

10:00 a.m. **Break**

10:30 a.m. **Neuroimaging of the basal ganglia**
A. Jon Stoessl
Vancouver, Canada

11:00 a.m. **Clinical trials: The latest**
Werner Poewe
Innsbruck, Austria

11:30 a.m. **Surgical interventions**
Andres M. Lozano
Toronto, Canada

12:00 p.m. **Therapeutics pipeline**
Peter Jenner
London, United Kingdom

Abstract Session

Admission to this session is by delegate name badge. No ticket is required for admission to Poster Sessions or Platform Presentations.

12:30 p.m. to 2:15 p.m.
3501 Poster Session 2
Location: Mardi Gras Ballroom, Third Floor, Marriott
Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:30 p.m. to 2:15 p.m.
Abstracts: 188-383

Plenary Session

Admission to this session is by delegate name badge. No ticket is required for admission to Plenary Sessions.

2:15 p.m. to 5:15 p.m.
3502 Controversies
Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet Room, Third Floor, Marriott
Chairs: Oscar S. Gershanik
Buenos Aires, Argentina
Eldad Melamed
Petah Tiqva, Israel

2:15 p.m. **Trophic factor delivery using pumps and catheters is not viable as a treatment for Parkinson's disease?**
Clive N. Svendsen
Madison, WI, USA
John G. Nutt
Portland, OR, USA

2:45 p.m. **Stem cells are the future for the treatment of Parkinson's disease?**
José A. Obeso
Pamplona, Spain
Speaker to be announced

3:15 p.m. **Parkinson's disease should be treated at the time of diagnosis?**
Yves Agid
Paris, France
Matthew B. Stern
Philadelphia, PA, USA

3:45 p.m. **Post traumatic dystonia is a psychogenic Movement Disorder?**
Joseph Jankovic
Houston, TX, USA
Anthony E. Lang
Toronto, Canada

4:15 p.m. **Double-blind placebo controlled trials are required to establish the efficacy of surgical trials for Movement Disorders?**
Alim L. Benabid
Grenoble, France
C. Warren Olanow
New York, NY, USA

4:45 p.m. **Microrecording is essential for the best outcomes of functional neurosurgery?**
Marwan I. Hariz
London, United Kingdom
Jerrold Lee Vitek
Cleveland, OH, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify controversial issues related to diagnosis and treatment of Parkinson's disease and other Movement Disorders; 2. Identify the pros and cons of different treatment strategies presently being discussed for the management of Parkinson's disease as well as methodological approaches used in the surgical treatment of Parkinson's disease and the validity of current methods of evaluation of the outcome of such procedures; 3. Define whether post-traumatic dystonia is a psychogenic disorder or not.

Scientific Program ~ Monday

Skills Workshops

A ticket is required for admission to these smaller, interactive sessions. Attendance for Skills Workshops is limited. There are no additional fees for tickets. Delegates that do not have tickets to these sessions, but would like to attend, are asked to check at the On-Site Registration Desk for ticket availability.

5:30 p.m. to 7:30 p.m.

3701 Botulinum toxin (Session A)

Location: Napoleon A, Third Floor, Sheraton

Allison Brashear
Indianapolis, IN, USA
Austen Peter Moore
Liverpool, United Kingdom

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the indications, appropriate patient selection, dosing and technique for using botulinum toxin in the treatment of Movement Disorders and spasticity; 2. Understand the differences of each serotype of botulinum toxin and strategies for choosing between them; 3. Discuss how to assess the role of botulinum toxin in new indications, such as the treatment of pain and headache.

3702 Botulinum toxin (Session B)

Location: Balcony I, Fourth Floor, Marriott

Francisco Cardoso
Belo Horizonte MG, Brazil
Charles H. Adler
Scottsdale, AZ, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the mechanism of action of botulinum toxin and differences between botulinum toxin type A and B; 2. Recognize the different disorders for which botulinum toxin is indicated; 3. Explain how to inject botulinum toxin for different disorders and the dosing needed to start treatment.

3703 Programming for DBS (Session A)

Location: Napoleon B1, Third Floor, Sheraton

Jens Volkmann
Kiel, Germany
Michele Tagliati
New York, NY, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Understand and apply the principals of deep brain stimulation programming for each basal ganglia target; 2. Perform a systematic initial programming session and manage patients at follow-up visits; 3. Evaluate common problems and trouble shooting strategies.

3704 Programming for DBS (Session B)

Location: Balcony L, Fourth Floor, Marriott

Jean-Michel Gracies
New York, NY, USA
Elena Moro
Toronto, Canada

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify the patients who are candidates for the DBS of the subthalmic nucleus, quorus pallidus and thalamus; 2. Discuss and apply the principles of DBS programming and of management of patients with DBS; 3. Identify problems and apply troubleshooting strategies associated with DBS treatment.

3705 Functional surgery targeting

Location: Napoleon C1, Third Floor, Sheraton

Maria Rodriguez-Oroz
Pamplona, Spain
Philip Starr
San Francisco, CA, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the major techniques used to localize the thalamus, global pallidus and subthalmic nucleus for placement of lesions or stimulators for the treatment of Movement Disorders; 2. Recognize patterns of single unit discharge encountered during microelectrode recording in the subthalmic nucleus and global pallidus; 3. Identify MRI based stereotactic targeting of basal ganglia neuclei.

3706 Clinical trial design

Location: Napoleon D, Third Floor, Sheraton

Karl D. Kieburtz
Rochester, NY, USA
Olivier Rascol
Toulouse, France

3707 Electrophysiological study of Movement Disorder patients

Location: La Galerie 6, Second Floor, Marriott

Robert Chen
Toronto, Canada
John C. Rothwell
London, United Kingdom

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify the type of patients in whom electrophysiological study of Movement Disorders patients may be helpful in establishing the diagnosis or to further understand the pathophysiology; 2. Describe the electrophysiological studies commonly used, the necessary equipment and the limitations of the tests; 3. Discuss the physiological findings in several Movement Disorders including tremor, psychogenic Movement Disorders and myoclonus.

3708 Writing and publishing manuscripts

Location: Napoleon C3, Third Floor, Sheraton

Günther Deuschl
Kiel, Germany
Christopher G. Goetz
Chicago, IL, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Explain the editorial process from manuscript submission, through review and eventual publishing; 2. Understand the criteria used by editors in evaluating manuscripts and selecting them for acceptance in scientific journals, using Movement Disorders as a prototype; 3. List advised steps for authors to follow in developing a manuscript from concept through full completion; 4. Discuss strategies to enhance manuscript quality and presentation.

Scientific Program ~ Monday/Tuesday

3709 How to get a grant
Location: Napolean B2, Third Floor, Sheraton
Katrina Gwinn-Hardy
Bethesda, MD, USA
Jeffrey H. Kordower
Chicago, IL, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Understand writing strategies for each section of the application; 2. Understand the infrastructure of NIH grant funding (e.g. composition of a study section, job of council, who are SRAs, program officers, etc.); 3. Gain an overview of NIH granting mechanisms; 4. Become acquainted with common pitfalls of NIH grant writing; 5. Be made aware of web based resources for grant applications; 6. Be made aware of strategies for funding at NIH.

3710 Animal models
Location: Napoleon C2, Third Floor, Sheraton
Kevin S. McNaught
New York, NY, USA
Serge Przedborski
New York, NY, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Be familiarized with experimental models of Parkinson's disease and to discuss their strengths and weaknesses; 2. Provide an overview of the current understanding of the mechanism of cell death in Parkinson's disease using these animal models; 3. Discuss how those molecular targets could be used to devise neuroprotective strategies for Parkinson's disease.

3711 Canceled

3712 Digitizing and editing your videotapes and creating a digital videotape library
Location: Balcony M, Fourth Floor, Marriott
Gregory F. Molnar
Toronto, Canada
Mandar Jog
London, Canada

Session Objectives: At the conclusion of this session, participants should be able to: 1. Identify the necessary computer hardware, software and connections to digitize videotapes; 2. Describe the basic editing steps for video files and how to create a digital video library; 3. Explain the most effective way to incorporate video clips into PowerPoint presentations and how to save them for transport to other computers.

3713 The role of the Movement Disorders nurse
Location: Balcony N, Fourth Floor, Marriott
Lisa A. Johnston
Toronto, Canada
Carol Brown Moskowitz
New York, NY, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the expanded scope of practice for nurses in Movement Disorders; 2. Discuss the evolving role of the Movement Disorders nurse with deep brain stimulation (DBS), recognize the spectrum of care for the patient undergoing DBS and their family to help achieve a realistic outcome for DBS; 3. Discuss the implications of genetic testing on the Movement Disorder patient and the role of the nurse in counseling, define useful explanatory models to use as families consider genetic testing.

Tuesday, March 8

Plenary Sessions

Admission to these sessions is by delegate name badge. No ticket is required for admission to Plenary Sessions.

8:30 a.m. to 9:30 a.m.
4101 Dystonia and apraxia
Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet Room, Third Floor, Marriott
Chair: Reiner Benecke
Rostock, Germany
8:30 a.m. **What's hot in dystonia**
Stanley Fahn
New York, NY, USA
9:00 a.m. **Update on apraxia**
Mark Hallett
Bethesda, MD, USA

Session Objectives: At the conclusion of this session, participants should be able to: 1. Explain the pathophysiology and neurobiology of the various manifestations of dystonia and the basics of genotype-phenotype interactions; 2. Explain the pathophysiology and clinical manifestations of apraxia in neurovascular and degenerative neurological diseases.

9:30 a.m. to 10:00 a.m.
4102 Stanley Fahn Lecture: Is the brain pathology in sporadic Parkinson's disease uniform?
Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet Room, Third Floor, Marriott
Heiko Braak
Frankfurt, Germany

Evaluations

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When completed, evaluations may be returned to your meeting room attendants, the evaluation drop boxes, the MDS Registration Desk or the CME Desk.

Scientific Program ~ Tuesday

Parallel Sessions

A ticket is required for admission to these smaller, interactive sessions. Attendance for Parallel Sessions is limited. There are no additional fees for tickets. Delegates that do not have tickets to these sessions, but would like to attend, are asked to check at the On-Site Registration Desk for ticket availability.

10:30 a.m. to 12:45 p.m.

4201 Psychiatric aspects of Parkinson's disease

Location: Carondelet Room, Third Floor, Marriott

Chair: Christopher G. Goetz
Chicago, IL, USA

10:30 a.m. **Psychosis**
Christopher G. Goetz
Chicago, IL, USA

11:05 a.m. **Discussion**

11:15 a.m. **Mood - depression**
Valerie Voon
Toronto, Canada

11:50 a.m. **Discussion**

12:00 p.m. **Other behavior - impulsivity, addiction (hedonistic homeostatic dysregulation)**
Andrew H. Evans
London, United Kingdom

12:35 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Explain the major clinical elements of psychosis, depression, obsessive and impulsive behaviors seen in Parkinson's disease; 2. Understand the anatomical and biochemical bases of these disorders in the context of Parkinson's disease; 3. List the features of each behavioral disorder that are distinctive in the context of Parkinson's disease compared to the same disorder occurring without Parkinson's disease; 4. Discuss current therapies for each behavioral disorder.

4202 Non-dopaminergic features of Parkinson's disease

Location: Napoleon A, Third Floor, Sheraton

Chair: Olivier Rascol
Toulouse, France

10:30 a.m. **Sleep disorders**
Bradley F. Boeve
Rochester, MN, USA

11:00 a.m. **Autonomic dysfunction**
Horacio Kaufmann
New York, NY, USA

11:30 a.m. **Pain**
Olivier Rascol
Toulouse, France

12:00 p.m. **Discussion**

4203 Multiple system atrophy

Location: Napoleon B, Third Floor, Sheraton

Chair: Gregor K. Wenning
Innsbruck, Austria

10:30 a.m. **Clinical overview - including epidemiology, phenomenology, diagnosis (including imaging)**
Carlo Colosimo
Rome, Italy

11:00 a.m. **Etiology/pathogenesis**
Gregor K. Wenning
Innsbruck, Austria

11:30 a.m. **Therapy - current and experimental**
Niall P. Quinn
London, United Kingdom

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the clinical presentation and useful diagnostic tests in patients with MSA; 2. Discuss the etiopathogenesis of MSA; 3. List practical and experimental management strategies in MSA.

4204 Primary dystonias

Location: Borgne Room, Third Floor, Sheraton

Chair: Xandra Breakefield
Charlestown, MA, USA

10:30 a.m. **Clinical overview - including epidemiology, phenomenology, diagnosis (including imaging)**
Cynthia L. Comella
Chicago, IL, USA

11:00 a.m. **Etiology/pathogenesis**
Xandra Breakefield
Charlestown, MA, USA

11:30 a.m. **Therapy - current and experimental**
Kailash P. Bhatia
London, United Kingdom

12:00 p.m. **Discussion**

Scientific Program ~ Tuesday

4205 Huntington's disease
Location: Balcony L, Fourth Floor, Marriott
Chair: Anne B. Young
Boston, MA, USA

10:30 a.m. **Clinical overview - including epidemiology, phenomenology, diagnosis (including imaging)**
Kathleen M. Shannon
Chicago, IL, USA

11:00 a.m. **Etiology/pathogenesis**
M. Flint Beal
New York, NY, USA

11:30 a.m. **Therapy - current and experimental**
Anne B. Young
Boston, MA, USA

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the pathophysiology and neurobiology of Huntington's disease; 2. Discuss the diagnostic approaches and tools available for Huntington's disease; 3. Discuss the pharmacological and non-pharmacological treatment options available for Huntington's disease.

4206 Physiology and the basal ganglia model
Location: Napoleon D, Third Floor, Sheraton
Chair: Alan Crossman
Manchester, United Kingdom

10:30 a.m. **Anatomy**
Yoland Smith
Atlanta, GA, USA

11:00 a.m. **Physiology**
Jerrold Lee Vitek
Cleveland, OH, USA

11:30 a.m. **Pharmacology**
Alan Crossman
Manchester, United Kingdom

12:00 p.m. **Discussion**

Evaluations

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4207 Update on alphasynuclein and the Lewy body
Location: Napoleon C, Third Floor, Sheraton
Chair: Katrina Gwinn-Hardy
Bethesda, MD, USA

10:30 a.m. **Clinical aspects of the familial synucleinopathies**
Katrina Gwinn-Hardy
Bethesda, MD, USA

11:00 a.m. **Update on alphasynuclein and its role in disease**
Andrew Singleton
Bethesda, MD, USA

11:30 a.m. **Lewy bodies**
Kevin S. McNaught
New York, NY, USA

12:00 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Recognize some common features of the synucleinopathies clinically, pathologically; 2. Discuss clinical features that are not distinctive for the synucleinopathies when compared to other genetic causes of parkinsonism; 3. Discuss synucleinopathies in the context of other neurodegenerative disorders, and consider the current understanding genetics has contributed to neurodegenerative diseases.

Abstract Sessions

Admission to these sessions is by delegate name badge. No ticket is required for admission to Poster Sessions or Platform Presentations.

12:45 p.m. to 2:30 p.m.
4501 Poster Session 3
Location: Mardi Gras Ballroom, Third Floor, Marriott
Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:45 p.m. to 2:30 p.m.
Abstracts: 384-592

2:30 p.m. to 3:30 p.m.
4502 Highlights of poster sessions
Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet, Third Floor, Marriott

Clinical Highlights
Chairs: Mark A. Stacy
Durham, NC, USA
Joseph Jankovic
Houston, TX, USA

Scientific Highlights
Chairs: Mark Cookson
Bethesda, MD, USA
Etienne C. Hirsch
Paris, France

Scientific Program ~ Tuesday

Parallel Sessions

A ticket is required for admission to these smaller, interactive sessions. Attendance for Parallel Sessions is limited. There are no additional fees for tickets. Delegates that do not have tickets to these sessions, but would like to attend, are asked to check at the On-Site Registration Desk for ticket availability.

4:00 p.m. to 6:15 p.m.

4601 Cognitive dysfunction and dementia in Parkinson's disease

Location: Carondelet Room, Third Floor, Marriott

Chair: Bruno Dubois
Paris, France

4:00 p.m. **Definitions and clinical features**
Bruno Dubois
Paris, France

4:30 p.m. **Pathogenesis/neuroanatomy/pathology**
Dennis Dickson
Jacksonville, FL, USA

5:00 p.m. **Management**
David John Burn
Newcastle Upon Tyne, United Kingdom

5:30 p.m. **Discussion**

4602 PSP/CBD

Location: Napoleon A, Third Floor, Sheraton

Chair: Irene Litvan
Louisville, KY, USA

4:00 p.m. **Clinical overview - including epidemiology, phenomenology, diagnosis (including imaging)**
David R. Williams
Haggerston, United Kingdom

4:30 p.m. **Etiology/pathogenesis**
Irene Litvan
Louisville, KY, USA

5:00 p.m. **Therapy - current and experimental**
Peter Paul Pramstaller
Bolzano, Italy

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe current epidemiologic and genetic findings; 2. Identify major risk factors; 3. Discuss currently hypothesized etiopathogenic mechanisms.

4603 Spinocerebellar ataxias and other ataxias

Location: Napoleon B, Third Floor, Sheraton

Chair: Thomas Klockgether
Bonn, Germany

4:00 p.m. **Clinical overview - including epidemiology, phenomenology, diagnosis (including imaging)**
Alessandro Filla
Napoli, Italy

4:30 p.m. **Etiology/pathogenesis**
Alexis Brice
Paris, France

5:00 p.m. **Therapy - current and experimental**
Thomas Klockgether
Bonn, Germany

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe the clinical spectrum and epidemiology of spinocerebellar ataxias; 2. Discuss the genetic and molecular basis of spinocerebellar ataxias; 3. Describe the current treatment approaches in spinocerebellar ataxias.

4604 Adult-onset focal dystonia

Location: Borgne Room, Third Floor, Sheraton

Chair: Alfredo Berardelli
Rome, Italy

4:00 p.m. **Clinical overview - including epidemiology, phenomenology, diagnosis (including imaging)**
Steven Frucht
New York, NY, USA

4:30 p.m. **Etiology/pathogenesis/pathophysiology**
Alfredo Berardelli
Rome, Italy

5:00 p.m. **Therapy - current and experimental**
Thomas T. Warner
London, United Kingdom

5:30 p.m. **Discussion**

Scientific Program ~ Tuesday

4605 Epidemiology

Location: Napoleon C, Third Floor, Sheraton

Chair: Caroline M. Tanner
Sunnyvale, CA, USA

4:00 p.m. **Epidemiological methods**
Caroline M. Tanner
Sunnyvale, CA, USA

4:30 p.m. **Epidemiologic studies in Parkinson's disease and parkinsonisms**
Sigurlaug Sveinbjornsdottir
Reykjavik, Iceland

5:00 p.m. **Epidemiologic studies in dystonia and other Movement Disorders**
Caroline M. Tanner
Sunnyvale, CA, USA
Giovanni Defazio
Bari, Italy

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Describe basic epidemiologic approaches to studying Movement Disorders; 2. Discuss the epidemiology of Parkinson's disease and parkinsonism; 3. Discuss the epidemiology of dystonia.

4606 Hereditary and non-hereditary choreas

Location: Balcony L, Fourth Floor, Marriott

Chair: Ruth Walker
Bronx, NY, USA

4:00 p.m. **Pathophysiology of choreas**
Jonathan M. Brotchie
Toronto, Canada

4:30 p.m. **Neurocanthocytosis and McLeod's syndrome**
Adrian Danek
Munich, Germany

5:00 p.m. **Huntington's disease-like syndromes, benign hereditary chorea and other choreas**
Ruth Walker
Bronx, NY, USA

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Discuss the differential diagnosis of inherited and non-hereditary chorea; 2. Discuss the use of diagnostic laboratory and genetic tests; 3. Discuss the pathophysiology of chorea at the level of the basal ganglia.

