



The *Movement* Disorder Society's

12th International Congress of Parkinson's Disease and Movement Disorders

CHICAGO

Chicago, IL, USA | June 22-26, 2008

FINAL PROGRAM

The *Movement* Disorder Society's

12th International Congress of Parkinson's Disease and Movement Disorders

WELCOME LETTER

Dear Colleagues,

On behalf of The *Movement* Disorder Society (MDS), we are pleased to welcome you back to North America for the 12th International Congress of Parkinson's Disease and Movement Disorders in Chicago, IL, USA.

This program has come together through the coordination and work of the Congress Scientific Program Committee. We encourage you to take every opportunity to participate in the Scientific Program which has drawn world renowned speakers and experts in their respective fields. The 2008 Scientific Program will incorporate educational courses, Opening Symposia, Plenary and Parallel Sessions, Video and Meet the Expert sessions, poster presentations and Guided Poster Tours. We would like to express our gratitude to the large number of our volunteer committees for designing this innovative International Congress.

We are excited to offer several new events this year including Guided Poster Tours, Corporate Therapeutic Sessions and the Video Olympics. Just as the city of Chicago is an exciting place to be with many things to do and see, so is our International Congress. We hope that you will be able to allot time in your schedule to participate in our detailed program, visit the exhibit hall and poster areas, view the History Exhibit and attend the social events in the evening.

Thank you for your support of The *Movement* Disorder Society and welcome to our 12th International Congress of Parkinson's Disease and Movement Disorders.

With best regards,



A handwritten signature in black ink that reads "A. E. Lang".

Anthony E. Lang
President, The Movement Disorder Society, 2007-2009



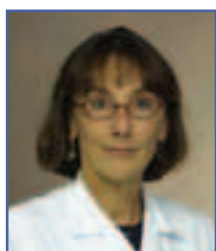
A handwritten signature in black ink that reads "S. Przedborski".

Serge Przedborski
Chair, Congress Scientific Program Committee, 2007-2008



A handwritten signature in black ink that reads "Christopher Goetz".

Christopher Goetz
Co-Chair, Congress Scientific Program Committee, 2008



A handwritten signature in black ink that reads "Cynthia Comella".

Cynthia Comella
Chair, Congress Local Organizing Committee, 2008



A handwritten signature in black ink that reads "Kathleen Shannon MS".

Kathleen Shannon
Co-Chair, Congress Local Organizing Committee, 2008

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Important Safety Information

Zelapar is contraindicated in patients with a known hypersensitivity to any formulation of selegiline or any of the inactive ingredients of **Zelapar**. **Zelapar** is also contraindicated for use with meperidine and should not be administered with the analgesic agents tramadol, methadone, and propoxyphene. **Zelapar** should not be used with the antitussive agent dextromethorphan and should not be administered along with other selegiline products. Daily doses of **Zelapar** should not exceed 2.5 mg/day because of the risks associated with nonselective inhibition of MAO. In general, the combination of **Zelapar** and tricyclic antidepressants, as well as **Zelapar** and serotonin reuptake inhibitors, should be avoided. In clinical trials, the incidence of adverse orthostatic hypotension was higher in geriatric patients than in nongeriatric patients. **Zelapar** may potentiate the dopaminergic side effects of levodopa and may cause or worsen preexisting dyskinesia. Decreasing the dose of levodopa may improve this side effect. **Zelapar** should be used during pregnancy only if the potential benefit to the mother justifies the potential risk to the fetus.

The most commonly observed adverse events reported during clinical trials were dizziness, nausea, pain, headache, insomnia, rhinitis, dyskinesia, back pain, skin disorders, stomatitis, and dyspepsia. In addition, 5.2% of patients discontinued **Zelapar** therapy due to adverse events (versus 1% with placebo).

References: 1. Zelapar [package insert]. Costa Mesa, CA: Valeant Pharmaceuticals International; 2006. 2. Tetrad JW, Koller WC. A novel formulation of selegiline for the treatment of Parkinson's disease. *Neurology*. 2004;63(7)(suppl 2):S2-S6. 3. Waters CH, Sethi KD, Hauser RA, Molho E, Bertoni JM, and the Zydys Selegiline Study Group. Zydys selegiline reduces off time in Parkinson's disease patients with motor fluctuations: a 3-month, randomized, placebo-controlled study. *Mov Disord*. 2004;19:426-432.