4607 Motor stereotypies: bridging clinical practice and basic science

Location: Napoleon D, Third Floor, Sheraton

Chair: Harvey S. Singer
Baltimore, MD, USA

4:00 p.m. **Stereotypies in childhood**
Harvey S. Singer
Baltimore, MD, USA

4:30 p.m. **Stereotypies in adulthood**
Joseph Jankovic
Houston, TX, USA

5:00 p.m. **Neural circuits involved in stereotypies**
Ann M. Graybiel
Cambridge, MA, USA

5:30 p.m. **Discussion**

Session Objectives: At the conclusion of this session, participants should be able to: 1. Define, identify and discuss physiological and pathological types of motor stereotypies in children and adults; 2. Discuss pharmacological and non-pharmacological treatment options for motor stereotypies; 3. Discuss the pathophysiology and neurobiology of motor stereotypy disorders.

Plenary Session

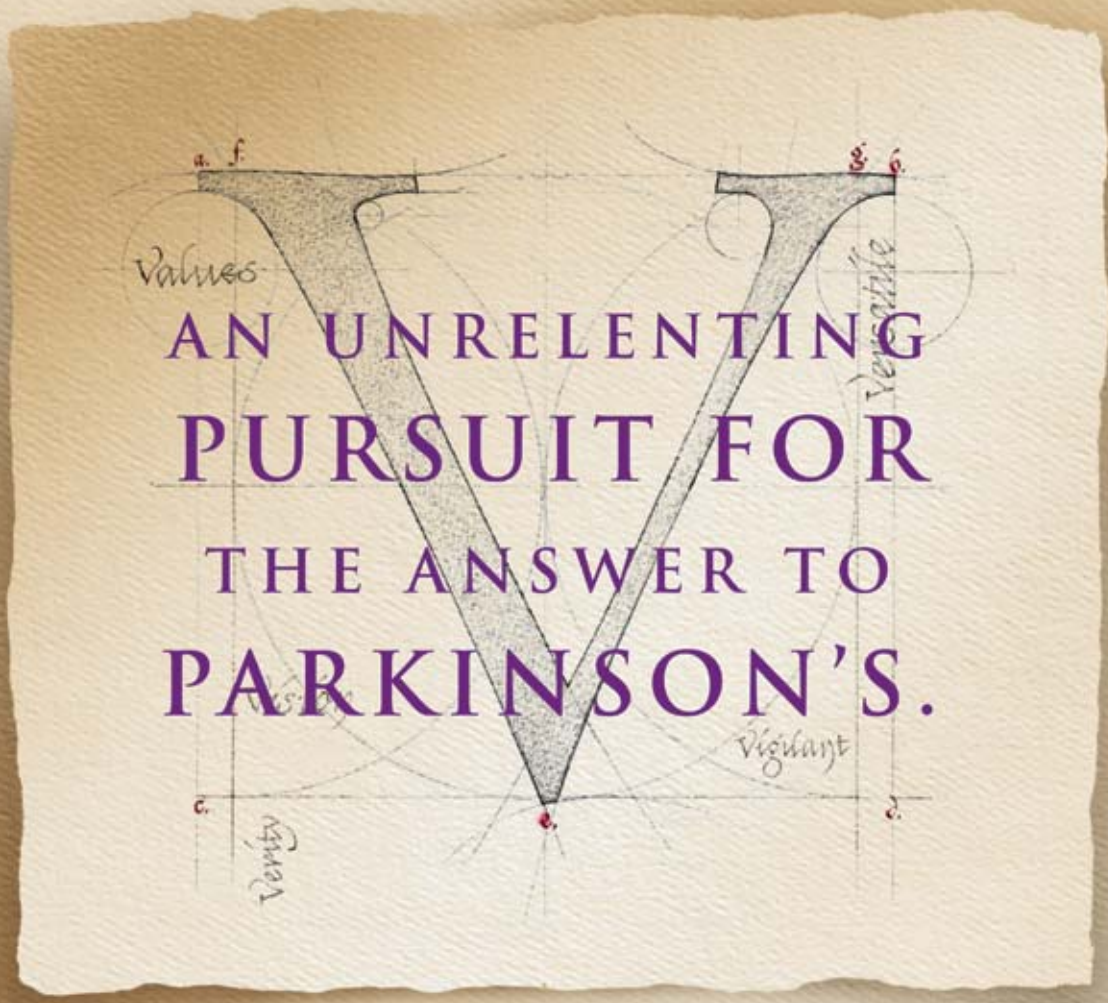
Admission to this session is by delegate name badge. No ticket is required for admission to Plenary Sessions.

6:30 p.m. to 8:30 p.m.

4901 Lessons my patients taught me

Location: Acadia Room, Third Floor, Marriott
Overflow: Carondelet Room, Third Floor, Marriott

Chair: Werner Poewe
Innsbruck, Austria
Anthony E. Lang
Toronto, Canada
Christopher G. Goetz
Chicago, IL, USA
Eduardo Tolosa
Barcelona, Spain
Andrew J. Lees
London, United Kingdom
John G.L. Morris
Sydney, Australia



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- Provide increased "on" time and decreased "off" time[1]
- Demonstrate rapid and significant improvement in activities of daily living and motor function[1]
- Sustain benefits over the long term[2]
- Provide more consistent and reliable delivery of levodopa to the brain[1]

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

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, Worm-Petersen J, Sidén Å, et al. The tolerability and efficacy of entacapone over 3 years in patients with Parkinson's disease. *Eur J Neurol.* 2003;10:137-146.

5/04

C-STA-1018

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, hyper- or hypotension; 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 isoprenaline (isoproterenol) 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 isoprenaline and epinephrine, respectively. Therefore, drugs known to be metabolized by COMT, such as isoproterenol, epinephrine, norepinephrine, dopamine, dobutamine, alpha-methyldopa, apomorphine, isoetherine, and bitolterol 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 60 of 603 (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 603 (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 antiparkinsonian 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 everyday 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 PRECAUTIONS, 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 patients receiving antihypertensive 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, CYP2C9, CYP2C19, CYP2D6, CYP2E1 and CYP3A. *In vitro* studies of human CYP enzymes showed that entacapone inhibited the CYP enzymes 1A2, 2A6, 2C9, 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, levodopa 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 two 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 embryo-fetal development studies, entacapone was administered to pregnant animals throughout organogenesis at doses of up to 1000 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 160 mg/kg/day (plasma AUCs seven 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, dyspepsia, 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, falling, 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)*, flushing. ***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/hyperkinesia (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, 2, 1; **CENTRAL & PERIPHERAL NERVOUS SYSTEM DISORDERS,** Dyskinesia, 25, 15; 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, 8; Diarrhea, 10, 4; Abdominal Pain, 8, 4; Constipation, 6, 4; 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 postmarketing 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 15-30°C (59-86°F). [see USP Controlled Room Temperature.] Dispense in tight container (USP).

Stalevo
(carbidopa, levodopa and entacapone) tablets

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Manufactured by:
Orion Corporation
ORION PHARMA
Orionintie 1, FIN-02200 Espoo, Finland

Marketed by:
Novartis Pharmaceuticals Corporation
East Hanover, New Jersey 07936

Committee, Task Force and Regional Section Meetings

Saturday, March 5, 2005

7:00 a.m. to 8:00 a.m.

Financial Affairs Committee
Location: Iberville Room, Fourth Floor, Marriott

Sunday, March 6, 2005

7:00 a.m. to 8:30 a.m.

Archives Committee
Location: Iberville Room, Fourth Floor, Marriott

Bylaws Committee
Location: Beauregard Room, Fifth Floor, Marriott

Industrial Relations Committee
Location: Bacchus Room, Fourth Floor, Marriott

Young Members
Location: La Galerie 6, Second Floor, Marriott

12:45 p.m. to 2:30 p.m.

***Movement* Disorder Society – European Section**
Location: St. Charles Suite, 41st Floor, Marriott

Education Committee
Location: Iberville Room, Fourth Floor, Marriott

Journal Oversight Committee
Location: Beauregard Room, Fifth Floor, Marriott

Task Force for the Development of Rating Scales for Parkinson's Disease
Location: Bacchus Room, Fourth Floor, Marriott

Delegates Meeting
Location: La Galerie 6, Second Floor, Marriott

Monday, March 7, 2005

6:30 a.m. to 7:30 a.m.

UPDRS Revision Task Force
Location: Beauregard Room, Fifth Floor, Marriott

7:30 a.m. to 8:30 a.m.

MDS Annual Business Meeting
Location: Acadia Room, Third Floor Marriott

12:45 p.m. to 2:15 p.m.

Membership Committee
Location: Beauregard Room, Fifth Floor, Marriott

Neurosurgery Section Task Force
Location: Napoleon Suite, 41st Floor, Marriott

6:00 p.m. to 7:30 p.m.

Advocacy Groups Reception
Location: St. Charles Suite, 41st Floor, Marriott

Tuesday, March 8, 2005

7:00 a.m. to 8:30 a.m.

Scientific Issues Committee
Location: Beauregard Room, Fifth Floor, Marriott

Liaison/Public Relations Committee
Location: Lafayette Suite, 41st Floor, Marriott

Continuing Medical Education (CME) Committee
Location: Napoleon Suite, 41st Floor, Marriott

12:00 p.m. to 1:00 p.m.

International Congress Oversight Committee
Location: Napoleon Suite, 41st Floor, Marriott

1:00 p.m. to 2:00 p.m.

Awards Committee
Location: Beauregard Room, Fifth Floor, Marriott

Task Force on Epidemiology
Location: La Galerie 6, Second Floor, Marriott

1:00 p.m. to 2:30 p.m.

Congress Scientific Program Committee
Location: St. Charles Suite, 41st Floor, Marriott

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A Proven Drug in a New Delivery System

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(carbidopa-levodopa
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The most common adverse reactions reported with carbidopa-levodopa therapy have included dyskinesias, such as choreiform, dystonic, and other involuntary movements and nausea. Other side effects may include mental disturbances and symptoms resembling neuroleptic malignant syndrome. Individualize therapy to reduce adverse reactions.

PARCOPA™ 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.

When patients are receiving levodopa without a decarboxylase inhibitor, levodopa must be discontinued at least 12 hours before PARCOPA™ is started.

Each 10/100 mg and each 25/100 mg orally disintegrating tablet contains phenylalanine 3.4 mg; each 25/250 mg orally disintegrating tablet contains phenylalanine 8.4 mg.

Please see Brief Summary of Prescribing Information on adjacent page.

For more information visit www.PARCOPA.com.

PARCOPA™

(carbidopa-levodopa orally disintegrating tablets)

10 mg/100 mg • 25 mg/100 mg • 25 mg/250 mg

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BRIEF SUMMARY: Before prescribing PARCOPA™, please see package insert for full prescribing information.

INDICATIONS AND USAGE: PARCOPA™ is indicated in the treatment of the symptoms of idiopathic Parkinson's disease (paralysis agitans), postencephalitic parkinsonism, and symptomatic parkinsonism which may follow injury to the nervous system by carbon monoxide intoxication and/or manganese intoxication. PARCOPA™ is indicated in these conditions to permit the administration of lower doses of levodopa with reduced nausea and vomiting, with more rapid dosage titration, with a somewhat smoother response, and with supplemental pyridoxine (vitamin B_6). In some patients, a somewhat smoother antiparkinsonian effect results from therapy with carbidopa-levodopa than with levodopa. However, patients with markedly irregular ("on-off") responses to levodopa have not been shown to benefit from carbidopa-levodopa therapy. Although the administration of carbidopa permits control of parkinsonism and Parkinson's disease with much lower doses of levodopa, there is no conclusive evidence at present that this is beneficial other than in reducing nausea and vomiting, permitting more rapid titration, and providing a somewhat smoother response to levodopa. Certain patients who responded poorly to levodopa have improved when carbidopa-levodopa was substituted. This is most likely due to decreased peripheral decarboxylation of levodopa which results from administration of carbidopa rather than to a primary effect of carbidopa on the nervous system. Carbidopa has not been shown to enhance the intrinsic efficacy of levodopa in parkinsonian syndromes. In considering whether to give PARCOPA™ to patients already on levodopa who have nausea and/or vomiting, the practitioner should be aware that, while many patients may be expected to improve, some do not. Since one cannot predict which patients are likely to improve, this can only be determined by a trial of therapy. It should be further noted that in controlled trials comparing carbidopa-levodopa with levodopa, about half of the patients with nausea and/or vomiting on levodopa improved spontaneously despite being retained on the same dose of levodopa during the controlled portion of the trial.

CONTRAINDICATIONS: Nonselective monoamine oxidase (MAO) inhibitors are contraindicated for use with PARCOPA™. These inhibitors must be discontinued at least two weeks prior to initiating therapy with PARCOPA™. PARCOPA™ may be administered concomitantly with the manufacturer's recommended dose of an MAO inhibitor with selectivity for MAO type B (e.g., selegiline HCl) (See PRECAUTIONS, *Drug Interactions*). PARCOPA™ is contraindicated in patients with known hypersensitivity to any component of this drug, and in patients with narrow-angle glaucoma. Because levodopa may activate a malignant melanoma, PARCOPA™ should not be used in patients with suspicious, undiagnosed skin lesions or a history of melanoma. **WARNINGS: When PARCOPA™ (carbidopa-levodopa orally disintegrating tablets) is to be given to patients who are being treated with levodopa, levodopa must be discontinued at least twelve hours before therapy with PARCOPA™ (carbidopa-levodopa orally disintegrating tablets) is started. In order to reduce adverse reactions, it is necessary to individualize therapy. See DOSAGE AND ADMINISTRATION section before initiating therapy.** The addition of carbidopa with levodopa in the form of PARCOPA™ 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 permits more levodopa to reach the brain and more dopamine to be formed, certain adverse CNS effects, e.g., dyskinesias (involuntary movements), may occur at lower dosages and sooner with PARCOPA™ than with levodopa alone. Levodopa alone, as well as PARCOPA™, is associated with dyskinesias. The occurrence of dyskinesias may require dosage reduction. As with levodopa, PARCOPA™ 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. PARCOPA™ 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 PARCOPA™ to patients with a history of myocardial infarction who have residual atrial, nodal, or ventricular arrhythmias. In such patients, cardiac function should be monitored with particular care during the period of initial dosage adjustment, in a facility with provisions for intensive cardiac care. As with levodopa, treatment with PARCOPA™ 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 PARCOPA™ 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, hyper- or hypotension; 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. **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 PARCOPA™ provided the intraocular pressure is well controlled and the patient is monitored carefully for changes in intraocular pressure during therapy. ***Information for Patients: Phenylketonurics:*** Phenylketonuric patients should be informed that PARCOPA™ contains phenylalanine 3.4 mg per 25/100 orally disintegrating tablet, 3.4 mg per 10/100 orally disintegrating tablet, and 8.4 mg per 25/250 orally disintegrating tablet. Patients should be instructed not to remove PARCOPA™ Tablets from the bottle until just prior to dosing. With dry hands, the tablet should be gently removed and immediately placed on the tongue to dissolve and be swallowed with the saliva. The patient should be informed that PARCOPA™ is an immediate-release formulation of carbidopa-levodopa that is designed to begin release of ingredients within 30 minutes. It is important that PARCOPA™ 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 antiparkinson 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 if such response poses a problem to lifestyle. Patients should be advised that occasionally, dark color (red, brown, or black) may appear in saliva, urine, or sweat after ingestion of PARCOPA™. 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 or carbidopa-levodopa therapy. NOTE: The suggested advice to patients being treated with PARCOPA™ 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. ***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 carbidopa-levodopa than with levodopa. Carbidopa-levodopa 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 levodopa or carbidopa-levodopa therapy. ***Drug Interactions:*** *Caution should be exercised when the following drugs are administered concomitantly with* PARCOPA™ (carbidopa-levodopa orally disintegrating tablets). Symptomatic postural hypotension has occurred when carbidopa-levodopa was added to the treatment of a patient receiving antihypertensive drugs. Therefore, when therapy with PARCOPA™ is started, dosage adjustment of the antihypertensive drug may be required. For patients receiving MAO inhibitors (Type A or B), see CONTRAINDICATIONS. Concomitant therapy with selegiline and carbidopa-levodopa may be associated with severe orthostatic hypotension not attributable to carbidopa-levodopa alone (see CONTRAINDICATIONS). There have been rare reports of adverse reactions, including hypertension and dyskinesia, resulting from the concomitant use of tricyclic antidepressants and carbidopa-levodopa. Dopamine D_2 receptor antagonists (e.g., phenothiazines, butyrophenones, risperidone) and isoniazid may reduce the therapeutic effects of levodopa. In addition, the beneficial effects of levodopa in Parkinson's disease have been reported to be reversed by phenytoin and papaverine. Patients taking these drugs with PARCOPA™ should be carefully observed for loss of therapeutic response. Iron salts may reduce the bioavailability of levodopa and carbidopa. The clinical relevance is unclear. 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. ***Carcinogenesis, Mutagenesis, Impairment of Fertility:*** In a two-year bioassay of carbidopa and 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. In reproduction studies with carbidopa and 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. ***Pregnancy: Pregnancy Category C:*** No teratogenic effects were observed in a study in mice receiving up to 20 times the maximum recommended human dose of carbidopa and 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. Carbidopa and 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 are no adequate or well-controlled studies in pregnant women. 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. Use of PARCOPA™ in women of child-bearing potential requires that the anticipated benefits of the drug be weighed against possible hazards to mother and child. ***Nursing Mothers:*** It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when PARCOPA™ is administered to a nursing woman. ***Pediatric Use:*** Safety and effectiveness in pediatric patients have not been established. Use of the drug in patients below the age of 18 is not recommended. **ADVERSE REACTIONS:** The most common adverse reactions reported with carbidopa-levodopa therapy 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, dyspepsia, dry mouth, taste alterations. *Hematologic:* agranulocytosis, hemolytic and non-hemolytic anemia, thrombocytopenia, leukopenia. *Hypersensitivity:* angioedema, urticaria, pruritus, Henoch-Schonlein 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 PARCOPA™ 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, falling, 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), flushing. *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. **OVERDOSAGE:** Management of acute overdosage with PARCOPA™ is the same as management of acute overdosage with levodopa. Pyridoxine is not effective in reversing the actions of PARCOPA™. General supportive measures should be employed, along with immediate gastric lavage. Intravenous fluids should be administered judiciously and an adequate airway maintained. Electrocardiographic monitoring should be instituted and the patient carefully observed for the development of arrhythmias; if required, appropriate anti-arrhythmic therapy should be given. The possibility that the patient may have taken other drugs as well as PARCOPA™ should be taken into consideration. To date, no experience has been reported with dialysis; hence, its value in overdosage is not known. Based on studies in which high doses of levodopa and/or carbidopa were administered, a significant proportion of rats and mice given single oral doses of levodopa of approximately 1500-2000 mg/kg are expected to die. A significant proportion of infant rats of both sexes are expected to die at a dose of 800 mg/kg. A significant proportion of rats are expected to die after treatment with similar doses of carbidopa. The addition of carbidopa in a 1:10 ratio with levodopa increases the dose at which a significant proportion of mice are expected to die to 3360 mg/kg. **DOSAGE AND ADMINISTRATION:** ***Instructions for Use/Handling PARCOPA™ Tablets:*** Just prior to administration, GENTLY remove the tablet from the bottle with dry hands. IMMEDIATELY place the PARCOPA™ Tablet on top of the tongue where it will dissolve in seconds, then swallow with saliva. Administration with liquid is not necessary. The optimum daily dosage of PARCOPA™ must be determined by careful titration in each patient. PARCOPA™ is available in a 1:4 ratio of carbidopa to levodopa (PARCOPA™ 25/100) as well as 1:10 ratio (PARCOPA™ 25/250 and PARCOPA™ 10/100). Tablets of the two ratios may be given separately or combined as needed to provide the optimum dosage. Studies show that peripheral dopa decarboxylase is saturated by carbidopa at approximately 70 to 100 mg a day. Patients receiving less than this amount of carbidopa are more likely to experience nausea and vomiting. ***Usual Initial Dosage:*** Dosage is best initiated with one tablet of PARCOPA™ 25/100 three times a day. This dosage schedule provides 75 mg of carbidopa per day. Dosage may be increased by one tablet every day or every other day, as necessary, until a dosage of eight tablets of PARCOPA™ 25/100 a day is reached. If PARCOPA™ 10/100 is used, dosage may be initiated with one tablet three or four times a day. However, this will not provide an adequate amount of carbidopa for many patients. Dosage may be increased by one tablet every day or every other day until a total of eight tablets (2 tablets q.i.d.) is reached. ***How to Transfer Patients from Levodopa:*** **Levodopa must be discontinued at least twelve hours before starting PARCOPA™ (carbidopa-levodopa orally disintegrating tablets).** A daily dosage of PARCOPA™ should be chosen that will provide approximately 25 percent of the previous levodopa dosage. Patients who are taking less than 1500 mg of levodopa a day should be started on one tablet of PARCOPA™ 25/100 three or four times a day. The suggested starting dosage for most patients taking more than 1500 mg of levodopa is one tablet of PARCOPA™ 25/250 three or four times a day. ***Maintenance:*** Therapy should be individualized and adjusted according to the desired therapeutic response. At least 70 to 100 mg of carbidopa per day should be provided. When a greater proportion of carbidopa is required, one tablet of PARCOPA™ 25/100 may be substituted for each tablet of PARCOPA™ 10/100. When more levodopa is required, PARCOPA™ 25/250 should be substituted for PARCOPA™ 25/100 or PARCOPA™ 10/100. If necessary, the dosage of PARCOPA™ 25/250 may be increased by one-half or one tablet every day or every other day to a maximum of eight tablets a day. Experience with total daily dosages of carbidopa greater than 200 mg is limited. Because both therapeutic and adverse responses occur more rapidly with PARCOPA™ than with levodopa alone, patients should be monitored closely during the dose adjustment period. Specifically, involuntary movements will occur more rapidly with PARCOPA™ than with levodopa. The occurrence of involuntary movements may require dosage reduction. Blepharospasm may be a useful early sign of excess dosage in some patients. ***Addition of Other Antiparkinsonian Medications:*** Standard drugs for Parkinson's disease, other than levodopa without a decarboxylase inhibitor, may be used concomitantly while PARCOPA™ is being administered, although dosage adjustments may be required. ***Interruption of Therapy:*** Sporadic cases of a symptom complex resembling Neuroleptic Malignant Syndrome (NMS) have been associated with dose reductions and withdrawal of carbidopa-levodopa. Patients should be observed carefully if abrupt reduction or discontinuation of PARCOPA™ is required, especially if the patient is receiving neuroleptics. (See WARNINGS.) If general anesthesia is required, PARCOPA™ may be continued as long as the patient is permitted to take fluids and medication by mouth. If therapy is interrupted temporarily, the patient should be observed for symptoms resembling NMS, and the usual daily dosage may be administered as soon as the patient is able to take oral medication.

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Mylan Bertek Pharmaceuticals Inc., based in Research Triangle Park, NC, was founded in 1996 as the proprietary products division of Mylan Laboratories Inc. Mylan Bertek Pharmaceuticals has medical and clinical expertise in neurology, as well as an experienced marketing and sales staff in the field of neurology products. The company has actively pursued new products for the treatment of neurological diseases, including epilepsy and Parkinson's disease. Stop by our booth to see how we are making a difference in the lives of patients with Parkinson's disease.

Exhibitor Information and Directory

National Parkinson Foundation
1501 NW 9th Avenue
Miami, FL 33136
USA
Telephone: 1-800-327-4545
Fax: +1 305-243-7851
Booth #: 410

The National Parkinson Foundation (NPF) is the largest worldwide organization serving persons affected by Parkinson's disease. The Foundation supports scientific research into the cause and cure for Parkinson's disease, as well as programs to improve the delivery of care and the quality of the lives of those who must deal with the disease every day. In addition to research, NPF funds education, specialized training, outreach services, and advocacy for persons with Parkinson's disease, their caregivers and families, and health-care professionals.

National Spasmodic Dysphonia Association
230 Park Boulevard
Suite 203
Itasca, IL 60143
USA
Telephone: 1-800-795-NSDA
Booth #: 417

Spasmodic Dysphonia is a neurological voice disorder that involves involuntary "spasms" of the vocal cords causing interruptions of speech and affecting the voice quality. The National Spasmodic Dysphonia Association strives to advance medical research, promote physician and public awareness, and sponsor support groups for patients and their families.

The National Spasmodic Torticollis Association
9920 Talbert Ave. #233
Fountain Valley, CA 92708
USA
Telephone: +1 714-378-7837
Fax: +1 714-378-7830
Web site: www.torticollis.org
Booth #: 118

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.

Neurotoxin Institute
100 Avenue of the Americas
7th Floor
New York, NY 10013
USA
Telephone: 1-866-682-6368
Fax: +1 212-925-1026
Web site: www.neurotoxininstitute.com
Booth #: 403

The Neurotoxin Institute (NTI) is a multi-disciplinary organization created to serve as a comprehensive independent source of information related to the basic science and the clinical applications of neurotoxin therapies. The Institute fosters the learning and teaching of both theory and practical techniques, and encourages further research in support of these goals.

Novartis International AG
Lichstr. 35
Basel CH-4002
Switzerland
Telephone: + 41 61 324 1111
Fax: + 41 61 324 6652
Web site: www.novartis.com
Booth #: 214

Novartis AG is a world leader in pharmaceuticals and consumer health, headquartered in Basel, Switzerland. Novartis researches, develops, manufacturers and markets leading innovative prescription drugs used to treat a number of diseases and conditions and has been a leader in the Neuroscience area for more than 50 years.

Orion Corporation Orion Pharma
Orionintie 1
FIN-02200 Espoo
Finland
Tel: + 358 10 429 4701
Global Brand Manager, Susanna Laajava
Fax: + 358 10 429 3815
Web site: www.orionpharma.com
Booth #: 214

Orion Pharma, the pharmaceutical division of the Orion Group, is a North European R&D-based, business-driven pharmaceuticals company with special emphasis on developing innovative treatments for global markets. Operationally its businesses comprise Core Therapy Areas, Speciality Products, Animal Health and Fermion. The R&D and product strategies are focused on central nervous system disorders, cardiovascular diseases and intensive care, and hormone therapies. Partnerships and networking are increasingly important throughout the value chain, both in research and development and in reaching the global markets.

Please feel invited to visit the combined exhibition of Novartis and Orion Pharma.

For further information please visit the companies' websites.
www.novartis.com
www.orion.fi/english

Exhibitor Information and Directory

The Parkinson's Association of Louisiana
2030 Dickory Avenue
#202
Harahan, LA 70123
USA
Telephone: +1 504-733-7203
Fax: +1 504-733-7203
Web site: www.parkinsonsla.org
Booth #: 402

The Parkinson's Association of Louisiana (PAL), is a non-profit, 501 (c) 3 corporation, made up of dedicated volunteers sharing a common vision. The mission of the PAL is to provide support, education and funding for the Parkinson's community of Louisiana with the ultimate goal of finding a cure.

Prestwick Pharmaceuticals, Inc.
1825 K Street NW
Suite 1475
Washington, DC 20006
USA
Telephone: +1 202-296-1400
Fax: +1 202-296-7450
Web site: www.prestwickpharma.com
Booth #: 394

Prestwick Pharmaceuticals, Inc. is an emerging Specialty Pharmaceutical company that focuses on treatments for Central Nervous System (CNS) disorders. The company's lead product, Tetrabenazine, has been approved in Europe, Canada and Australia for a series of disabling hyperkinetic movement disorders. Prestwick has completed two pivotal phase III clinical trials with Tetrabenazine in the U.S. and plans to file an NDA in 2005.

Priority Healthcare
250 Technology Park
Lake Mary, FL
USA
Telephone: 1-800-892-9622
Fax: +1 407-804-3921
Web site: www.priorityhealthcare.com
Booth #: 405

Priority Healthcare Corporation is a national specialty pharmacy and distributor that provides biopharmaceuticals, complex therapies, and related disease treatment services. Priority Healthcare provides comprehensive programs for patients, payors, physicians, and pharmaceutical manufacturers for a growing number of disease states including cancer, hepatitis C, respiratory and pulmonary conditions, infertility, rheumatoid arthritis, hemophilia, multiple sclerosis, and macular degeneration.

Restless Legs Syndrome Foundation
819 Second Street SW
Rochester, MN 55902
USA
Telephone: +1 507-287-6465
Fax: +1 507-287-6312
Web site: www.rls.org
Booth #: 401

The Restless Legs Syndrome Foundation (RLS) is a non-profit organization dedicated to improving the lives of the millions of men, women and children who live with this often devastating disease, through increased awareness, improved treatments and research to find a cure.

Schwarz Pharma AG
Alfred-Nobel-Strasse 10
Monheim 40789
Germany
Telephone: +49 2173 48 0
Fax: +49 2173 48 1108
Web site: www.schwarzpharma.com
Booth #: 119

SCHWARZ PHARMA, a multi-national pharmaceutical company, develops and markets innovative drugs for unmet medical needs. With an established reputation for excellence in cardiology, the company has extended its focus to include urological and neurological diseases. Within neurology, ongoing projects include treatments for Parkinson's disease, restless legs syndrome, epilepsy and neuropathic pain. SCHWARZ PHARMA is based in Monheim, Germany and employs over 3,800 professionals worldwide.

Schwarz Pharma, Inc. (USA)
6140 West Executive Drive
Mequon, WI 53092
USA
Telephone: +1 262-238-5441
Fax: +1 262-238-0961
Booth #: 314

PARCOPA™ (carbidopa-levodopa orally disintegrating tablets) is a therapeutic alternative to Sinemet® Tablets that provides all the benefits of carbidopa-levodopa in a unique orally dissolving formulation. Featuring RapiTab™ orally dissolving technology, PARCOPA offers patients suffering from Parkinson's disease an easy, accessible way to take levodopa replacement therapy.

Exhibitor Information and Directory

The Society for Progressive Supranuclear Palsy
11350 McCormick Road
Executive Plaza III, Suite 906
Hunt Valley, MD 21031
USA
Telephone: +1 410-486-3330 or 1-800-457-4777
Fax: +1 410-486-4283
Web site: www.psp.org
Booth #: 324

Progressive supranuclear palsy (PSP) is an under-recognized terminal brain disease. The Society for Progressive Supranuclear Palsy is dedicated to increasing awareness of PSP, advancing research toward a cure and providing support and education for persons with PSP, their families and healthcare professionals.

Valeant Pharmaceuticals International
3300 Hyland Ave.
Costa Mesa, CA 92626
USA
Telephone: +1 714-545-0100
Fax: +1 714-668-3139
Web site: www.valeant.com
Booth #: 114

Valeant Pharmaceuticals International is a global, publicly traded specialty pharmaceutical company that discovers, develops, manufactures and markets a broad range of pharmaceutical products in three therapeutic areas, neurology, infectious disease and dermatology.

Vitaline Formulas
825 Challenger Drive
Green Bay, WI 54211
USA
Telephone: +1 920-406-3614
Fax: +1 920-469-4410
Web site: www.vitalinecoq10.com
Booth #: 408

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WE MOVE
204 West 84th Street
New York, NY 10024
USA
Telephone: +1 212-875-8312
Fax: +1 212-875-8389
Web site: www.wemove.org
Booth #: 322

WE MOVE is a not-for-profit organization providing Movement Disorder information and education to health professionals and patients. At www.mdvu.org, health care professionals will find research news, diagnostic and treatment information, online CME, practice tools and more. Physicians can refer patients and families to www.wemove.org for information and support.

World Parkinson Congress, Inc.
710 West 168th Street
Room 336
New York, NY 10032
USA
Telephone: +1 212-923-4700
Fax: +1 212-923-4778
Web site: www.worldPDcongress.org
Booth #: 393

The World Parkinson Congress is dedicated to providing an international forum for the best scientific discoveries, medical practices and caregiver initiatives related to Parkinson's disease. By uniting the global Parkinson's community, the Congress will highlight best international treatment practices and hopefully expedite the discovery of a cure for this devastating disease.

For more information regarding patient demonstration videos, webcast training sessions and patient information kits, please call 1-877-7APOKYN (727-6596).

Visit www.apokyn.com.

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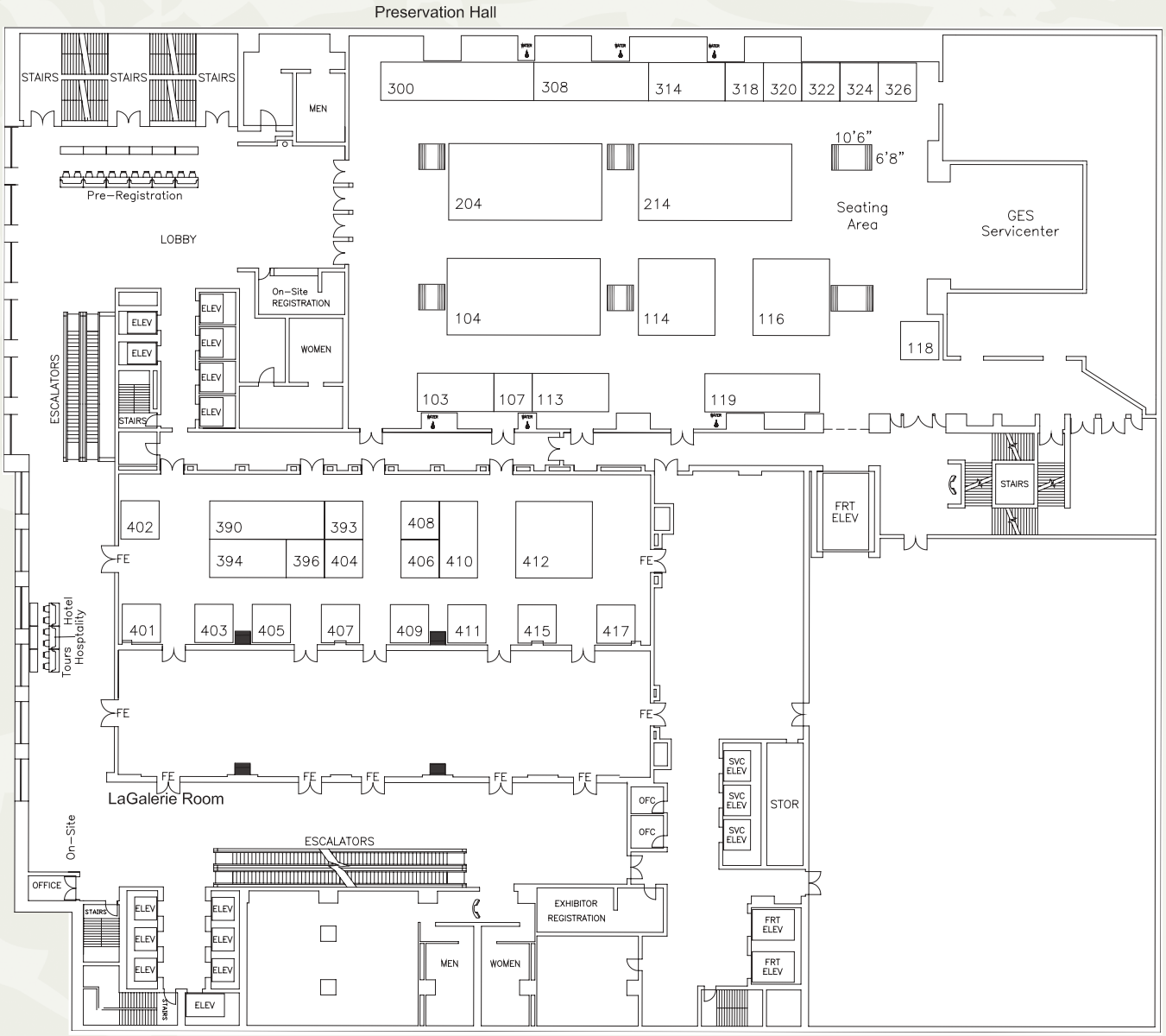
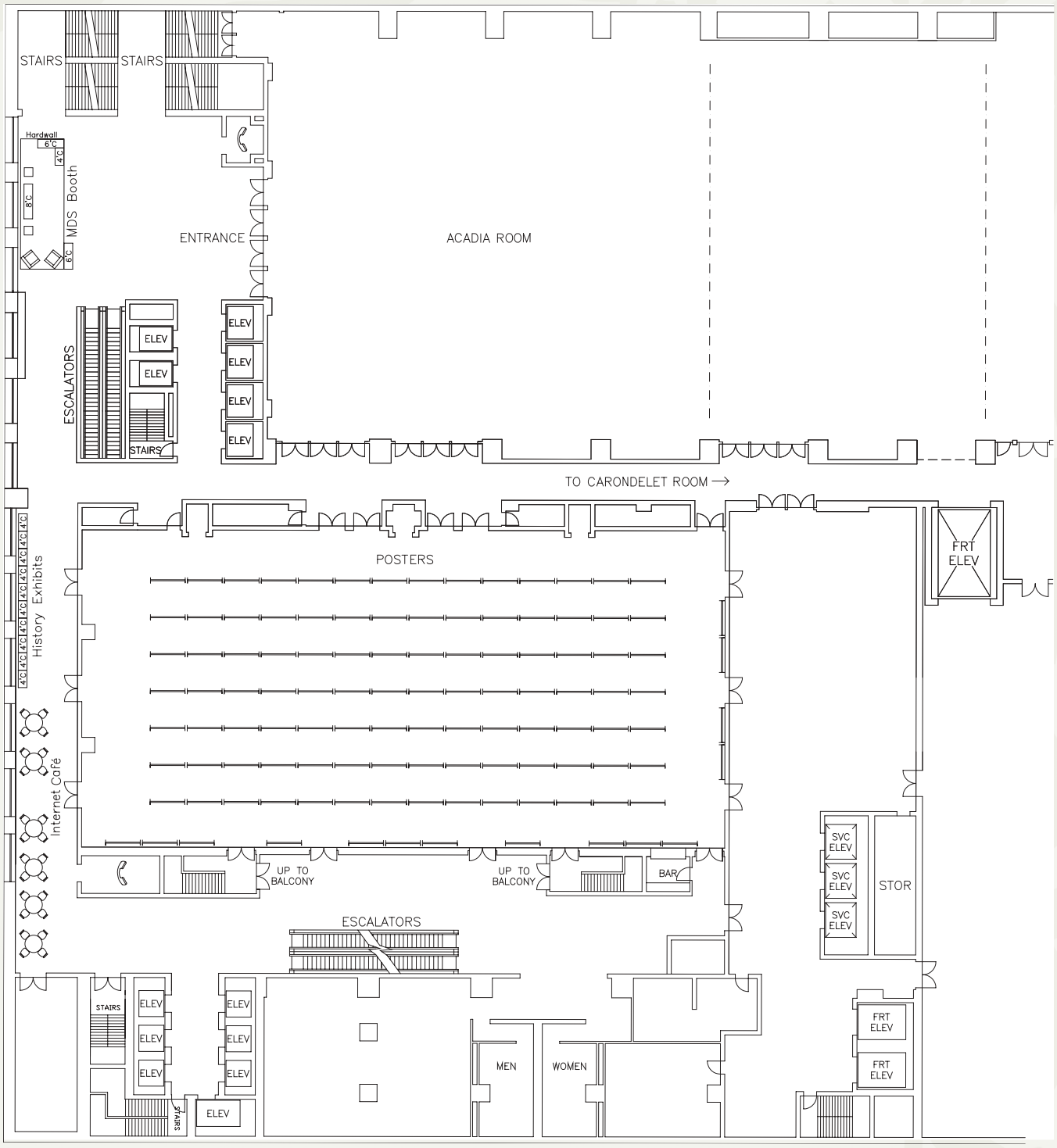
MYLAN BERTEK PHARMACEUTICALS INC.