*Versus 0.6 hours less OFF time with L-dopa + placebo after 12 weeks ($P < 0.001$).

†Versus 0.4 hours more dyskinesia-free ON time with L-dopa + placebo after 12 weeks ($P = 0.006$). The proportion of ON time that was dyskinesia-free may be similar between Zelapar and placebo.

Please visit www.zelapar.com for Full Prescribing Information.



ACKNOWLEDGEMENTS

The International Congress Oversight Committee of the 12th International Congress of Parkinson's Disease and Movement Disorders wishes to acknowledge and thank the following companies for their support:

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FOR THE INITIAL AND LONG-TERM TREATMENT OF PARKINSON'S DISEASE (PD)

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(01/07)

BP/MSU

MIRAPEX delivers significant efficacy as a foundation therapy for early PD and beyond

In early PD...

- **Significant improvements in daily activities seen in as early as 3 weeks.¹**
— In a multicenter, randomized, double-blind, placebo-controlled, 35-week trial in 335 (333 analyzed) patients with early PD.
- **Helps you reserve the use of levodopa until patients need it most.²**
— As measured by a pooled survival analysis from long-term, open-label extension trials of 3 double-blind clinical trials in a total of 707 patients with early PD.

In advancing PD...

- **A nearly 5-fold improvement in resting tremor when used with levodopa.³**
— In a multicenter, placebo-controlled, 31-week trial in 354 patients with advanced PD experiencing motor fluctuations.
- **Significantly improves activities of daily living and motor symptoms scores when added to levodopa.⁴**
— In a multicenter, placebo-controlled, 32-week trial in 380 (351 analyzed) patients with advanced PD experiencing motor fluctuations.

IMPORTANT INFORMATION ABOUT MIRAPEX:

- 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, constipation, and nausea.

Patients and caregivers should be informed that impulse control disorders/compulsive behaviors may occur while taking medicines, including pramipexole, for treat Parkinson's disease.

References: 1. Steiner HK, Borodi Z, Friedman JH, et al. Pramipexole in early-stage PD: a randomized, double-blind, placebo-controlled trial to evaluate Parkinson's disease. *Neurology*. 2010;74(21):225. 2. Borodi Z, Steiner H. Compulsive without levodopa as early treatment for Parkinson's disease: a long-term follow-up of pramipexole. *Parkinsonism Relat Disord*. 2012;17(10):1000-1003. 3. Friedman JH, et al. Pramipexole in early-stage PD: a randomized, double-blind, placebo-controlled trial to evaluate Parkinson's disease. *Neurology*. 2010;74(21):225. 4. Friedman JH, et al. Pramipexole in early-stage PD: a randomized, double-blind, placebo-controlled trial to evaluate Parkinson's disease. *Neurology*. 2010;74(21):225.

Mirapex® (pramipexole dihydrochloride)
0.125 mg, 0.25 mg, 0.5 mg, 1 mg, and 1.5 mg tablets

Brief Summary of Prescribing Information

INDICATIONS AND USAGE

Parkinson's Disease: MIRAPEX tablets are indicated for the treatment of the signs and symptoms of idiopathic Parkinson's disease.

Restless Legs Syndrome: MIRAPEX tablets are indicated for the treatment of moderate-to-severe primary Restless Legs Syndrome (RLS).

CONTRAINDICATIONS: MIRAPEX tablets are contraindicated in patients who have 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 tablets, 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 had been reported as late as one year after the initiation of treatment.

Somnolence is a common occurrence in patients receiving MIRAPEX tablets at doses above 1.5 mg/day (0.5 mg TID) for Parkinson's disease. In controlled clinical trials in RLS, patients treated with MIRAPEX tablets at doses of 0.25-0.75 mg once a day, the incidence of somnolence was 6% compared to an incidence of 3% for placebo-treated patients (see ADVERSE EVENTS). 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 MIRAPEX tablets, patients should be advised of the potential to develop drowsiness and specifically asked about factors that may increase the risk with MIRAPEX tablets 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 tablets should ordinarily be discontinued. If a decision is made to continue MIRAPEX tablets, 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: Dopamine agonists, in clinical studies and clinical experience, appear to impair the systemic regulation of blood pressure, with resulting orthostatic hypotension, especially during dose escalation. Parkinson's disease patients, in addition, appear to have an impaired capacity to respond to an orthostatic challenge. For these reasons, both Parkinson's disease patients and RLS patients being treated with dopaminergic agonists ordinarily require careful monitoring for signs and symptoms of orthostatic hypotension, especially during dose escalation, and should be informed of this risk (see PRECAUTIONS, Information for Patients).