APO-ADV-004

Map of Second Floor

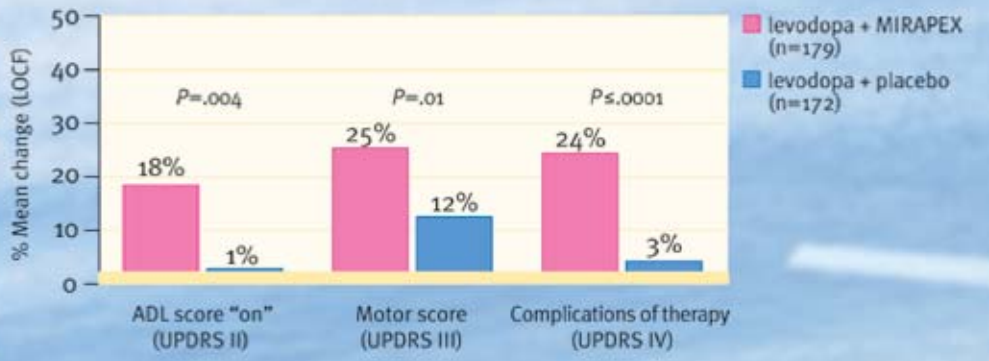


Map of Third Floor



Adjunctive MIRAPEX improves functioning vs placebo[1,2*]

UPDRS ADL "ON," MOTOR, AND THERAPY-RELATED COMPLICATION SCORES[1*]

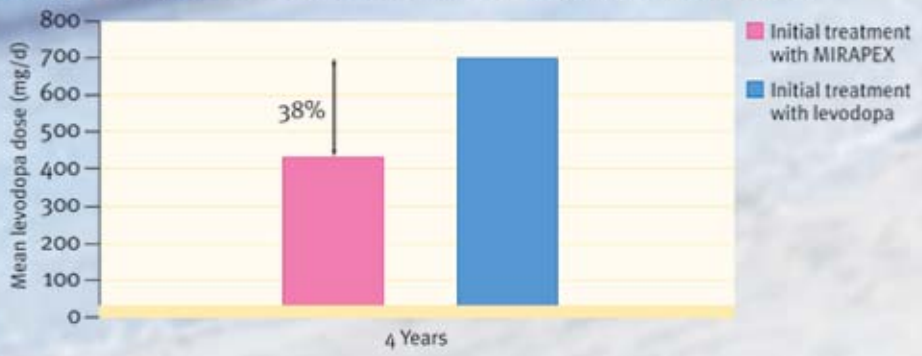


And significantly decreased the levodopa dose[1*]

- In this same trial, adjunctive MIRAPEX significantly decreased the levodopa dose vs placebo (27% vs 5%, *P*≤.0001)[1*]

Initial MIRAPEX maintained control while sparing the dose of levodopa[3†]

AT 4 YEARS, PATIENTS TREATED WITH MIRAPEX NEEDED 38% LESS LEVODOPA TO MAINTAIN EFFECTIVE CONTROL (mean total daily dose)[3†]



*Multicenter, double-blind, placebo-controlled, randomized, 32-week trial of 360 patients (ITT cohort=351) with advanced idiopathic PD (Hoehn and Yahr stages II-IV) on stable doses of levodopa experiencing motor fluctuations. Dosing: MIRAPEX was titrated up to 4.5 mg/d. Analysis: primary endpoints were change from baseline to final maintenance visit of average "on" and "off" ratings for UPDRS parts II and III. Secondary endpoints included change from baseline to final maintenance visit of UPDRS parts I and IV.

†Multicenter, parallel-group, double-blind, randomized, controlled trial of 301 patients with early PD (Hoehn and Yahr stages I-III). Dosing: patients were titrated to a maximum dose of 4.5 mg MIRAPEX or 600 mg levodopa. Supplemental levodopa was permitted. Analysis: primary outcome variable was time until first occurrence of any 1 of 3 specified dopaminergic complications: wearing-off, dyskinesia, or "on-off" fluctuations. Secondary outcome variables included the Unified Parkinson's Disease Rating Scale (UPDRS).

References: 1. Lieberman A, Ranhosky A, Korts D. Clinical evaluation of pramipexole in advanced Parkinson's disease: results of a double-blind, placebo-controlled, parallel-group study. *Neurology*. 1997;49:162-168. **2.** Pinter MM, Pogarell O, Oertel WH. Efficacy, safety, and tolerance of the non-ergoline dopamine agonist pramipexole in the treatment of advanced Parkinson's disease: a double blind, placebo controlled, randomised, multicentre study. *J Neurol Neurosurg Psychiatry*. 1999;66:436-441. **3.** The Parkinson Study Group. Pramipexole vs levodopa as initial treatment for Parkinson disease: a 4-year randomized controlled trial. *Arch Neurol*. 2004;61:1044-1053.

Adjunctive MIRAPEX improves functioning while sparing levodopa[1,2]

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 activities of daily living, 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, extrapyramidal syndrome, hallucinations, headache, insomnia, somnolence, and nausea.

Please see Brief Summary of Prescribing Information on adjacent page.

Boehringer Ingelheim

Pfizer

Mirapex®
pramipexole dihydrochloride tablets

MP-10076/MR233100

Now and for the future

MIRAPEX® Tablets
brand of pramipexole dihydrochloride tablets

Brief Summary of Prescribing Information.

INDICATIONS AND USAGE

Treatment of the signs and symptoms of idiopathic Parkinson's disease.

CONTRAINDICATIONS

Demonstrated hypersensitivity to the drug or its ingredients.

WARNINGS

Falling Asleep During Activities of Daily Living: Patients treated with MIRAPEX 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 MIRAPEX, 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 the initiation of treatment. Somnolence is a common occurrence in patients receiving MIRAPEX at doses above 1.5 mg/day. Many clinical experts believe that falling asleep while engaged in activities of daily living always occurs in a setting of preexisting 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 MIRAPEX, patients should be advised of the potential to develop drowsiness and specifically asked about factors that may increase the risk with MIRAPEX, such as concomitant sedating medications, the presence of sleep disorders, and concomitant medications that increase pramipexole plasma levels (e.g., cimetidine—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.), MIRAPEX should ordinarily be discontinued. If a decision is made to continue MIRAPEX, patients should be advised to not drive and to avoid other potentially dangerous activities. While dose reduction clearly reduces the degree of somnolence, there is insufficient information to establish that dose reduction will eliminate episodes of falling asleep while engaged in activities of daily living.

Symptomatic hypotension: Carefully monitor Parkinson's disease patients treated with dopaminergic agonists for signs and symptoms of orthostatic hypotension, especially during dose escalation, and inform them of this risk (see **PRECAUTIONS**, *Information for Patients*). Despite clear orthostatic effects in normal volunteers, clinically significant orthostatic hypotension in clinical trials was not more frequent among those taking MIRAPEX Tablets than among those taking placebo. While this unexpected finding could reflect a unique property of pramipexole, it might also be due to study conditions and the nature of the clinical trial populations. Patients were carefully titrated, and those with active cardiovascular disease or significant orthostatic hypotension at baseline were excluded.

Hallucinations: In three double-blind, placebo-controlled trials in early Parkinson's disease, hallucinations were observed in 9% (35/388) of patients on MIRAPEX compared with 2.6% (6/235) of patients on placebo. In four double-blind, placebo-controlled trials in advanced Parkinson's disease where patients received MIRAPEX and concomitant levodopa, hallucinations were observed in 16.5% (43/260) of patients on MIRAPEX compared with 3.8% (10/264) of patients on placebo. Hallucinations caused treatment discontinuation in 3.1% of early Parkinson's disease patients and 2.7% of advanced Parkinson's disease patients compared with about 0.4% of placebo patients in both populations. Age appears to increase the risk of hallucinations attributable to pramipexole. In early Parkinson's disease patients, the risk of hallucinations was 1.9 times greater than placebo in patients <65 years and 6.8 times greater than placebo in patients >65 years. In advanced Parkinson's disease patients, the risk of hallucinations was 3.5 times greater than placebo in patients <65 years and 5.2 times greater than placebo in patients >65 years.

PRECAUTIONS

Rhabdomyolysis: A single case occurred in a 49-year-old man with advanced Parkinson's disease treated with MIRAPEX Tablets. The patient was hospitalized with an elevated CPK (10,631 IU/L). Symptoms resolved with medication discontinuation.

Renal: Exercise caution when prescribing MIRAPEX to patients with renal insufficiency (see full Prescribing Information, **DOSAGE AND ADMINISTRATION**).

Dyskinesia: MIRAPEX may potentiate the dopaminergic side effects of levodopa and may cause or exacerbate preexisting dyskinesia. Levodopa dose reduction may ameliorate this side effect.

Retinal pathology in albino rats: Pathologic changes (degeneration and loss of photoreceptor cells) were observed in the retina of albino rats in the 2-year carcinogenicity study. While retinal degeneration was not diagnosed in pigmented rats treated for 2 years, a thinning in the outer nuclear layer of the retina was slightly greater in rats given drug compared with controls. No similar changes were seen in albino mice, monkeys, and minipigs. 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 (i.e., disk shedding) may be involved (see full Prescribing Information, **ANIMAL TOXICOLOGY**).

Events Reported With Dopaminergic Therapy

Although the events listed below have not been reported in pramipexole clinical trials, they are associated with the use of other dopaminergic drugs. The expected incidence of these events, however, is so low that even if pramipexole caused these events at rates similar to those attributable to other dopaminergic therapies, it would be unlikely that even a single case would have occurred in a cohort of the size exposed to pramipexole in studies to date.

Withdrawal-emergent hyperpyrexia and confusion: 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 with rapid dose reduction, withdrawal of, or changes in antiparkinsonian 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 with drug discontinuation, 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.

Information for patients: Instruct patients to take MIRAPEX only as prescribed. Alert patients to the potential sedating effects associated with MIRAPEX, 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 MIRAPEX to gauge whether or not it affects their mental and/or motor performance adversely. Advise patients that if increased somnolence or new 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 MIRAPEX and when taking concomitant medications that increase plasma levels of pramipexole (e.g., cimetidine). Inform patients that hallucinations can occur and that the elderly are at a higher risk than younger patients. Patients may develop postural (orthostatic) hypotension with or without symptoms such as dizziness, nausea, fainting or blackouts, and, sometimes, sweating. Hypotension may occur more frequently during initial therapy. Accordingly, caution patients 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. Because the teratogenic potential of pramipexole is not completely established, and because experience in humans is limited, advise patients to notify their physicians if they become pregnant or intend to become pregnant during therapy (see **PRECAUTIONS**, *Pregnancy*). Because pramipexole may be excreted in breast milk, advise patients to notify their physicians if they intend to breast-feed or are breast-feeding an infant. Advise patients who develop nausea that taking MIRAPEX with food may reduce the occurrence of nausea.

Laboratory tests: During the development program, no systematic abnormalities on routine laboratory testing were noted. Therefore, no specific guidance is offered regarding routine monitoring; the practitioner retains responsibility for determining how best to monitor the patient.

Drug Interactions

Carbidopa/levodopa: Carbidopa/levodopa did not influence pramipexole pharmacokinetics in healthy volunteers. Pramipexole did not alter the extent of absorption (AUC) or the elimination of carbidopa/levodopa, although it caused an increase in levodopa C_{max} by about 40% and a decrease in T_{max} from 2.5 to 0.5 hours.

Selegiline: Selegiline did not influence pramipexole pharmacokinetics in healthy volunteers.

Amantadine: Population pharmacokinetic analysis suggests that amantadine is unlikely to alter oral pramipexole clearance.

Cimetidine: Cimetidine, a known inhibitor of renal tubular secretion of organic bases via the cationic transport system, caused a 50% increase in pramipexole AUC and a 40% increase in half-life.

Probenecid: Probenecid, a known inhibitor of renal tubular secretion of organic acids via the anionic transporter, did not noticeably influence pramipexole pharmacokinetics.

Other drugs eliminated via renal secretion: Population pharmacokinetic analysis suggests that coadministration of drugs secreted by cationic transport (e.g., cimetidine, ranitidine, diltiazem, triamterene, verapamil, quinidine, and quinine) decreases oral pramipexole clearance by about 20%, while those secreted by anionic transport (e.g., cephalosporins, penicillins, indomethacin, hydrochlorothiazide, and chlorpropamide) are likely to have little effect on oral pramipexole clearance.

CYP interactions: Cytochrome P450 enzyme inhibitors are not expected to affect pramipexole elimination, because pramipexole is not appreciably metabolized by these enzymes in vivo or in vitro. Studies indicate that pramipexole will not inhibit CYP enzymes at plasma concentrations observed following the highest recommended clinical dose (1.5 mg tid).

Dopamine antagonists: Dopamine antagonists, such as the neuroleptics (phenothiazines, butyrophenones, thioxanthenes) or metoclopramide, may diminish the effectiveness of MIRAPEX.

Drug/laboratory test interactions: No known interactions.

Carcinogenesis, mutagenesis, fertility impairment: Two-year pramipexole carcinogenicity studies were conducted in mice and rats. Pramipexole was fed to Chbb:NMRI mice at doses 0.3, 2.2, and 11 times the highest recommended human dose (1.5 mg tid) on a mg/m² basis and to Wistar rats at doses resulting in plasma AUCs equal to 0.3, 2.5, and 12.5 times the AUC in humans receiving 1.5 mg tid. No significant increases in tumors occurred in either species. Pramipexole was not mutagenic or clastogenic in the in vitro Ames assay, V79 gene mutation assay for HGPRT mutants, chromosomal aberration assay in Chinese hamster ovary cells, and in vivo mouse micronucleus assay. In rat fertility studies, a pramipexole dose 5.4 times the highest human dose on a mg/m² basis prolonged estrus cycles and inhibited implantation. These effects were associated with reduced serum prolactin levels, a hormone necessary for implantation and maintenance of early pregnancy in rats.

Pregnancy: Pregnancy Category C. Pramipexole given to female rats throughout pregnancy inhibited implantation at a dose 5.4 times the highest human dose on a mg/m² basis. Pregnant rats given pramipexole during the period of organogenesis (gestation days 7 through 16) at a dose resulting in a plasma AUC 4.3 times the AUC in humans receiving 1.5 mg tid resulted in a high incidence of total resorption of embryos. These findings are probably due to pramipexole's prolactin-lowering effect, since prolactin is necessary for implantation and maintenance of early pregnancy in rats (but not rabbits or humans). Because of pregnancy disruption and early embryonic loss in these studies, pramipexole's teratogenic potential could not be adequately evaluated. In pregnant rabbits given pramipexole during organogenesis, there was no evidence of adverse effects on embryo-fetal development following administration of doses resulting in a plasma AUC 71 times the AUC in humans receiving 1.5 mg tid. Postnatal growth was inhibited in the offspring of rats treated with a dose approximately equivalent to the highest human dose on a mg/m² basis or greater during latter pregnancy and throughout lactation. Pramipexole was not studied in human pregnancy. Because animal reproduction studies are not always predictive of human response, use pramipexole during pregnancy only if the potential benefit outweighs the potential risk to the fetus.

Nursing mothers: A single-dose, radio-labeled study showed that drug-related materials were excreted into breast milk of lactating rats. Radioactivity concentrations in milk were three to six times higher than plasma concentrations at equivalent time points. Other studies have shown that pramipexole inhibits prolactin secretion in humans and rats. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because pramipexole may cause potentially serious adverse reactions in nursing infants, a decision should be made to discontinue nursing or discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric use: Safety and efficacy have not been established.

Geriatric use: Pramipexole total oral clearance was approximately 30% lower in subjects >65 years compared with younger subjects because of a decline in pramipexole renal clearance due to an age-related reduction in renal function. This resulted in an increase in elimination half-life from approximately 8.5 hours to 12 hours. In clinical studies, 38.7% of patients were >65 years. There were no apparent differences in efficacy or safety between older and younger patients, except that the relative risk of hallucination associated with MIRAPEX was increased in the elderly.

ADVERSE REACTIONS

Patients with either early or advanced Parkinson's disease were enrolled in clinical trials. Apart from disease severity and duration, the two populations differed in use of concomitant levodopa. Patients with early disease did not receive concomitant levodopa during treatment with pramipexole; those with advanced Parkinson's disease all received concomitant levodopa. Because these two populations may have differential risks for various adverse events, data are presented separately by population. Because all premarketing controlled trials used a titration design, confounding time and dose, it is impossible to adequately evaluate effects of dose on incidence of adverse events.

Early Parkinson's Disease

In three double-blind, placebo-controlled trials of patients with early Parkinson's disease, the most commonly observed adverse events (>5%) that were more frequent in the group treated with MIRAPEX Tablets were nausea, dizziness, somnolence, insomnia, constipation, asthenia, and hallucinations.

In double-blind, placebo-controlled trials, approximately 12% of 388 patients treated with MIRAPEX discontinued treatment due to adverse events compared with 11% of 235 patients who received placebo. Adverse events most commonly causing discontinuation for MIRAPEX and placebo, respectively, were hallucinations (3.1% vs 0.4%), dizziness (2.1% vs 1%), somnolence (1.6% vs 0%), extrapyramidal syndrome (1.6% vs 6.4%), headache (1.3% vs 0%), confusion (1.0% vs 0%), and nausea (2.1% vs 0.4%).

Adverse-event incidence in controlled clinical studies in early Parkinson's disease: Table 1 lists treatment-emergent adverse events in double-blind, placebo-controlled studies that were reported by ≥1% of patients treated with MIRAPEX and were more frequent than in the placebo group. Adverse-event intensity was usually mild or moderate. These figures cannot be used to predict adverse-event incidence in usual medical practice where patient characteristics and other factors differ from those in clinical studies. Similarly, the cited frequencies cannot be compared with figures obtained from other clinical investigations. However, the cited figures do provide some basis for estimating the relative contribution of drug and nondrug factors to the adverse-event incidence rate in the population studied.

Table 1.—Treatment-Emergent Adverse-Event* Incidence in Double-Blind, Placebo-Controlled Trials in Early Parkinson's Disease (Events ≥1% of Patients Treated With MIRAPEX and Numerically More Frequent Than in the Placebo Group)

Body System/ Adverse Event	MIRAPEX N=388	Placebo N=235	Body System/ Adverse Event	MIRAPEX N=388	Placebo N=235
Body as a Whole			Nervous System		
Asthenia	14	12	Dizziness	25	24
General edema	5	3	Somnolence	22	9
Malaise	2	1	Insomnia	17	12
Reaction unevaluable	2	1	Hallucinations	9	3
Fever	1	0	Confusion	4	1
Digestive System			Amnesia	4	2
Nausea	28	18	Hypesthesia	3	1
Constipation	14	6	Dystonia	2	1
Anorexia	4	2	Akathisia	2	0
Dysphagia	2	0	Thinking abnormalities	2	0
Metabolic & Nutritional System			Decreased libido	1	0
Peripheral edema	5	4	Myoclonus	1	0
Decreased weight	2	0	Special Senses		
			Vision abnormalities	3	0
			Urogenital System		
			Impotence	2	1

*Patients may have reported multiple adverse experiences during the study or at discontinuation; thus, patients may be included in more than one category.