In clinical trials of pramipexole, however, and despite clear orthostatic effects in normal volunteers, the reported incidence of clinically significant orthostatic hypotension was not greater among those assigned to MIRAPEX tablets than among those assigned to placebo. This result, especially with the higher doses used in Parkinson's disease, is clearly unexpected in light of the previous experience with the risks of dopamine agonist therapy.

While this finding could reflect a unique property of pramipexole, it might also be explained by the conditions of the study and the nature of the population enrolled in the clinical trials. Patients were very carefully titrated, and patients with active cardiovascular disease or significant orthostatic hypotension at baseline were excluded. Also, clinical trials in patients with RLS did not incorporate orthostatic challenges with intensive blood pressure monitoring done in close temporal proximity to dosing.

Hallucinations: In the three double-blind, placebo-controlled trials in early Parkinson's disease, hallucinations were observed in 9% (35 of 388) of patients receiving MIRAPEX tablets, compared with 2.6% (6 of 235) of patients receiving placebo. In the four double-blind, placebo-controlled trials in advanced Parkinson's disease, where patients received MIRAPEX tablets and concomitant levodopa, hallucinations were observed in 16.5% (43 of 260) of patients receiving MIRAPEX tablets compared with 3.8% (10 of 264) of patients receiving placebo. Hallucinations were of sufficient severity to cause discontinuation of treatment in 3.1% of the early Parkinson's disease patients and 2.7% of the 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 the early Parkinson's disease patients, the risk of hallucinations was 1.9 times greater than placebo in patients younger than 65 years and 6.8 times greater than placebo in patients older than 65 years. In the advanced Parkinson's disease patients, the risk of hallucinations was 3.5 times greater than placebo in patients younger than 65 years and 5.2 times greater than placebo in patients older than 65 years.

In the RLS clinical program, one pramipexole-treated patient (of 889) reported hallucinations; this patient discontinued treatment and the symptoms resolved.

PRECAUTIONS

Rhabdomyolysis: A single case of rhabdomyolysis occurred in a 49-year-old male with advanced Parkinson's disease treated with MIRAPEX tablets. The patient was hospitalized with an elevated CPK (10,631 IU/L). The symptoms resolved with discontinuation of the medication. **Renal:** Since pramipexole is eliminated through the kidneys, caution should be exercised when prescribing MIRAPEX tablets to patients with renal insufficiency (see DOSAGE AND ADMINISTRATION in full Prescribing Information). **Dyskinesia:** MIRAPEX tablets may potentiate the dopaminergic side effects of levodopa and may cause or exacerbate preexisting dyskinesia. Decreasing the dose of levodopa 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. Evaluation of the retinas of albino mice, monkeys, and minipigs did not reveal similar changes. 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 ANIMAL TOXICOLOGY).

Events Reported with Dopaminergic Therapy: Although the events enumerated below may not have been reported in association with the use of pramipexole in its development program, 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:** Although not reported with pramipexole in the clinical development program, 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 antiparkinsonian therapy. **Fibrotic Complications:** Although not reported with pramipexole in the clinical development program, cases of retroperitoneal fibrosis, pulmonary infiltrates, pleural effusion, and pleural thickening, pericarditis, and cardiac valvulopathy 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.

A small number of reports have been received of possible fibrotic complications, including peritoneal fibrosis, pleural fibrosis, and pulmonary fibrosis in the post-marketing experience for MIRAPEX tablets. While the evidence is not sufficient to establish a causal relationship between MIRAPEX tablets and these fibrotic complications, a contribution of MIRAPEX tablets cannot be completely ruled out in rare cases. **Melanoma:** Some epidemiologic studies have shown that patients with Parkinson's disease have a higher risk (perhaps 2- to 4-fold higher) of developing melanoma than the general population. Whether the observed increased risk was due to Parkinson's disease or other factors, such as drugs used to treat Parkinson's disease, was unclear. MIRAPEX tablets are one of the dopamine agonists used to treat Parkinson's disease. Although MIRAPEX tablets have not been associated with an increased risk of melanoma specifically, its potential role as a risk factor has not been systematically studied. Patients using MIRAPEX tablets for any indication should be made aware of these results and should undergo periodic dermatologic screening.

Impulse Control/Compulsive Behaviors: Cases of pathological gambling, hypersexuality, and compulsive eating (including binge eating) have been reported in patients treated with dopamine agonist therapy, including pramipexole therapy. As described in the literature, such behaviors are generally reversible upon dose reduction or treatment discontinuation.

Rebound and Augmentation in RLS: Reports in the literature indicate treatment of RLS with dopaminergic medications can result in a shifting of symptoms to the early morning hours, referred to as rebound. Rebound was not reported in the clinical trials of MIRAPEX tablets but the trials were generally not of sufficient duration to capture this phenomenon. Augmentation has also been described during therapy for RLS. Augmentation refers to the earlier onset of symptoms in the evening (or even the afternoon), increase in symptoms, and spread of symptoms to involve other extremities. In a controlled trial of MIRAPEX tablets for RLS, approximately 20% of both the MIRAPEX- and placebo-treated patients reported at least a 2-hour earlier onset of symptoms during the day by the end of 3 months of treatment. The frequency and severity of augmentation and/or rebound after longer-term use of MIRAPEX tablets and the appropriate management of these events have not been adequately evaluated in controlled clinical trials.

Information for Patients (also see Patient Package Insert): Patients should be instructed to take MIRAPEX tablets only as prescribed.

Patients should be alerted to the potential sedating effects associated with MIRAPEX tablets, 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 tablets to gauge whether or not it affects their mental and/or motor performance adversely. Patients should be advised 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 tablets

and when taking concomitant medications that increase plasma levels of pramipexole (e.g., cimetidine).

Patients should be informed that hallucinations can occur and that the elderly are at a higher risk than younger patients with Parkinson's disease. In clinical trials, patients with RLS treated with pramipexole rarely reported hallucinations.

Patients and caregivers should be informed that impulse control disorders/compulsive behaviors may occur while taking medicines to treat Parkinson's disease or RLS, including MIRAPEX tablets. These include pathological gambling, hypersexuality, and compulsive eating (including binge eating). If such behaviors are observed with MIRAPEX tablets, dose reduction or treatment discontinuation should be considered.

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, 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 MIRAPEX tablets.

Because the teratogenic potential of pramipexole has not been completely established in laboratory animals, and because experience in humans is limited, patients should be advised to notify their physicians if they become pregnant or intend to become pregnant during therapy (see PRECAUTIONS, Pregnancy).

Because of the possibility that pramipexole 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. If patients develop nausea, they should be advised that taking MIRAPEX tablets with food may reduce the occurrence of nausea.

Laboratory Tests: During the development of MIRAPEX tablets, 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 in his or her care.

Drug Interactions: *Carbidopa/levodopa:* Carbidopa/levodopa did not influence the pharmacokinetics of pramipexole in healthy volunteers (N=10). 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:* In healthy volunteers (N=11), selegiline did not influence the pharmacokinetics of pramipexole. *Amantadine:* Population pharmacokinetic analyses suggest that amantadine may slightly decrease the oral clearance of pramipexole. *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 (N=12). *Probenecid:* Probenecid, a known inhibitor of renal tubular secretion of organic acids via the anionic transporter, did not noticeably influence pramipexole pharmacokinetics (N=12). *Other drugs eliminated via renal secretion:* Population pharmacokinetic analysis suggests that coadministration of drugs that are secreted by the cationic transport system (e.g., cimetidine, ranitidine, diltiazem, triamterene, verapamil, quinidine, and quinidine) decreases the oral clearance of pramipexole by about 20%, while those secreted by the anionic transport system (e.g., cephalosporins, penicillins, indomethacin, hydrochlorothiazide, and chlorpromazine) are likely to have little effect on the oral clearance of pramipexole. *CYP Interactions:* Inhibitors of cytochrome P450 enzymes would not be expected to affect pramipexole elimination because pramipexole is not appreciably metabolized by these enzymes in vivo or in vitro. Pramipexole does not inhibit CYP enzymes CYP1A2, CYP2C9, CYP2C19, CYP2E1, and CYP3A4. Inhibition of CYP2D6 was observed with an apparent Ki of 30 μM, indicating that pramipexole will not inhibit CYP enzymes at plasma concentrations observed following the clinical dose of 4.5 mg/day (1.5 mg TID). *Dopamine antagonists:* Since pramipexole is a dopamine agonist, it is possible that dopamine antagonists, such as the neuroleptics (phenothiazines, butyrophenones, thioxanthenes) or metoclopramide, may diminish the effectiveness of MIRAPEX tablets.