Other events reported by ≥1% of patients treated with MIRAPEX but reported equally or more frequently in the placebo group were infection, accidental injury, headache, pain, tremor, back pain, syncope, postural hypotension, hypertonia, depression, abdominal pain, anxiety, dyspepsia, flatulence, diarrhea, rash, ataxia, dry mouth, extrapyramidal syndrome, leg cramps, twitching, pharyngitis, sinusitis, sweating, rhinitis, urinary tract infection, vasodilation, flu syndrome, increased saliva, tooth disease, dyspnea, increased cough, gait abnormalities, urinary frequency, vomiting, allergic reaction, hypertension, pruritus, hypokinesia, increased creatine PK, nervousness, dream abnormalities, chest pain, neck pain, paresthesia, tachycardia, vertigo, voice alteration, conjunctivitis, paralysis, accommodation abnormalities, tinnitus, diplopia, and taste perversions. In a fixed-dose study in early Parkinson's disease, occurrence of the following events increased in frequency as the dose increased over the range from 1.5 mg/day to 6 mg/day: postural hypotension, nausea, constipation, somnolence, and amnesia. The frequency of these events was generally 2-fold greater than placebo for pramipexole doses greater than 3 mg/day. The incidence of somnolence with pramipexole at a dose of 1.5 mg/day was comparable to that reported for placebo.

Advanced Parkinson's Disease

In four double-blind, placebo-controlled trials of patients with advanced Parkinson's disease, the most commonly observed adverse events (>5%) that were more frequent in the group treated with MIRAPEX and concomitant levodopa were postural (orthostatic) hypotension, dyskinesia, extrapyramidal syndrome, insomnia, dizziness, hallucinations, accidental injury, dream abnormalities, confusion, constipation, asthenia, somnolence, dystonia, gait abnormality, hypertonia, dry mouth, amnesia, and urinary frequency. Approximately 12% of 260 patients with advanced Parkinson's disease who received MIRAPEX and concomitant levodopa in double-blind, placebo-controlled trials discontinued treatment due to adverse events compared with 16% of 264 patients who received placebo and concomitant levodopa. Events most commonly causing treatment discontinuation for MIRAPEX and placebo, respectively, were hallucinations (2.7% vs 0.4%), dyskinesia (1.9% vs 0.8%), extrapyramidal syndrome (1.5% vs 4.9%), dizziness (1.2% vs 1.5%), confusion (1.2% vs 2.3%), and postural (orthostatic) hypotension (2.3% vs 1.1%).

Adverse-event incidence in controlled clinical studies in advanced Parkinson's disease: Table 2 lists treatment-emergent adverse events that occurred in the double-blind, placebo-controlled studies that were reported by ≥1% of patients treated with MIRAPEX and were more frequent than in the placebo group. In these studies, MIRAPEX or placebo was administered to patients who were also receiving concomitant levodopa. Adverse-event intensity was usually mild or moderate. These figures cannot be used to predict adverse-event incidence in usual medical practice where patient characteristics and other factors differ from those in clinical studies. Similarly, the cited frequencies cannot be compared with figures obtained from other clinical investigations. However, the cited figures do provide some basis for estimating the relative contribution of drug and nondrug factors to the adverse-events incidence rate in the population studied.

Table 2.—Treatment-Emergent Adverse-Event* Incidence in Double-Blind, Placebo-Controlled Trials in Advanced Parkinson's Disease (Events ≥1% of Patients Treated With MIRAPEX and Numerically More Frequent Than in the Placebo Group)

Body System/ Adverse Event	MIRAPEX† N=260	Placebo† N=264	Body System/ Adverse Event	MIRAPEX† N=260	Placebo† N=264
Body as a Whole			Nervous System (cont)		
Accidental injury	17	15	Somnolence	9	6
Asthenia	10	8	Dystonia	8	7
General edema	4	3	Gait abnormalities	7	5
Chest pain	3	2	Hypertonia	7	6
Malaise	3	2	Amnesia	6	4
Cardiovascular System			Akathisia	3	2
Postural hypotension	53	48	Thinking abnormalities	3	2
Digestive System			Paranoid reaction	2	0
Constipation	10	9	Delusions	1	0
Dry mouth	7	3	Sleep disorders	1	0
Metabolic & Nutritional System			Respiratory System		
Peripheral edema	2	1	Dyspnea	4	3
Increased creatine PK	1	0	Rhinitis	3	1
Musculoskeletal System			Pneumonia	2	0
Arthritis	3	1	Skin & Appendages		
Twitching	2	0	Skin disorders	2	1
Bursitis	2	0	Special Senses		
Myasthenia	1	0	Accommodation abnormalities	4	2
Nervous System			Vision abnormalities	3	1
Dyskinesia	47	31	Diplopia	1	0
Extrapyramidal syndrome	28	26	Urogenital System		
Insomnia	27	22	Urinary frequency	6	3
Dizziness	26	25	Urinary tract infection	4	3
Hallucinations	17	4	Urinary incontinence	2	1
Dream abnormalities	11	10			
Confusion	10	7			

*Patients may have reported multiple adverse experiences during the study or at discontinuation; thus, patients may be included in more than one category.
†Patients received concomitant levodopa.

Other events reported by ≥1% of patients treated with MIRAPEX but reported equally or more frequently in the placebo group were nausea, pain, infection, headache, depression, tremor, hypokinesia, anorexia, back pain, dyspepsia, flatulence, ataxia, flu syndrome, sinusitis, diarrhea, myalgia, abdominal pain, anxiety, rash, paresthesia, hypertension, increased saliva, tooth disorder, apathy, hypotension, sweating, vasodilation, vomiting, increased cough, nervousness, pruritus, hypesthesia, neck pain, syncope, arthralgia, dysphagia, palpitations, pharyngitis, vertigo, leg cramps, conjunctivitis, and lacrimation disorders.

Adverse events—relationship to age, gender, and race: Among the treatment-emergent adverse events in patients treated with MIRAPEX, hallucination appeared to exhibit a positive relationship to age. No gender-related differences were observed. An evaluation of adverse events related to race was not possible (only 4% non-Caucasian enrollees).

Other adverse events observed during all phase 2 and 3 clinical trials: 1,408 individuals received MIRAPEX during all clinical trials (Parkinson's disease and other patient populations), 648 of whom were in seven double-blind, placebo-controlled Parkinson's disease trials. During these trials, all adverse events were recorded by the clinical investigators using their own terminology. Listed below are similar types of events, grouped into a smaller number of standardized categories using modified COSTART dictionary terminology. These events occurred in <1% of the 1,408 individuals exposed to MIRAPEX and occurred on at least two occasions (one if the event was serious). All reported events, except those already listed above, are included without regard to determination of a causal relationship to MIRAPEX. Events are listed within body-system categories in order of decreasing frequency.

Body as a Whole: enlarged abdomen, death, fever, and suicide attempt. ***Cardiovascular System:*** peripheral vascular disease, myocardial infarction, angina pectoris, atrial fibrillation, heart failure, arrhythmia, atrial arrhythmia, and pulmonary embolism. ***Digestive System:*** thirst. ***Musculoskeletal System:*** joint disorder and myasthenia. ***Nervous System:*** agitation, CNS stimulation, hyperkinesia, psychosis, and convulsions. ***Respiratory System:*** pneumonia. ***Special Senses:*** cataract, eye disorder, and glaucoma. ***Urogenital System:*** dysuria, abnormal ejaculation, prostate cancer, hematuria, and prostate disorder. ***Falling Asleep During Activities of Daily Living:*** Patients treated with MIRAPEX have reported falling asleep while engaged in activities of daily living, including operation of a motor vehicle, which sometimes resulted in accidents (see bolded WARNING). ***Post-Marketing Experience:*** In addition to the adverse events reported during clinical trials, the following adverse reactions have been identified during post-approval use of MIRAPEX Tablets. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure. Decisions to include these reactions in labeling are typically based on one or more of the following factors: (1) seriousness of the reaction, (2) frequency of reporting, or (3) strength of causal connection to MIRAPEX Tablets. Similar types of events were grouped into a smaller number of standardized categories using the MedDRA dictionary: accidents (including fall), compulsive behaviors (including sexual and pathological gambling), fatigue, hallucinations (all kinds), headache, hypotension (including postural hypotension), libido disorders, syncope, and blackouts.

DRUG ABUSE AND DEPENDENCE

Pramipexole is not a controlled substance. Although not systematically studied for abuse, tolerance, or physical dependence potential, pramipexole had little or no effect in a rat model of cocaine self-administration.

OVERDOSAGE

There is no clinical experience with massive overdosage. No adverse events were reported when one patient took 11 mg/day of pramipexole for 2 days (two to three times the protocol recommended daily dose). Blood pressure remained stable, although pulse rate increased to between 100 and 120 beats/minute. The patient withdrew from the study at the end of week 2 due to lack of efficacy. There is no known antidote for dopamine agonist overdosage. If signs of CNS stimulation are present, a phenothiazine or other butyrophenone neuroleptic agent may be indicated; but efficacy in reversing overdosage effects has not been assessed. General supportive measures along with gastric lavage, intravenous fluids, and electrocardiogram monitoring may be required.

DOSAGE AND ADMINISTRATION

In all clinical studies, dosage was initiated at a subtherapeutic level to avoid intolerable adverse effects and orthostatic hypotension. Gradually titrate dosage in all patients. Increase dosage to achieve a maximum therapeutic effect, balanced against the principal side effects of dyskinesia, hallucinations, somnolence, and dry mouth.

Dosing in Patients With Normal Renal Function

Initial treatment: Increase dosages gradually, i.e., not more frequently than every 5 to 7 days, from a starting dose of 0.375 mg/day given in three divided doses. Refer to full Prescribing Information for the suggested ascending dosage schedule that was used in clinical studies.

Maintenance treatment: MIRAPEX Tablets were effective and well tolerated over a dosage range of 1.5 to 4.5 mg/day, administered in equally divided doses three times per day with or without concomitant levodopa (approximately 800 mg/day). In a fixed-dose study in early Parkinson's disease patients, doses of 3 mg, 4.5 mg, and 6 mg per day of MIRAPEX were not shown to provide any significant benefit beyond that achieved at a daily dose of 1.5 mg/day. However, in the same fixed-dose study, the following adverse events were dose related: postural hypotension, nausea, constipation, somnolence, and amnesia. The frequency of these events was generally 2-fold greater than placebo for pramipexole doses greater than 3 mg/day. The incidence of somnolence reported with pramipexole at a dose of 1.5 mg/day was comparable to placebo. Consider reducing levodopa dosage when MIRAPEX is used in combination. In a controlled study in advanced Parkinson's disease, levodopa dosage was reduced by an average of 27% from baseline.

Patients with renal impairment: Dosage adjustments are recommended for patients with moderate to severe impairment (see full Prescribing Information, **DOSAGE AND ADMINISTRATION**). The use of MIRAPEX has not been studied in patients with very severe impairment.

Treatment discontinuation: Discontinue MIRAPEX over a period of 1 week; in some studies, however, abrupt discontinuation was uneventful.

Store at 25°C (77°F); excursions permitted to 15°C to 30°C (59°F to 86°F) [see USP Controlled Room Temperature]. Protect from light.

Store in a safe place out of the reach of children.

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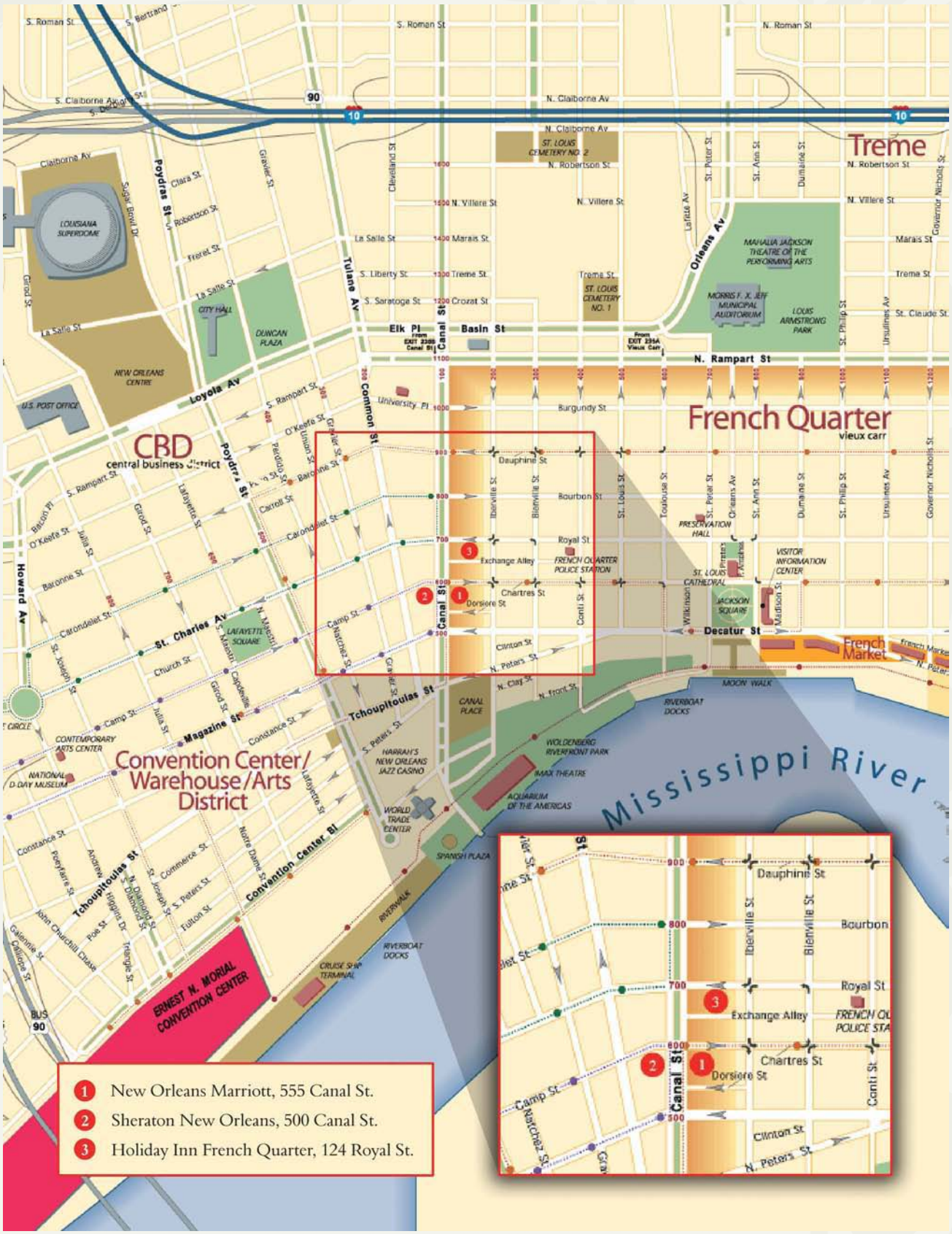

MOMENTS

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*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, Tremor Control Therapy, and Dystonia 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.

Dystonia Therapy: Unilateral or bilateral stimulation of the internal globus pallidus (GPi) or the subthalamic nucleus (STN) by the Medtronic Activa System is indicated as an aid in the management of chronic, intractable (drug refractory) primary dystonia, including generalized and segmental dystonia, hemidystonia, and cervical dystonia (torticollis), for individuals 7 years of age and older.

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 who are unable to properly operate the neurostimulator, or for Parkinson's disease and Essential Tremor, patients for whom test stimulation is unsuccessful. 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). For patients with Dystonia, age of implant is suggested to be that at which brain growth is approximately 90% complete or above. Additionally, the abrupt cessation of stimulation for any reason should be avoided as it may cause a return of disease symptoms. In some cases, symptoms may return with an intensity greater than was experienced prior to system implant ("rebound" effect). 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.

Humanitarian Device (Dystonia Therapy): Authorized by Federal Law for the use as an aid in the management of chronic, intractable (drug refractory) primary dystonia, including generalized and segmental dystonia, hemidystonia, and cervical dystonia (torticollis), for individuals 7 years of age and older.

For further information, please call Medtronic at 1-800-633-8766.

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Poster Session 1

Sunday, March 6, 2005

Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:45 p.m. to 2:30 p.m.
Abstracts: 1-187

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1 **SCA 8 presenting with cervical dystonia and parkinsonism (case report)**
F. I. Khan, R. L. Scales

2 **Quantitation of ramp tracking accuracy and tapping rhythm variability in ataxic and control subjects**
J. W.S. Wong, K. G. Tuck, D. J. Needham, B. McGragh, E. Storey

3 **Fragile–X–associated tremor/ataxia syndrome presenting in a woman on chemotherapy**
J. P. O'Dwyer, C. Clabby, J. C. Crown, D. E. Barton, M. Hutchinson

4 **A new form of tauopathy: clinico–pathologic correlation in a case with syndrome of progressive ataxia and palatal tremor (PAPT)**
Z. Mari, A. Vortmeyer, V. Zhukareva, K. Uryu, V. Lee, M. Hallett

5 **Particular phenotypes of autosomal dominant spinocerebellar ataxias in Russian families**
S. N. Illarioshkin, S. A. Klyushnikov, T. N. Proskokova, L. V. Novikova, I. A. Ivanova-Smolenskaya, E. D. Markova

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Poster numbers 6-16

6 **Premotor control of orbicularis oculi motoneurons by the trigeminal nuclear complex**
S. Gong, M. DeCuypere, M. S. LeDoux

7 **Biochemical differences exist between clinical phenotypes of progressive supranuclear palsy: Richardson's syndrome and PSP–Parkinsonism**
D. R. Williams, R. de Silva, A. J. Lees

8 **Changes of homotopic and heterotopic short–latency afferent inhibition on a surround muscle during finger movement**
B. Voller, A. St Clair Gibson, S. Pirio Richardson, M. Lomarev, E. Auff, M. Hallett

9 **Striosome–dominant neuronal activity in dorsolateral striatum is correlated with the severity of dyskinesia in a rat model of parkinsonism**
E. Saka, A. M. Graybiel

10 **Early– and higher–order projections from the primate cerebellum to orbicularis oculi motoneurons**
M. DeCuypere, S. Gong, M. LeDoux

11 **Expression of an heparin–binding growth associated molecule (pleiotrophin) by striatal interneurons and nigral dopaminergic neurons**
I. R. Taravini, J. E. Ferrario, J. Courty, G. M. Murer, R. Raisman, O. S. Gershanik

12 **Impairment of protein degradation in PC12 cells expressing mutant a–synuclein**
K. Vekrellis, A. M. Cuervo, L. Stefanis

13 **Pathoarchitectonic staging of sporadic Parkinson's disease brains**
K. Del Tredici, R. de Vos, U. Rueb, E. Jansen Steur, H. Braak

14 **Silencing of UCP5 enhances MPP^+–induced neurotoxicity in SH–SY5Y cells**
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15 **Distribution of Lewy pathology in the basal ganglia of the Lewy body disorders suggests involvement in neuropsychiatric symptoms**
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16 **Full and partial dopamine agonists and the relationship to dyskinesia priming in MPTP–treated marmosets**
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S. Kamath, N. Bajaj

18 **Motor and cognitive impairment in Huntingtons disease**
J. Klempir, J. Roth, N. Spackova, O. Novakova, R. Jech, E. Ruzicka

19 **Longitudinal study in Huntington's disease patients with IBZM–SPECT**
S. Lanfranchi, M. Perini, E. Viganò, C. Gobbo

20 **McLeod syndrome presenting with hepatic disease**
R. H. Walker, A. Danek, H. H. Jung, R. Davey, M. Reid

21 **Drug–enhanced acantocytic chorea: a case report**
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A. L. Teixeira, D. P. Maia, F. Cardoso

23 **Vocal tics in Sydenham's chorea**
A. L. Teixeira, D. P. Maia, F. Cardoso

24 **The UFMG Sydenham's chorea rating scale (USCRS): discrimination properties**
A. L. Teixeira, D. P. Maia, F. Cardoso

25 **Efficacy and safety of risperidone in the treatment of Sydenham's chorea**
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26 **Persistent bradykinesia following Sydenham's chorea**
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27 **Co–occurrence of seizure and chorea in a patient with nonketotic hyperglycemia**
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29 **Longitudinal changes in MRI and neuropsychological measures in early Huntington's disease – a pilot study**
S. M. Henley, T. T. Warner, E. K. Warrington, J. M. Stevens, J. Ashburner, N. C. Fox

30 **Falling in Huntington's disease (HD)**
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33 **A pilot study of the clinical efficacy and safety of memantine for Huntington's disease**
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40 **Disturbed surround inhibition in idiopathic Parkinson's disease during voluntary movement**
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41 **Contribution of transcranial magnetic stimulation to the differential diagnosis of parkinsonian syndromes. A comparative study**
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42 **Evaluation of remote effects of chronic botulinum toxin type a treatment by single fibre electromyography**
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48 **Effect of typical and atypical neuroleptics on nigral NFkB expression in an experimental model of tardive dyskinesia**
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49 **Incidence of presumptive tardive dyskinesia in elderly patients treated with olanzapine or conventional antipsychotics**
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50 **Cholinergic stimulation does not worsen impaired complex motion performance in cholinergic neurodegeneration**
S. Muhlack, H. Przuntek, F. Tracik, T. Müller

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56 **Dyskinesia occurs in 34% of Parkinson's disease patients receiving dopaminergic treatment: findings from a retrospective international study**
T. Mueller, H. Russ, D. Haeger

57 **Drug–induced movement disorders caused by medications other than dopamine receptor blockers in the Veteran population**
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58 **Results of a series of 55 consecutive patients with movement disorders treated by bilateral micro–electrode–guided stereotactic STN, GPI and VIM – DBS with a time – saving intraoperative protocol of reduced teststimulation, looking mainly for stimulation – induced side effects**
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61 **Improvement of cervical dystonia: role of transcranial magnetic stimulation simulating sensory tricks effect**
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62 **Long–term, prospective, open label trial of botulinum toxin type B in cervical dystonia**
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67 **Motor imagery in patients with cervical dystonia, an fMRI study**
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68 **Spinal axial Injections of botulinum toxin in generalized dystonia: two case reports**
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69 **Impaired motor learning in mice expressing torsinA with the DYT1 dystonia mutation**
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73 **Evaluation of the Methylentetrahydrofolate reductase (MTHFR) gene in patients with primary dystonia**
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74 **Motor system inhibition in dopa–responsive dystonia and its modulation by treatment**
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75 **Prevalence of cervical dystonia and spasmodic torticollis in the U.S**
J. Jankovic, J. Tsui, C. Bergeron

76 **An unusual phenotype in dYT1 dystonia: stiff man syndrome**
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77 **Botulinum toxin type–*A* and pain responsiveness in cervical dystonia: a dose rasponse study**
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78 **Measuring clinical change in cervical dystonia: a comparison of the CDIP–58 with existing rating scales**
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79 **An enigmatic relationship between dopamine and intracellular purine metabolism**
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80 **The usefulness of SPECT DAT scans in differentiating dystonic rest tremor from Parkinson's disease: some illustrative cases**
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81 **Adult–onset primary lower limb dystonia: clinical description of a rare entity**
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82 **DYT1 and dopa–responsive dystonia during menses and pregnancy**
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83 **Motor system inhibition in dopa–responsive dystonia and its modulation by levodopa treatment**
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84 **Determination of the contribution of the clinical examination and the EMG in planning and application of botulinum toxin treatment in cervical dystonia**
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85 **High frequency oscillations (HFOs) in focal dystonia: abnormal patterns sign for the disease**
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89 **CADASIL presenting as a movement disorder: a clinical study of a chilean kindred**
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92 **Contrasting effect of bilateral dorsal versus ventral pallidal stimulation in primary generalized dystonia**
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95 **Chorea as the initial presentation of dopa–responsive dystonia**
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96 **Secondary dystonia caused by chronic encapsulated intracerebral hematoma in the right frontal lobe–case report–**
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98 **Increased self–reported stress decreases subjective improvement following botulinum toxin injections for cervical dystonia**
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99 **Sensory abnormalities in unaffected relatives in familial adult–onset primary torsion dystonia: a surrogate phenotype marker?**
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100 **Long–term botulinum toxin treatment for focal hand dystonia**
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101 **Usefulness of botulinum toxin–A in psychogenic dystonia**
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102 **A novel, autosomal dominant, complicated spastic paraplegia in adults with genetic anticipation and severe generalized dystonia in childhood**
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103 **A direct comparison of botulinum toxin type A and type B in cervical dystonia: a multicenter, randomized, double blind, parallel study**
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104 **Evaluation of the lerman minerva cervical orthosis for treatment of cervical and upper truncal dystonias**
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105 **Botulinum toxin type B observational study (BOS)**
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107 **Novel expression of a visual "geste antagoniste" in cervical dystonia**
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108 **The influence of two different injection strategies in the long–term treatment of cervical dystonia with dysport®**
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109 **Successful treatment of a patient with tardive dystonia with stereotactic bilateral GPI deep brain stimulation**
F. Hertel, C. Decker, M. Zuchner, M. Pohlau, M. Bettag

110 **Successful treatment of hemidystonia due to striatal lesions by bilateral stereotactic GPI – DBS – a report of 2 cases**
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111 **Characterization of a rodent model of dystonia**
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112 **Increased microglial activation following neural transplantation in Huntington's disease: an ^{11}C–(R)PK11195 PET study**
Y. F. Tai, N. Pavese, R. A. Barker, S. B. Dunnett, D. J. Brooks, P. Piccini

113 **Movement disorder models via viral vector gene transfer to adult rats and targets for gene therapy**
R. L. Klein, R. Dayton, R. Zweig, W.-L. Lin, D. W. Dickson

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114 **A novel mutation (K317M) in the *MAPT* gene causes a familial form of Parkinsonism–ALS–Dementia complex**
J. J. Zarranz, I. Ferrer, E. Lezcano, I. Forcadas, J. Pérez-Tur, B. Atares

115 **Frequency of *PINK1*, *Parkin,* and *DJ–1* mutations in early–onset parkinsonism**
A. Hiller, A. Djarmati, K. Hedrich, P. P. Pramstaller, P. Martinelli, C. Klein

116 **A case of maternally transmitted juvenile Huntington's disease (HD) with a very large trinucleotide repeat**
S. Papapetropoulos, R. Lopez-Alberola, L. Baumbach, A. Russell, M. A. Gonzalez, C. CInger

117 **Population–based confirmation of the 12q RLS locus in Iceland**
A. A. Hicks, D. B. Rye, K. Kristjansson, T. Sigmundsson, A. P. Sigurdsson, I. Eiriksdottir

118 **Linkage disequilibrium fine–mapping and haplotype association analysis of the *tau* gene in progressive supranuclear palsy and corticobasal degenerarion**
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119 **e–sarcoglycan expression in skeletal muscle from patients with myoclonus–dystonia syndrome (MDS)**
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120 **Canine multiple system degeneration locus maps to the interval containing the PARK2 ortholog**
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121 **PINK1 mutations in early–onset and recessive Parkinson's disease in Norway**
M. Toft, L. Pielsticker, I. F. Mata, J. T. Stone, J. Aasly, M. Farrer

122 **Metabolic gene polymorphisms in a population–based case control study of Parkinson's disease**
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123 **MRI, motor and cognitive measures correlate with predicted years to disease onset in presymptomatic HD: the predict–HD study**
C. A. Ross, K. Biglan, E. Aylward, J. C. Stout, D. R. Langbehn, J. S. Paulsen

124 **Huntington's disease in Russia: ten–years experience of genetic testing and management of affected families**
S. A. Klyushnikov, S. N. Illarioshkin, I. A. Ivanova-Smolenskaya, E. D. Markova, P. A. Fedin

125 **A large family with idiopathic basal ganglia calcifications not linked to chromosome 14q**
C. B. Volpato, E. Buffone, A. De Grandi, F. Tezzon, G. Casari, P. P. Pramstaller

126 **Lewy body parkinsonism in a large pedigree with 77 *Parkin* mutation carriers**
P. P. Pramstaller, T. S. Jacques, F. Scaravilli, K. Hedrich, P. L. Kramer, C. Klein

127 **A novel *LRRK2* mutation is a common cause of autosomal dominant parkinsonism in families from several european populations**
M. Toft, J. Kachergus, I. F. Mata, M. Hulihan, J. Aasly, M. Farrer

128 **Glucocerebrosidase gene mutations are not associated with Parkinson's disease in the Norwegian population**
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129 **Post–traumatic rhythmic brainstem myoclonus in the outpatient clinic**
S. Jain, S. J. Frucht, P. E. Greene

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130 **Effects of subthalamic nucleus stimulation on random number generation in Parkinson's disease**
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131 **Hemispheric lateralization of normal and impaired praxis movements: an event–related functional MRI study**
S. Bohlhalter, E. Fridman, L. Wheaton, N. Hattori, J. Grafman, M. Hallett

132 **Magnetic resonance spectroscopy in differential diagnosis of parkinsonism**
L. F. Vasconcellos, S. P. Novis, D. M. Moreira, A. L. Rosso, A. C. Leite

133 **Reduced sensorimotor activation in laryngeal dystonia: a 'silent event related' fMRI study**
B. Haslinger, P. Erhard, F. Castrop, M. Roettinger, A. O. Ceballos-Baumann

134 **Eye–of–the–tiger sign in an asymptomatic patient**
J. C. Martinez-Castrillo, J. Masjuan, B. Pilo, P. Simal, J. Martinez-Sanmillan

135 **fMRI studies investigating the neural bases of executive dysfunctions in Parkinson's disease**
O. Monchi, M. Petrides, J. Doyon, A. P. Strafella

136 **Accuracy of ^{123}I–ioflupane (DaTSCAN™) in the diagnosis of patients with clinically uncertain parkinsonism: a two year follow up study**
E. Tolosa, T. vander Borght, The CUPS Study Group

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137 Validation study of cardiac 123I–MIBG scintigraphy in Juntendo University
H. Mochizuki, M. Komine-Kobayashi, K. Sato, Y. Mizuno

138 Striatal and extrastriatal dopamine transporter availability in patients with SCA2 mutations. A [123I]b–CIT SPECT study
C. Scherfler, S. M. Boesch, E. Donnemiller, K. Seppi, G. K. Wenning, W. Poewe

139 3D–SSP SPECT before and after onset of dementia in cases originally diagnosed as Parkinson's disease
Y. Osaki, Y. Morita, M. Fukumoto, N. Akagi, S. Yoshida, Y. Doi

140 Neuroprotection and natural history in Parkinson plus syndromes study (NNIPPS): results at inclusion of the magnetic resonance imaging (MRI) study in progressive supranuclear palsy (PSP) and multiple system atrophy (MSA)
M. Verin, Y. Rolland, C. Payan, G. Bensimon, The NNIPPS Study Group, UK, Germany

141 In vivo measuring of the topographic profile of putaminal degeneration in the parkinson variant of multiple system atrophy: a diffusion MR study
K. Seppi, M. F.H. Schocke, K. J. Mair, R. Esterhammer, G. K. Wenning, W. Poewe

142 Relationship between non–motor signs and asymmetry of striatal dopamine transporter (DAT) loss in patients with early Parkinson's diseases measured with SPECT and (123I)Ioflupane–SPECT
I. U. Isaias, S. Hesse, R. Benti, M. Zini, J. Schwarz, A. Antonini

143 Hemifacial spasm: a nerve–vessel–conflict?
M. Kloss, B. Kress, C. Schwark, M. Krause

144 IBZM–SPECT in patients with parkinsonism: a prospective 3 years study
R. Cilia, M. Perini, A. Antonini, C. Gobbo

145 Brain parenchyma sonography differentiates RLS patients from normal–controls and patients with Parkinson's disease
C. Schmidauer, M. Sojer, H. Stockner, B. Högel, G. Wenning, W. Poewe

146 Dopamine transporter imaging with ^{123}I–FP–CIT SPECT in the differential diagnosis of dementia with Lewy bodies, Alzheimer's disease and other parkinsonism
P. Mir, J. J. Rodriguez-Uranga, E. Gil-Neciga, D. Garcia-Solis, F. Carrillo, R. Alberca

147 The amygdala of patients with Parkinson's disease is silent in response to fearful facial expressions
M. Kawamura, N. Yoshimura

148 An MEG/EEG study of tempo production in Parkinson's disease
Y. Cojan, L. Garnero, V. Pouthas, M. Vidailhet

149 In vivo assessment of Intrasynaptic dopamine in Parkinson patients following treatment with L–dopa or L–dopa/entacapone
J. P. Seibyl, R. Tabamo, D. Jennings, G. Tamagnan, A. Rabinowicz, K. Marek

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150 New onset diabetes in Parkinsonian patients on long–term quetiapine
K. McCown, J. Romrell, M. E. Trieschmann, J. H. Friedman, M. S Okun, H. H. Fernandez

151 Management of augmentation in patients with restless legs syndrome
C. Trenkwalder, M. Canelo

152 A retrospective "real life" study of pramipexole tolerability in young and old Parkinson's disease patients attending Movement Disorders clinic
L. Kwan, V. Dhawan, A. Forbes, G. Brand, K. R. Chaudhuri

153 Switch from Levodopa/Carbidopa to the corresponding Stalevo™ dosage improves motor symptoms and elevates levodopa plasma levels in patients with Parkinson's disease
T. Müller, C. Erdmann, S. Muhlack, D. Bremen, H. Przuntek, D. Woitalla

154 The retention rate, tolerability and efficacy on nighttime symptoms of stalevo as a switch or "add on" therapy in Parkinson's disease (PD) and restless legs syndrome (RLS)
V. Dhawan, G. Brand, A. Forbes, S. Tluk, K. R. Chaudhuri

155 The effect of quetiapine in Parkinson's disease (PD) psychotic patients: a double blind labeled study of 3 months duration
J. M. Rabey, T. Prokhorov, A. Miniovich, C. Klein

156 Clarithromycin, a CYP3A4 inhibitor, increased the antiparkinsonian effect of cabergoline, an ergote dopamine agonist in the treatment of Parkinson's desease
A. Nakatsuka, M. Nagai, H. Yabe, H. Moritoyo, T. Moritoyo, M. Nomoto

157 Tetrabenazine: does prior history of depression preclude this drug as a treatment option?
C. Hunter, K. Vuong, N. Mejia, J. Jankovic

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158 Excessive daytime sleepiness in Parkinson's disease patients: a prospective controlled study
P. Stathis, G. Hilakos, V. Vagenas, K. Hapibi, M. Maltezou

159 Taste sense is abnormal in idiopathic Parkinson's disease
M. Fernando, M. Shah, C. Hawkes

160 Smell identification declines from age 36 years and mainly affects pleasant odours
C. Hawkes, M. Shah, A. Fogo

161 Cognitive dysfunction in Machado–Joseph disease
J. Stokholm, J. Nielsen

162 Levodopa does not adversely affect cognition in parkinsonism
S. A. Molloy, J. T. O'Brien, I. G. McKeith, T. Manktelow, K. Wesnes, D. J. Burn

163 Self perception of age in older parkinsonian patients
H. D. Weiss

164 The use of the Parkinson's disease sleep scale (PDSS) in the assessment of "sleepy" Parkinson's disease patients with excessive daytime sleepiness (EDS) compared to controls
V. Dhawan, M. Baxi, L. Taurah, M.-J. Medrano, P. Martinez-Martin, K. R. Cahudhuri

165 Mortality and tardive dyskinesia: a long–term study utilizing the national death Index
C. E. Dean, P. D. Thuras

166 Pure autonomic failure: not such a pure entity? A propos of a case with altered striatal uptake on DATscan
Y. Compta, M. J. Marti, P. Paredes, A. Lladó, C. Gaig, E. Tolosa

167 Dopamine and the uncoupling of fronto–striatal circuits: from primate experiments to clinical studies
M. Pessiglione, B. Dubois, Y. Agid, L. Tremblay

168 An international multicentre study validating the first screening questionnaire (NMS–quest) for comprehensive assessment of non motor symptoms of Parkinson's disease
K. R. Chaudhuri, P. Martinez-Martin, A. H.V. Schapira, W. Ondo, K. Sethi, Members of The PD NMS Scale Development Group

169 Cognitive evaluation of a population of patients with Parkinson's disease with mini mental Parkinson test
P. Chana, T. Parrao, C. Juri, C. Kunstmann, J. Tapia, D. Alburquerque

170 Lateralization of STN DBS's effects on cognitive control
T. Hershey, D. C. Perantie, S. L. Warren, P. Scheider Gibson, A. Wernle, S. Tabbal

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171 **Follow–up of psychological features in patients treated with botulinum toxin type A**
M. Coletti Moja, E. Milano, L. Ostacoli, P. Furlan, L. Durelli

172 **Factors associated with drug–induced visual hallucinations in Parkinson's disease**
S. Papapetropoulos, A. A. Argyriou, J. Ellul

173 **No correlation between the clinical severity of autonomic symptoms (SCOPA–AUT) and electrophysiological test abnormalities in advanced Parkinson's disease**
S. Papapetropoulos, A. A. Argyriou, E. Chroni

174 **Cognitive functions in MSA–P and MSA–C patients**
M. Balas, Y. Balash, N. Giladi, T. Gurevich

175 **Olfaction in familial Parkinsonism (FP)**
K. Hentschel, Y. Baba, L. N. Williams, R. L. Doty, R. J. Uitti, Z. K. Wszolek

176 **Non–motor symptoms frequently herald end–of–dose wearing off in Parkinson's disease**
S. H. Isaacson

177 **Prevalence of rapid eye movement behavior disorder. A retrospective study based on the review of the clinical records and polysomnography**
T. Simuni, A. Huang, C. Hammond, P. Zee

178 **Polysomnographic features of REM sleep behavior disorder: differences in REM sleep behavior disorder preceding parkinsonism versus parkinsonism preceding REM sleep behavior disorder**
H.-I. Ma, Y.-J. Kim, M.-K. Chu, K.-H. Yu, J.-Y. Kim, B.-C. Lee

179 **Time course of developing psychosis–like behaviour in the MPTP–marmoset model of Parkinson's disease**
J. Gomez-Ramirez, T. Johnston, N. Visanji, V. Voon, J. M. Brotchie, S. H. Fox

180 **A study of the effect of age on the spatial discrimination threshold– normative data for the study of multiplex adult onset primary torsion dystonia families**
J. P. O'Dwyer, I. Sheikh, F. Molloy, M. Hutchinson

181 **Subthalamic stimulation reduced behavioral disorders induced by basal ganglia dysfunction in monkey**
D. Grabli, C. Karachi, N. Baup, S. Mounayar, E. C. Hirsch, J. Yelnik

182 **Hypersexuality and Parkinson's disease: the role of dopamine replacement**
K. J. Klos, J. H. Bower, K. A. Josephs, J. Y. Matsumoto, J. E. Ahlskog

183 **Abnormal cardiovascular function distinguishes Parkinson's disease (PD) and multiple systems atrophy (MSA) from other atypical Parkinsonian syndromes (APS)**
J. Srinivasan, S. Scala, J. Gutrecht, D. Apetauerova