Drug/Laboratory Test Interactions: There are no known interactions between MIRAPEX tablets and laboratory tests.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Two-year carcinogenicity studies with pramipexole have been conducted in mice and rats. Pramipexole was administered in the diet to Chbb;NMR1 mice at doses of 0.3, 2, and 10 mg/kg/day [0.3, 2.2, and 11 times the Maximum Recommended Human Dose (MRHD) (MRHD of 1.5 mg TID on a mg/m² basis)]. Pramipexole was administered in the diet to Wistar rats at 0.3, 2, and 8 mg/kg/day (plasma AUCs were 0.3, 2.5, and 12.5 times the AUC in humans at the MRHD). No significant increases in tumors occurred in either species.

Pramipexole was not mutagenic or clastogenic in a battery of assays, including 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, pramipexole at a dose of 2.5 mg/kg/day (5 times the MRHD on a mg/m² basis), prolonged estrus cycles and inhibited implantation. These effects were associated with reductions in serum levels of prolactin, a hormone necessary for implantation and maintenance of early pregnancy in rats.

Pregnancy: Teratogenic Effect: Pregnancy Category C: When pramipexole was given to female rats throughout pregnancy, implantation was inhibited at a dose of 2.5 mg/kg/day (5 times the MRHD on a mg/m² basis). Administration of 1.5 mg/kg/day of pramipexole to pregnant rats during the period of organogenesis (gestation days 7 through 16) resulted in a high incidence of total resorption of embryos. The plasma AUC in rats at this dose was 4 times the AUC in humans at the MRHD. These findings are thought to be due to the prolactin-lowering effect of pramipexole, 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, the teratogenic potential of pramipexole could not be adequately evaluated. There was no evidence of adverse effects on embryo-fetal development following administration of up to 10 mg/kg/day to pregnant rabbits during organogenesis (plasma AUC was 71 times that in humans at the MRHD). Postnatal growth was inhibited in the offspring of rats treated with 0.5 mg/kg/day (approximately equivalent to the MRHD on a mg/m² basis) or greater during the latter part of pregnancy and throughout lactation. There are no studies of pramipexole in human pregnancy. Because animal reproduction studies are not always predictive of human response, pramipexole should be used 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 the breast milk of lactating rats. Concentrations of radioactivity in milk were three to six times higher than concentrations in plasma at equivalent time points.

Other studies have shown that pramipexole treatment resulted in an inhibition of 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 of the potential for serious adverse reactions in nursing infants from pramipexole, a decision should be made as to whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: The safety and efficacy of MIRAPEX tablets in pediatric patients has not been established.

Geriatric Use: Pramipexole total oral clearance was approximately 30% lower in subjects older than 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 with Parkinson's disease patients, 38.7% of patients were older than 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 the use of MIRAPEX tablets was increased in the elderly. In clinical studies with RLS patients, 22% of patients were at least 65 years old. There were no apparent differences in efficacy or safety between older and younger patients.

ADVERSE EVENTS

Parkinson's Disease: During the premarketing development of pramipexole, patients with either early or advanced Parkinson's disease were enrolled in clinical trials. Apart from the severity and duration of their disease, the two populations differed in their use of concomitant levodopa therapy. Patients with early disease did not receive concomitant levodopa therapy during treatment with pramipexole; those with advanced Parkinson's disease all received concomitant levodopa treatment. Because these two populations may have differential risks for various adverse events, this section will, in general, present adverse-event data for these two populations separately.

Because the controlled trials performed during premarketing development all used a titration design, with a resultant confounding of time and dose, it was impossible to adequately evaluate the effects of dose on the incidence of adverse events.

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

Approximately 12% of 388 patients with early Parkinson's disease and treated with MIRAPEX tablets who participated in the double-blind, placebo-controlled trials discontinued treatment due to adverse events compared with 11% of 235 patients who received placebo. The adverse events most commonly causing discontinuation of treatment were related to the nervous system (hallucinations [3.1% on MIRAPEX tablets vs 0.4% on placebo]; dizziness [2.1% on MIRAPEX tablets vs 1% on placebo]; somnolence [1.6% on MIRAPEX tablets vs 0% on placebo]; extrapyramidal syndrome [1.6% on MIRAPEX tablets vs 6.4% on placebo]; headache and confusion [1.3% and 1.0%, respectively, on MIRAPEX tablets vs 0% on placebo]); and gastrointestinal system (nausea [2.1% on MIRAPEX tablets vs 0.4% on placebo]).

Adverse-event Incidence in Controlled Clinical Studies in Early Parkinson's Disease: This section lists treatment-emergent adverse events that occurred in the double-blind, placebo-controlled studies in early Parkinson's disease that were reported by 1% or more of patients treated with MIRAPEX tablets and were numerically more frequent than in the placebo group. In these studies, patients did not receive concomitant levodopa. Adverse events were usually mild or moderate in intensity.

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. However, the cited figures do provide the prescribing physician with some basis for estimating the relative contribution of drug and nondrug factors to the adverse-event incidence rate in the population studied.

Treatment-emergent adverse events are listed by body system in order of decreasing incidence for MIRAPEX tablets (N=388) vs placebo (N=235), respectively. **Body as a whole:** asthenia (14% vs 12%), general edema (5% vs 3%), malaise (2% vs 1%), reaction unclassifiable (2% vs 1%), fever (1% vs 0%). **Digestive system:** nausea (28% vs 18%), constipation (14% vs 6%), anorexia (4% vs 2%), dysphagia (2% vs 0%). **Metabolic and nutritional system:** peripheral edema (6% vs 4%), decreased weight (2% vs 0%). **Nervous system:** dizziness (25% vs 24%), somnolence (22% vs 9%), insomnia (17% vs 12%), hallucinations (9% vs 3%), confusion (4% vs 1%), amnesia (4% vs 2%), hyposthesia (3% vs 1%), dystonia (2% vs 1%), akathisia (2% vs 0%), thinking abnormalities (2% vs 0%), decreased libido (1% vs 0%), myoclonus (1% vs 0%). **Special senses:** vision abnormalities (3% vs 0%). **Urogenital system:** impotence (2% vs 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% or more of patients with early Parkinson's disease and treated with MIRAPEX tablets 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 the four double-blind, placebo-controlled trials of patients with advanced Parkinson's disease, the most commonly observed adverse events (>5%) that were numerically more frequent in the group treated with MIRAPEX tablets 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® (pramipexole dihydrochloride) tablets and concomitant levodopa in the double-blind, placebo-controlled trials discontinued treatment due to adverse events compared with 16% of 264 patients who received placebo and concomitant levodopa. The events most commonly causing discontinuation of treatment were related to the nervous system (hallucinations [2.7% on MIRAPEX tablets vs 0.4% on placebo]; dyskinesia [1.9% on MIRAPEX tablets vs 0.8% on placebo]; extrapyramidal syndrome [1.5% on MIRAPEX tablets vs 4.9% on placebo]; dizziness [1.2% on MIRAPEX tablets vs 1.5% on placebo]; confusion [1.2% on MIRAPEX tablets vs 2.3% on placebo]); and cardiovascular system (postural [orthostatic] hypotension [2.3% on MIRAPEX tablets vs 1.1% on placebo]).

Adverse-event Incidence in Controlled Clinical Studies in Advanced Parkinson's Disease: This section lists treatment-emergent adverse events that occurred in the double-blind, placebo-controlled studies in advanced Parkinson's disease that were reported by 1% or more of patients treated with MIRAPEX tablets and were numerically more frequent than in the placebo group. In these studies, MIRAPEX tablets or placebo was administered to patients who were also receiving concomitant levodopa. Adverse events were usually mild or moderate in intensity.

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. 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-event incidence rate in the population studied.