184 **Olfaction, color vision, and quantitative motor testing abnormalities in patients with idiopathic REM sleep behavior disorder**
R. B. Postuma, A. E. Lang, J. Massicotte, J. Montplaisir

185 **Differences in olfactory dysfunction between sporadic PD and familial Parkinson's disease kindreds by UPSIT subgroup analysis**
K. Hentschel, S. Furtado, K. Markopoulou, R. L. Doty, R. J. Uitti, Z. K. Wszolek

186 **Adjunctive use of licorice to ameliorate orthostatic hypotension in primary autonomic failure: a case report**
H. J. Moore

187 **The motor–scanning system: a requirement to justify the high correlation between motor execution and motor imagery**
M. Sabate, B. Gonzalez, M. C. Sabate, E. Enriquez, J. Abreu, A. Ramirez, M. Rodriguez

Poster Session 2

Monday, March 7

Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:30 p.m. to 2:15 p.m.
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188 **Prophylactic treatment of migraine with botulinum toxin A in Koreans: an open–label study**
M. Y. Park, K. Y. Ahn

189 **Paroxysmal nonkinesigenic dyskinesia responsive to a gluten–free diet**
D. A. Hall, J. Parsons, M. A. Leehey, T. A. Benke

190 **Management of the extrapyramidal syndrome in acquired chronic hepatocerebral degeneration (ACHD)**
S. Papapetropoulos, C. Singer

191 **Pramipexole is effective in the treatment of restless legs syndrome (RLS): results of a 6 week, multi–centre, double blind, and placebo controlled study**
W. Oertel, K. Stiasny-Kolster

192 **Procedimental analyses in movement disorders trials: new approaches in methodological issues**
S. G. Echebarria

193 **24h relief of restless legs syndrome (RLS) symptoms with once daily pramipexole**
B. Hogl, W. Poewe

194 **Early and persistent effect of pramipexole in restless legs syndrome (RLS) patients already with the starting dose**
W. Oertel, K. Stiasny-Kolster

195 **Pramipexole – an effective treatment option in restless legs syndrome (RLS) patients with depressed mood**
K. Stiasny-Kolster, W. Oertel

196 **Pramipexole significantly improves sleep in patients with restless legs syndrome (RLS)**
B. Hogl, W. Poewe

197 **Prospective characterization of movement disorders in the Manhattan HIV brain bank project**
W. Tse, L. Estanislao, D. Polowetzky, S. Verma, S. Morgello

198 **Saccadic eye movements: a useful clinical biomarker in neurodegeneration?**
A. Michell, F. Ali, Z. Xu, D. Fritz, S. Lewis, R. Carpenter

199 **Gait disturbances in depressed patients: effects of pharmacologic therapy**
D. Paleacu, A. Shutzman, N. Giladi, T. Herman, J. M. Hausdorff

200 **Ancient descriptions of movement disorders. Cathedral El Burgo de Osma (Soria, Spain)**
P. J. Garcia Ruiz, J. J. Ruiz, A. Garcia Torres, S. Fanjul

201 **A clinical follow up study of the natural history and daytime somnolence in 94 patients with restless legs syndrome (RLS) attending a specialist clinic in the UK**
S. Tluk, J. Bland, P. Patel, K. R. Chaudhuri

202 **PLM counting in PSG: role of the amplitude**
V. Gschliesser, E. Brandauer, H. Ulmer, B. Hogl, W. Poewe

203 **Characteristics and antidepressant treatment outcomes of patients with psychogenic Movement Disorder: a naturalistic study**
V. Voon, A. E. Lang

204 **Ropinirole is an effective, well–tolerated treatment for moderate–to–severe RLS: results of a US study**
R. Bogan, M. G. Connolly, G. Rederich

205 **Walking is more like catching than tapping: gait in the elderly as a complex cognitive task**
J. M. Hausdorff, G. Yogev, S. Springer, E. S. Simon, N. Giladi

206 **Predictors of health–related quality of life (HRQL) in US respondents suffering from RLS**
L. Gaffney, B. Sherrill, S. Wolowacz, R. P. Allen, M. Connolly

207 **Establishing the relationship between RLS severity and work productivity impairment associated with RLS**
N. Douzinas, M. Connolly, S. Morris

208 **A study of occurrence of restless legs syndrome (RLS) and transferrin receptor assay in an anaemic and non–anaemic multi–ethnic population attending a haematology clinic in inner city, London, UK**
A. De Koker, V. Dhawan, F. Stegie, S. Brendemühl, L. Tillyer, K. R. Chaudhuri

209 **Screening for cognitive deficits in Parkinson's disease – which marker relates to disease severity**
M. W. Riepe, J. Kassubek, F. Tracik, G. Ebersbach

210 **Tolerability of a forced–dose escalating regimen of ropinirole in patients with RLS**
M. Kelly, P. Mistry

211 **Incidence of dyskinesias in a 10–year naturalistic follow–up of patients with early Parkinson's disease (PD) initially receiving ropinirole compared with L–dopa**
O. Rascol, A. D. Korczyn, P. P. De Deyn, A. E. Lang

212 **A segregation analysis of the restless legs syndrome (RLS) using age–of–onset models**
W. A. Hening, R. A. Mathias, R. P. Allen, M. Washburn, S. Lesage, C. J. Earley

213 **Continuous intrathecal morphine application in therapy refractory restless legs syndrome (RLS)**
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367 Association of dementia with parkinsonian signs in the elderly general population in central Spain
M. Pondal, F. Corral, T. Del Ser

368 Elevated titers of anti–thyroid antibodies in patients with multiple system atrophy, unexpected observation
B. Shihman, N. Giladi, T. Gurevich

369 Frequency and nature of dystonia in MSA and PSP
C. H. Schrader, T. Weiskirch, S. D. Suessmuth, B. Herting, The NNIPPS-Study-Group

370 Diagnostic value of ^{123} I–Ioflupane SPECT in psychogenic parkinsonism
C. Gaig, F. Nakamae, M. J. Martí, P. Paredes, F. Valldeoriola, E. Tolosa

371 Motor fluctuations in juvenile parkinsonism – management dilemma
A. B. Shah, P. M. Wadia, R. B. Baviskar, R. Ramdass

372 Can the clonidine–growth hormone (GH) stimulation test (CGHST) distinguish multiple system atrophy and Parkinson's disease early in the course of illness?
N. Sarangmath, M. Ragothaman, S. Koshy, D. K. Subbakrishna, C. J. Mathias, U. B. Muthane

373 Movement Disorders associated with fronto–temporal dementia with parkinsonism linked to chromosome 17 (FTDP–17)
M. M. Wickremaratchi, H. R. Morris

374 Multiple system atrophy genitourinary type: an emerging entity?
P. V. Swaminath, A. Mohan, M. Ragothaman, N. Sarangmath, C. J. Mathias, U. B. Muthane

375 The pathological basis of disproportionate antecollis in multiple system atrophy
T. Ozawa, D. Paviour, N. P. Quinn, T. Revesz, J. L. Holton, A. J. Lees

376 Delayed onset of freezing of gait following the bilateral necrosis of the globus pallidus
T.-B. Ahn, J.-W. Cho, S. S. Yoon, S. E. Kim

377 Deep brain stimulation of subthalamic nucleus can improve parkinsonism symptoms in sinemet non–responsive patients
O. V. Kopyov, R. F. Young, R. M. Hutchman, D. B. Jacques

378 Hemiparkinsonism and facial dyskinesia in a patient with a contralateral mesencephalic cyst
S. Zats, S. L. Lewis, C. L. Comella

379 Parkinsonism induced by bupropion
F. Grandas, L. Lopez-Manzanares

380 Gait and balance assessment in parkinsonian disorders
S. N. Azher, K. D. Vuong, J. Shahed, A. L. Diamond, J. Jankovic

381 Gait assessment in parkinsonism, dementia and normal ageing
A. Bernal, G. Arango, A. Granados, W. Fernandez

382 Extrapyramidal symptoms in a group of Italian dental laboratory technicians
E. Fabrizio, N. Vanacore, G. Di Brigida, G. Meco

383 Valvular heart disease in Parkinson's disease versus controls – an echocardiographic study–
C. M. Peralta, E. Wolf, K. Seppi, H. Alber, S. Mueller, W. Poewe

Poster Session 3

Tuesday, March 8

Poster Viewing: 8:30 a.m. to 5:00 p.m.
Authors Present: 12:45 p.m. to 2:30 p.m.
Abstracts: 384-592

Parkinson's disease 2

Poster numbers 384-500

384 **Sporadic Parkinson's disease: correlations between Hoehn and Yahr stages, cognitive status, and the neuropathological stages of a newly proposed staging protocol**
U. Rueb, K. Del Tredici, E. Jansen Steur, R. de Vos, H. Braak

385 **Entacapone provides additional benefit to Parkinson's disease patients with motor fluctuations treated with levodopa and selegiline**
J. Larsen, H. Nissinen, M. Vahteristo

386 **Repetitive transcranial magnetic stimulation in advanced Parkinson's disease: effects on cortical excitability, freezing of gait and executive functioning**
I. Rektorova, S. Sedlackova, S. Telecka, A. Hlubocky, I. Rektor

387 **Freezing of gait has significant effect on quality of life in Parkinson's disease**
O. Moore, C. Peretz, N. Giladi

388 **Effect of 4–week training with audio–visual cues on sit–to–stand in Parkinsonian patients**
M. K. Mak, C. W. Hui-Chan

389 **Gender representation among those donating brains for Parkinson's disease research**
A. Rajput, M. L. Rajput

390 **Extrapyramidal gait slowing predicts incident dementia in the Sydney older persons study: where is the brain lesion in motor slowing?**
G. A. Broe, S. R. Duma, G. M. Halliday, L. M. Waite

391 **Visuospatial impairment or executive dysfunction do not contribute to falling in Parkinsons disease**
B. D. Riggeal, G. P. Crucian, C. E. Jacobson IV, S. K. Munson, M. S. Okun, H. H. Fernandez

392 **Turning during walking in Parkinson's disease: footstep patterns**
F. E. Huxham, R. Iansek, M. E. Morris, R. Baker

393 **Cognitive decline parallels motor progression and not disease duration in Parkinson patients**
B. D. Riggeal, G. P. Crucian, C. E. Jacobson IV, S. K. Munson, M. S. Okun, H. H. Fernandez

394 **An abbreviated wearing–off patient questionnaire (WOPQ): sensitivity analysis**
M. Stacy

395 **Smoking among patients diagnosed with Parkinson's disease: is it neuroprotective?**
S. Papapetropoulos, J. M. Villar, C. Singer, J. Gonzalez, D. C. Mash

396 **Orthostatic hypotension in Parkinson's disease and multiple system atrophy**
K. Sergey, L. Igor, O. Miroslav

397 **Heat shock protects against alpha synuclein–triggered apoptosis in a yeast model of Parkinson's disease**
T. R. Flower, C. A. Froelich, S. N. Witt

398 **Family aggregation and geographical clustering of *LRRK2* associated Parkinson's disease in central Norway**
J. O. Aasly, M. Toft, I. Fernandez-Mata, L. White, M. Hulihan, M. Farrer

399 **A new self–assessment patient card for early detection and management of Parkinson's disease fluctuations: the PRECOCE survey**
J. P. Azulay, F. Durif, R. Rogez, C. Tranchant, I. Bourdeix, K. Rerat

400 **Evaluation of liver–related adverse events with tolcapone: a review of 7–years of worldwide safety data**
R. Watts, G. Kricorian

401 **Plasma homocysteine levels in L–dopa treated PD patients with cognitive dysfuntions: a causal link?**
S. Zoccolella, C. Diroma, A. Fraddosio, R. Mastronardi, I. Russo, S. Lamberti

402 **Long term follow up of fluctuating Parkinsonians on apomorphine continuous subcutaneous infusion by PUMP**
J.-E. G.M. Vanderheyden

403 **Mental loading increases gait asymmetry and stride–to–tride variability in patients with Parkinson's disease**
G. Yogev, N. Giladi, S. Springer, J. M. Hausdorff, C. Peretz, M. Plotnik

404 **Results from a 2–year centralized tolcapone liver enzyme monitoring program**
M. F. Lew, G. J. Kricorian

405 **Entacapone decreases axial symptoms and tremor in levodopa–treated Parkinson's disease patients experiencing wearing–off symptoms**
D. J. Brooks, M. Kuoppamäki, M. Vahteristo

406 **High frequency activity in the subthalamic nucleus and substantia nigra reticulata in Parkinson's disease**
P. Novak, J. Nazzaro, S. Daniluk, M. Diggin, S. L. Ellias

407 **Validation of the freezing of gait questionnaire (FOG–Q) for patients with Parkinson's disease**
N. Giladi, Y. Tal, R. Meiron, O. Rascol, D. Brooks, E. Melamed

408 **Elevated homocysteine level in patiens treated with levodopa**
M. Yilsen, T. Aydemir, H. Meral, F. Ozer, L. Hanoglu, S. Cetin

409 **Impact of pramipexole on mood and initiative in early Parkinson's disease**
K. Kieburtz, M. Romer, M. McDermott, C. Kamp, Parkinson Study Group

410 **New levodopa/carbidopa/entacapone tablets (Stalevo®) provide better quality of life for Parkinson's disease patients and save costs to the society**
L. Findley, H. Turunen, M. Apajasalo, A. Lees

411 **Stride variability in Parkinson's disease**
E. Ivashina, P. Novak

412 **Spectral analysis of the subthalamic nucleus responses to passive movement in Parkinson's disease**
P. Novak, S. E. Ellias, S. Daniluk, J. Nazzaro

413 **Expression pattern of synphilin–1 and parkin in control and Parkinson's disease brain**
R. Bandopadhyay, A. E. Kingsbury, A. J. Lees

414 **Drug holiday revisited – amantadine infusions for motor complications in levodopa treated Parkinson's disease**
A. Friedman, D. Koziorowski

415 **The prevalence of movement disorders in the general community**
G. K. Wenning, K. Seppi, J. Mueller, M. Koellensperger, S. Kiechl, J. Willeit

416 **Beneficial interactions between grapefruit juice and dopamine agonists in patients with Parkinson's disease**
M. Nagai, A. Nakatsuka, H. Yabe, H. Moritoyo, T. Moritoyo, M. Nomoto

Poster Session 3

417 Bilateral subthalamic nucleus deep brain stimulation in Parkinson's disease and mood disorders
I. Chereau-Boudet, P.-M. Llorca, J.-J. Lemaire, F. Durif

418 Memantine in Parkinson's disease dementia (PDD)–clinical experience
C. G. Fox, G. Umoh, M. Samuel, B. Barbara, M. Neil

419 Associative plasticity of motor cortex in Parkinson's disease
S. Bagnato, R. Agostino, N. Modugno, A. Quartarone, A. Berardelli

420 Behavioural and psychiatric outcome in patients with Parkinson's disease and subthalamic stimulation
P. Piombo, E. Wolf, T. Benke, K. Kramer-Reinstadler, G. K. Wenning, W. Poewe

421 Axial myopathy in parkinsonian disorders
M. Sawires, K. Seppi, J. Wanschitz, W. N. Löscher, G. K. Wenning, W. Poewe

422 Parkinson's disease, apathy, and attentional process: an event–related potentials study
S. Mathis, J.-L. Houeto, S. Karolewics, M.-N. Besson, V. Bonneau, R. Gil

423 Does cardiovascular dysautonomia occur early in Parkinson's disease and increase with its severity?
M. Ragothaman, N. Sarangmath, S. Koshy, P. V. Swaminath, C. J. Mathias, U. Muthane

424 Selegiline reduced tremor and bradykinesia in levodopa–treated Parkinson's disease patients in a long–term randomized double–blind study
S. E. Pålhagen and the Swedish Parkinson Study Group

425 Prevalence of Parkinson's disease in the Parsee (Zoroastrian) community
M. H. Bhatt, N. A. Chhaya, S. R. Vaidya, N. Quinn, K. P. Bhatia

426 Reaching kinematics not matched to postural context by PD patients
J. Doan, I. Q. Whishaw, S. M. Pellis, O. Suchowersky, L. A. Brown

427 Using wearable technology to monitor motor fluctuations in Parkinson's disease
D. M. Sherrill, R. Hughes, S. S. Salles, M. Akay, D. G. Standaert, P. Bonato

428 The influence of fixation offset on predictive and reflexive saccades in Parkinson's disease
R. A. Skinner, M. R. MacAskill, R. D. Jones, T. J. Anderson

429 Rater – blinded, prospective study comparing the therapeutic benefit of clozapine vs. quetiapine on psychosis in patients with Parkinson's disease
D. Merims, H. Shabtai, M. Balas, C. Peretz, N. Giladi

430 Small interfering RNA (SiRNA) targeting the PINK1 induces apoptosisin human SH–SY5Y cells
H. Deng, J. Jankovic, T. Pan, W. Xie, W. Le

431 The efficacy of quantitative gait analysis by GAITRite system in evaluation the parkinsonian bradykinesia
S.-L. Chien, C.-C. Liang, Y.-H. Pan, Y.-C. Chou, S.-Z. Lin, S.-Y. Chen

432 An integrated rehabilitation approach to Parkinson's disease: learning big & loud
C. M. Fox, B. F. Farley, L. O. Ramig, D. H. McFarland

433 To compare clinical improvement in young and old Parkinson's disease after STN DBS
P. K. Doshi, N. A. Chhaya, M. H. Bhatt

434 Bowel movement frequency in mid–life and substantia nigra neuron counts
H. Petrovitch, G.W. Ross, R. D. Abbott, J. Nelson, D. G. Davis, L. R. White

435 Treatment patterns for Parkinson's disease in the Pacific Northwest Veterans Health Network
M. A. Brodsky, R. Bourdage, K. Swarztrauber

436 Parkinson's patients with deep brain stimulation: effects of education and intelligence on depressive symptoms
K. L. Cox, C. R. Schadt, M. Tramontana, D. W. Byrne, T. L. Davis, P. D. Charles

437 Efficacy and tolerability of electroconvulsive therapy in advanced Parkinson's disease with symptoms not responsive to levodopa. A pilot study
F. Valldeoriola, L. Pintor, L. Rami, M. J. Martí, E. Tolosa, M. Bernardo

438 Effects of physical therapy on gait and balance in Parkinson's disease
A. A. Zylstra, A. F. Griffith, A. D. Mosley, B. C. Leis

439 Association of olfactory dysfunction with risk of future Parkinson's disease
W. Ross, H. Petrovitch, R. D. Abbott, C. M. Tanner, J. Popper, K. Masaki

440 Psychogenic Parkinsonism: combination of clinical, electro-physiological and [^{123}I] Ioflupane SPECT Scan explorations improves diagnostic accuracy
S. Benaderette, P. Zanotti Fragonara, E. Apartis, C. Nguyen, J. M. Trocello, M. Vidailhet

441 Paraspinal muscle asymmetry in Parkinson's disease
W. G. Ondo, H. Haykal

442 Physical activity performance and quality of life in individuals with early stage Parkinson's disease and their partners
C. C. Bassile, C. L. Curtis, H. E. David

443 Melatonin treatment for sleep–related symptoms in patients with Parkinson's disease
G. A. Dowling, J. Mastick, M. J. Aminoff, E. Colling, J. H. Carter, C. M. Singer

444 Retinal pigment epithelium (RPE) cells provide neurotrophic effect on striatal neurons
E. Erbe, B. McKay, P. S. Patel, J. Kennedy, S. L. Xiang, S. J. Sherman

445 Retinal pigment epithelium (RPE) cells provide neurotrophic effect on midbrain dopaminergic neurons
P. S. Patel, B. Goodman, B. McKay, E. Erbe, J. Kennedy, S. J. Sherman