Treatment-emergent adverse events are listed by body system in order of decreasing incidence for MIRAPEX tablets (N=260) vs placebo (N=264), respectively. **Body as a whole:** accidental injury (17% vs 15%), asthenia (10% vs 8%), general edema (4% vs 3%), chest pain (3% vs 2%), malaise (3% vs 2%). **Cardiovascular system:** postural hypotension (53% vs 48%). **Digestive system:** constipation (10% vs 9%), dry mouth (7% vs 3%). **Metabolic and nutritional system:** peripheral edema (2% vs 1%), increased creatine PK (1% vs 0%). **Musculoskeletal system:** arthritis (3% vs 1%), twitching (2% vs 0%), bursitis (2% vs 0%), myasthenia (1% vs 0%). **Nervous system:** dyskinesia (47% vs 31%), extrapyramidal syndrome (28% vs 26%), insomnia (27% vs 22%), dizziness (26% vs 25%), hallucinations (17% vs 4%), dream abnormalities (11% vs 10%), confusion (10% vs 7%), dystonia (8% vs 7%), gait abnormalities (7% vs 5%), hypertonia (7% vs 6%), asthenia (6% vs 4%), akathisia (3% vs 2%), thinking abnormalities (3% vs 2%), paranoid reaction (2% vs 0%), delusions (1% vs 0%), sleep disorders (1% vs 0%). **Respiratory system:** dyspnea (4% vs 3%), rhinitis (3% vs 1%), pneumonia (2% vs 0%). **Skin and appendages:** skin disorders (2% vs 1%). **Special senses:** accommodation abnormalities (4% vs 2%), vision abnormalities (3% vs 1%), diplopia (1% vs 0%). **Urogenital system:** urinary frequency (6% vs 3%), urinary tract infection (4% vs 3%), urinary incontinence (2% vs 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% or more of patients with advanced Parkinson's disease and treated with MIRAPEX tablets 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.

Restless Legs Syndrome: MIRAPEX tablets for treatment of RLS have been evaluated for safety in 889 patients, including 427 treated for over six months and 75 for over one year.

The overall safety assessment focuses on the results of three double-blind, placebo-controlled trials, in which 575 patients with RLS were treated with MIRAPEX tablets for up to 12 weeks. The most commonly observed adverse events with MIRAPEX tablets in the treatment of RLS (observed in >5% of pramipexole-treated patients and at a rate at least twice that observed in placebo-treated patients) were nausea and somnolence. Occurrences of nausea and somnolence in clinical trials were generally mild and transient.

Approximately 7% of 575 patients treated with MIRAPEX tablets during the double-blind periods of three placebo-controlled trials discontinued treatment due to adverse events compared to 5% of 223 patients who received placebo. The adverse event most commonly causing discontinuation of treatment was nausea (1%).

This section lists treatment-emergent events that occurred in three double-blind, placebo-controlled studies in RLS patients that were reported by 2% or more of patients treated with MIRAPEX tablets and were numerically more frequent than in the placebo group.

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. 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-event incidence rate in the population studied.

Treatment-emergent adverse events are listed by body system in order of decreasing incidence for MIRAPEX tablets (N=575) vs placebo (N=223), respectively. **Gastrointestinal disorders:** nausea (16% vs 5%), constipation (4% vs 1%), diarrhea (4% vs 1%), dry mouth (3% vs 1%). **General disorders and administration site conditions:** fatigue (9% vs 7%). **Infections and infestations:** influenza (3% vs 1%). **Nervous system disorders:** headache (16% vs 15%), somnolence (6% vs 3%). Patients may have reported multiple adverse experiences during the study or at discontinuation; thus, patients may be included in more than one category.

This section summarizes data for adverse events that appeared to be dose related in the 12-week fixed dose study. Dose related adverse events in a 12-week, double-blind, placebo-controlled, fixed dose study in Restless Legs Syndrome (occurring in 5% or more of all patients in the treatment phase) are listed by body system in order of decreasing incidence for MIRAPEX (0.25 mg [N=88]; 0.5 mg [N=80]; 0.75 mg [N=90]) vs placebo (n=86), respectively. **Gastrointestinal disorders:** nausea (11%; 19%; 27% vs 5%), diarrhea (3%; 1%; 7% vs 0%), dyspepsia (3%; 1%; 4% vs 7%). **Infections and infestations:** influenza (1%; 4%; 7% vs 1%). **General disorders and administration site conditions:** fatigue (3%; 5%; 7% vs 5%). **Psychiatric disorders:** insomnia (9%; 9%; 13% vs 9%), abnormal dreams (2%; 1%; 8% vs 2%). **Respiratory, thoracic and mediastinal disorders:** nasal congestion (0%; 3%; 6% vs 1%). **Musculoskeletal and connective tissue disorders:** pain in extremity (3%; 3%; 7% vs 1%).

Other events reported by 2% or more of RLS patients treated with MIRAPEX tablets but equally or more frequently in the placebo group were: vomiting, nasopharyngitis, back pain, pain in extremity, dizziness, and insomnia.