446 Co–expression of dopamine–1 and dopamine–2 receptor subtypes in striatal neurons
J. Kennedy, P. S. Patel, E. Erbe, T. Falk, S. J. Sherman

447 A functional reach task and cognitive impairment are associated with falls in Parkinson's disease
K. M. Biglan, J. R. Williams, S. Grill, T. Augustin, S. Bassett, L. Marsh

448 D1 receptor hypersensitivity in Parkinson's disease is related to persistent increase of the olfactory type G–protein alpha–subunit in the striatum
J.-C. Corvol, M.-P. Muriel, E. Valjent, J.-A. Girault, E. Hirsch, D. Herve

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449 Characterization of behavioral and neurodegenerative changes induced by convection–enhanced delivery of 6–hydroxydopamine into the rat striatum
A. G. DiLella, E. V. Lis, K. W. Jones, T. T. Cao, F. J. Hess, G. R. Seabrook

450 Changes in vowel acoustics following intensive voice treatment in Parkinson's disease: implications for speech intelligibility
J. Spielman, L. Ramig, C. Fox

451 Rasagiline does not promote tyramine pressor responses
J. A. de Marcaida, Parkinson Study Group

452 Correlation between punding and dyskinesias in Parkinson's disease (PD)
L. Silveira-Moriyama, A. H. Evans, R. Katzenschlager, A. J. Lees

453 5–yr follow–up REAL–PET study in patients with early Parkinson's disease (PD) initially receiving ropinirole or L–dopa
R. L. Watts, A. Lang, O. Rascol, W. Poewe, A. J. Stoessl, R. Hauser

454 Technology supported speech treatment for Parkinson's disease
A. Halpern, C. Matos, L. Ramig, J. Petska, J. Spielman, L. Will

455 Motor cortical excitability appears to differ between the tremor and the akinetic form of Parkinson's disease
L. Barbin, P. Sauleau, Y. Pereon, C. Magne, N. Alexandri, P. Damier

456 Increased switch costs in Parkinson's disease reflect deficient endogenous preparation in a set shifting paradigm
C. Daniels, K. Witt, J. Kernbichler, V. Daniel, S. Rehm, G. Deuschl

457 Chronic pain in Parkinson's disease: preliminary results of the DOPAMIP study, a cross–sectional survey in south–west of France
L. Negre-Pages, H. Grandjean, O. Rascol, DOPAMIP Study Group

458 Patterns of damage within magno, parvo and koniocellular pathways in Parkinson's disease
A. Freire, C. Januario, F. Silva, F. Regateiro, P. Faria, M. Castelo-Branco

459 Dynamic properties of saccadic intrusions in Parkinson's disease: a preliminary study
A. Becker, A. Kube, U. Hinterseher, A. Metz, C. J. Möller, W. H. Oertel

460 Survival among veterans with Parkinson's disease
D. L. White, E. C. Lai, S. Moore

461 Does the type of physician seen impact medications prescribed?
S. Oswald, M. A. Coles, H. Shill

462 Abnormal motor cortex plasticity in Parkinson's disease: evidence in rodents and humans
F. Battaglia, A. Quartarone, S. Bagnato, V. Rizzo, L. Morgante, P. Girlanda

463 Acoustic changes following behavioral speech treatment to train increased loudness in people with idiopathic Parkinson's disease
L. A. Will, J. Spielman, L. O. Ramig

464 Excessive daytime sleepiness and on–road driving performance in individuals with Parkinson's disease
M. Amick, A. D'Abreau, M. Moro-de-Casillas, K. Chou, B. Ott

465 Surgical microtrauma as a predictor of neuropsychological change following unilateral pallidal and subthalamic DBS
J. C. Rothlind, R. W. Cockshott, J. A. Walker, P. A. Starr, W. J. Marks, Jr., P. S. Larson

466 Learning big™: efficacy of a large–amplitude exercise approach for patients with Parkinson's disease – bradykinesia to balance
B. G. Farley, G. F. Koshland

467 Entacapone provides benefit to Parkinson's disease patients with motor fluctuations treated with levodopa and dopamine agonists: an analysis of four randomized trials
M. Kuoppamäki, M. Vahteristo, K. Kieburtz

468 Rasagiline is effective and well tolerated in the treatment of Parkinson's disease (PD) patients with levodopa–related motor fluctuations receiving other adjunctive therapy
L. Elmer

469 Improving speech intelligibility in patients with Parkinson's disease and dysarthria using SpeechEasy®, a speech fluency–enhancing device
E. Q. Wang, L. Verhagen Metman, M. H. de Vries

470 Depression subtypes are associated with motor subtypes of Parkinson's disease
P. Kavanagh, S. J. Ferrando, J. Godbold, M. Veytsman, A. Di Rocco

471 Pizza, mint and licorice: smell testing in Parkinson's disease in a UK population
L. Silveira-Moriyama, D. Williams, R. Katzenschlager, A. J. Lees

472 Parkinson's disease and Parkinsonism among US Veterans: demographic and geographic distribution in fY2002
E. C. Lai, S. Moore

473 Comparison of psychosis and sleep in hallucinating subjects with dementia with Lewy bodies (DLB) and Parkinson's disease with dementia (PDD) and without dementia (PDND)
J. G. Goldman, V. K. Hinson, C. G. Goetz, S. E. Leurgans

474 Density of small fibers in Parkinson's disease and multiple system atrophy
P. Novak, Whren, J. Bhawan

475 Homozygous 1311G>A *PINK1* mutation in a patient with Parkinson disease, psychiatric disorder, and apparently dominant transmission
C. Criscuolo, G. Volpe, A. De Rosa, A. Filla, E. M. Valente, G. De Michele

476 Rescue dose use: enhanced desire for drug or avoiding aversive'offs'
D. Ottaviani, A. Evans, R. Katzenschlager, A. Lees

477 Levodopa prevents falls in the absence of visual input in Parkinson's disease: case report
F. J. Revilla, G. Shahzad, M. Gartner, C. Cox, A. Bhattacharya

478 Effect of deep brain stimulation on posture control in Parkinson's disease
S. Mulligan, N. Krishnamurthi, P. Mahant, J. Samanta, J. Abbas

479 Neuropathological study 16 years after autologous adrenal medullary transplantation in a Parkinson's disease patient
K. Kompoliti, Y. Chu, K. M. Shannon, C. G. Goetz, J. H. Kordower

480 Electronic diaries to assess motor fluctuations and dyskinesias in Parkinson's disease patients
L. Correia Guedes, B. Marino, J. Ferreira, M. M. Rosa, A. Noronha, C. Sampaio

481 The frequency and causes of falls in moderately affected Parkinson's disease patients
A. Szczudlik, M. Rudzinska, S. Bukowczan, M. Marona, J. Furgai, M. Michalski

482 Regular dopaminergic therapy cross–sensitises to the effects of methylphenidate
A. H. Evans, A. D. Lawrence, A. J. Lees

483 Tolerability of subcutaneous apomorphine in Parkinson's disease patients not taking trimethobenzamide as prophylaxis against gastrointestinal adverse events
R. B. Dewey

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484 Depression and Parkinson's disease
E. Fabrizio, N. Vanacore, R. Martani, M. Colasanti, G. Brigida, G. Meco

485 Orolingual masticatory tardive dyskinesia induced by levodopa in a parkinsonian patient
C. C. Meyniel, P. P. Damier

486 Reduced NURR1 gene expression affects expression of dopaminergic phenotypic markers and Parkinson's disease–related genes
H. Deng, J. Jankovic, W. Xie, T. Pan, W. Le

487 Relationship among psychiatric, motor and cognitive functioning in non–demented individuals with Parkinson's disease
M. F. Dulay, H. S. Levin, J. Jankovic, E. C. Lai, M. K. York

488 Subcutaneous apomorphine in patients with advanced Parkinson's disease: ECG results from aPO302
J. H. Sherry, P. B. Bottini, G. A. Burkhart

489 Electrocardiographic assessment of subcutaneous apomorphine in patients with advanced Parkinson's disease
J. H. Sherry, P. B. Bottini, G. A. Burkhart

490 Repetitive transcranial magnetic stimulation (rTMS) and dyskinesias in Parkinson's disease – clinical and physiological effects of four daily sessions of slow (1Hz) rTMS
S. Filipovic, P. Heywood, J. Rothwell, K. Bhatia

491 Parkinson's disease and apolipoprotein E genotype: possible association?
A. S. Morgadinho, J. M. Bras, R. Guerreiro, M. H. Garrucho, C. Januario, C. Oliveira, L. Cunha

492 Can patients with PD assess their own disease severity? A self–assessment study of the Hoehn & Yahr Scale
R. Katzenschlager, N. Russo, M. Jahanshani, N. Quinn, A. L. Lees, A. Schrag

493 Treatment of memory and psychosis in patients with Parkinson's disease
S. H. Isaacson, D. L. Kreitzman

494 Implications of motor fluctuations in Parkinson s disease patients on chronic therapy (IMPACT): results from an observational registry
J. Anthony, J. Spears, B. Van Lunen

495 InSPECT: an investigation of the effects of short–term treatment with pramipexole or levodopa on [123I] b–CIT and SPECT imaging in early Parkinson disease
D. Jennings, R. Tabamo, J. P. Seibyl, K. Marek, The INSPECT Study Group

496 Olfactory identification testing in combination with dopamine transporter (DAT) imaging as an early marker for Parkinsonian syndrome
D. Jennings, R. Tabamo, J. P. Seibyl, K. Marek

497 Implications of motor fluctuations in Parkinson's disease patients on chronic therapy (IMPACT): results from an observational registry
Anthony, Spears, Van Lunen

498 Olfactory identification testing in combination with dopamine transporter (DAT) imaging as an early marker for Parkinsonian syndrome
Jennings, Tabamo, Seibyl, Marek

499 InSPECT: an Investigation of the effects of short-term treatment with pramipexole or levodopa on [123I] B-CIT and SPECT imaging in early Parkinson disease
Jennings, Tabamo, Seibyl, Marek, the INSPECT Study Group

500 The Journey of Parkinson disease patients, the quality of life and the role of the Clinical nurse specialist
Lachance

Spasticity

Poster numbers 501-504

501 Functional stereotactic operations for treatment of residual symptomes after stroke
A. Koulousakis, D. Lenartz

502 Correction of low spastic paraparesis by spinal cord stimulation
V. A. Shabalov, A. V. Dekopov, E. M. Troshina, A. A. Tomsky

503 Sonography in the '12Plus– concept' with botulinum toxin A (BOTOX®) in children with cerebral palsy – accuracy, safety and secondary non–response
S. Berweck, A. S. Schroeder, S.-H. Lee, I. Gutmann, A. Schwerin, F. Heinen

504 The impact of intensive voice treatment (LSVT) on speech intelligibility in children with spastic cerebral palsy
C. M. Fox, C. Boliek, L. O. Ramig

Surgical Therapy

Poster numbers 505-556

505 The use of two DBS leads for the treatment of post–traumatic and Multiple Sclerosis tremor
K. D. Foote, P. Seignourel, H. H. Fernandez, C. Jacobson, R. L. Rodriguez, M. S. Okun

506 Management of referred DBS failures
M. S. Okun, M. Tagliati, M. Pourfar, R. L. Rodriguez, H. H. Fernandez, K. D. Foote

507 Acute blinded stimulation of the anterior limb of the internal capsule and nucleus accumbens region: side effects and benefits
W. Knight, N. A. Shapira, K. D. Foote, U. Springer, W. Goodman, M. S. Okun

508 Are mild MRI abnormalities predictive of outcome after bilateral subthalamic stimulation for Parkinson's disease?
K. E. Lyons, S. B. Wilkinson, R. Pahwa

509 Functional outcome after subthalamic nucleus deep brain stimulation (STN–DBS) to treat Parkinson's disease (PD): 6 months follow–up
K. Robinson, H. Cianci, J. Norigian, P. Moberg, J. Jaggi, G. Baltuck

510 Accuracy of frameless stereotactic placement of deep brain stimulators: comparison to the Leksell frame
K. L. Holloway, J. M. Henderson, S. E. Gaede, J. M. Rosenow, MD

511 Unilateral deep brain stimulation in the subthalamic nucleus for advanced Parkinson's disease
N. R. Whaley, R. E. Wharen, J. D. Putzke, J. A. Wilden, R. J. Uitti

512 Behavioral and cognitive decline after subthalamic stimulation in a patient with Alzheimer's and Lewy body dementia mimicking Parkinson's disease
K. L. Chou, J. Grace, S. Delamonte, G. Friehs, H. H. Fernandez, J. H. Friedman

513 Worsening of Parkinsonism by stimulation outside the subthalamic nucleus
J. Gere, P. Krack, V. Fraix, J.-F. Lebas, A. L. Benabid, P. Pollak

514 Uneven benefit observed from parkinsonian patients who underwent subthalamic stimulaiton
Y. C. Chou, S. Y. Chen, S. H. Lin, C. W. Lee, P. Y.-H. Pan, S. Z. Lin

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515 Unilateral vs. bilateral subthalamic nucleus deep brain stimulation in Parkinson disease: preliminary results from staged bilateral stimulator implantations
A. Samii, V. E. Kelly, J. C. Slimp, A. Shumway-Cook, R. Goodkin

516 Deep brain stimulation in multiple system atrophy: a clinico-pathological report
V. Talmant, P. Esposito, B. Stilhart, M. Mohr, C. Tranchant

517 Effect of deep brain stimulation in a human with MPTP–induced parkinsonism
C. W. Christine, W. J. Langston, R. S Turner, P. A. Starr

518 Meta–analysis of outcome of deep brain stimulation for dystonia
K. L. Holloway, M. S. Baron, R. E. Brown

519 Long–term evaluation of quality of life after DBS for advanced Parkinson's disease
J. Volkmann, for the Deep-Brain Stimulation for Parkinson's Disease Study Group

520 Globus pallidus deep brain stimulation in dystonia
A. L. Diamond, J. Shahed, S. Azher, K. Dat Vuong, J. Jankovic

521 Pyramidal tract side effects induced by deep brain stimulation of the subthalamic nucleus
G. Tommasi, P. Krack, V. Fraix, A. L. Benabid, J. F. Le Bas, P. Pollak

522 Subthalamic nucleus stimulation and apraxia of eyelid opening in patients with Parkinson's disease
G. Tommasi, P. Krack, V. Fraix, A. Batir, A. L. Benabid, P. Pollak

523 Deep brain stimulation in Parkinson's disease following fetal nigral transplantation
J. Herzog, O. Pogarell, W. H. Oertel, H. M. Mehdorn, G. Deuschl, J. Volkmann

524 Severe aggravation of tremor after thalamic DBS in tremor–predominant PD
D. Apetauerova, J. Joseph, T. Norregaard, D. Tarsy

525 Long–term follow–up after subthalamic DBS for Parkinson's disease
A. Landi, A. Antonini, C. Ceresa, M. Parolin, F. Vergani, C. Mariani

526 Clinical–functional correlates of verbal fluency decrease in PD patients who underwent DBS stimulation: an eCD–SPET study
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The *Movement* Disorder Society

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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

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- Non-Members may apply for MDS membership - the registration fee includes MDS membership at a reduced rate ($50 savings) with limited benefits through 2005, and full membership status in 2006. New MDS Member applicants will be contacted by the MDS International Secretariat to provide additional information.

Membership Benefits as of 2005

- A subscription to the print, DVD, and online journal, *Movement* Disorders, including supplemental publications, such as *Management of Parkinson's Disease: An Evidence-Based Review and Pediatric Movement Disorders* CD-ROM.
- A unique selection of educational opportunities, including online Continuing Medical Education (CME) activities and reference materials on topics in Movement Disorders such as *The Movement Disorder Society's Guide to Botulinum Toxin Injections*.
- A reduction in fees charged for participation in the Society's educational programs. Among these are the annual International Congress of Parkinson's Disease and Movement Disorders, and various clinical and scientific programs, courses and workshops held separately from the Congress.
- A Members Only section of the MDS website at www.movementdisorders.org, including a searchable membership directory.
- A print directory listing addresses, telephone and fax numbers, and e-mail addresses for all members.
- A quarterly newsletter entitled, *Moving Along*.
- Participation in the election of Officers and International Executive Committee members.

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Future International Congresses of Parkinson's Disease and Movement Disorders

Kyoto, Japan
October 29 to November 2, 2006

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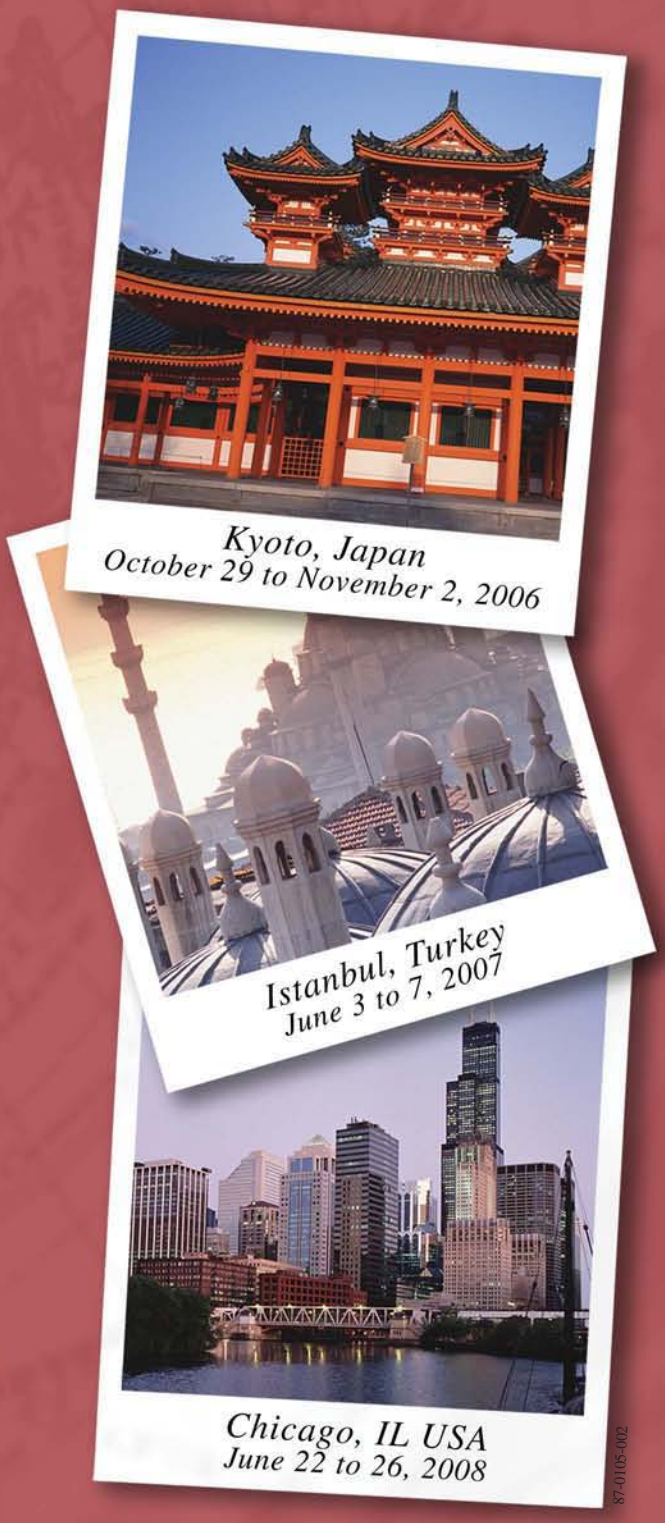
June 3 to 7, 2007

Chicago, IL USA
June 22 to 26, 2008



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