General

Adverse Events: Relationship to Age, Gender, and Race: Among the treatment-emergent adverse events in patients treated with MIRAPEX tablets, hallucination appeared to exhibit a positive relationship to age in patients with Parkinson's disease. Although no gender-related differences were observed in Parkinson's disease patients, nausea and fatigue, both generally transient, were more frequently reported by female than male RLS patients. Less than 4% of patients enrolled were non-Caucasian, therefore, an evaluation of adverse events related to race is not possible.

Other Adverse Events Observed During Phase 2 and 3 Clinical Trials: MIRAPEX tablets have been administered to 1620 Parkinson's disease patients and to 889 RLS patients in Phase 2 and 3 clinical trials. During these trials, all adverse events were recorded by the clinical investigators using terminology of their own choosing; similar types of events were grouped into a smaller number of standardized categories using MedDRA dictionary terminology. These categories are used in the listing below. Adverse events which are not listed above but occurred on at least two occasions (one occasion if the event was serious) in the 2509 individuals exposed to MIRAPEX tablets are listed below. The reported events below are included without regard to determination of a causal relationship to MIRAPEX tablets.

Blood and lymphatic system disorders: anemia, iron deficiency anemia, leukocytosis, leukopenia, lymphadenitis, lymphadenopathy, thrombocytopenia, thrombocytopenia. **Cardiac disorders:** angina pectoris, arrhythmia supraventricular, atrial fibrillation, atrioventricular block first degree, atrioventricular block second degree, bradycardia, bundle branch block, cardiac arrest, cardiac failure, cardiac failure congestive, cardiomegaly, coronary artery occlusion, cyanosis, extrasystoles, left ventricular failure, myocardial infarction, nodal arrhythmia, sinus arrhythmia, sinus bradycardia, sinus tachycardia, supraventricular extrasystoles, supraventricular tachycardia, tachycardia, ventricular fibrillation, ventricular extrasystoles, ventricular hypertrophy. **Congenital, familial and genetic disorders:** atrial septal defect, congenital foot malformation, spine malformation. **Ear and labyrinth disorders:** deafness, ear pain, hearing impaired, hypacusis, motion sickness, vestibular ataxia. **Endocrine disorders:** goiter, hyperthyroidism, hypothyroidism. **Eye disorders:** amaurosis fugax, blepharitis, blepharospasm, cataract, dacryostenosis acquired, dry eye, eye hemorrhage, eye irritation, eye pain, eyelid edema, eyelid ptosis, glaucoma, keratitis, macular degeneration, myopia, photophobia, retinal detachment, retinal vascular disorder, scotoma, vision blurred, visual acuity reduced, vitreous floaters. **Gastrointestinal disorders:** abdominal discomfort, abdominal distension, aphthous stomatitis, ascites, cheilitis, colitis, colitis ulcerative, duodenal ulcer, duodenal ulcer hemorrhage, enteritis, eructation, fecal incontinence, gastric ulcer, gastric ulcer hemorrhage, gastritis, gastrointestinal hemorrhage, gastroesophageal reflux disease, gingivitis, haematemesis, haematochezia, hemorrhoids, hiatus hernia, hyperchlorhydria, ileus, inguinal hernia, intestinal obstruction, irritable bowel syndrome, esophageal spasm, esophageal stenosis, esophagitis, pancreatitis, peritonitis, rectal hemorrhage, reflux esophagitis, tongue edema, tongue ulceration, toothache, umbilical hernia. **General disorders:** chest discomfort, chills, death, drug

withdrawal syndrome, face edema, feeling cold, feeling hot, feeling jittery, gait disturbance, impaired healing, influenza-like illness, irritability, localized edema, edema, pitting edema, thirst. **Hepatology disorders:** biliary colic, cholecystitis, cholelithiasis, cholelithiasis. **Immune system disorders:** drug hypersensitivity. **Infections and infestations:** abscess, acute tonsillitis, appendicitis, bronchitis, bronchitis, bronchopneumonia, cellulitis, cystitis, dental caries, diverticulitis, ear infection, eye infection, folliculitis, fungal infection, furuncle, gangrene, gastroenteritis, gingival infection, herpes simplex, herpes zoster, herpetic, intervertebral discitis, laryngitis, lobar pneumonia, nail infection, onychomycosis, oral candidiasis, orchitis, osteomyelitis, otitis externa, otitis media, paronychia, pyelonephritis, pyoderma, sepsis, skin infection, tonsillitis, tooth abscess, tooth infection, upper respiratory tract infection, urethritis, vaginal candidiasis, vaginal infection, viral infection, wound infection. **Injury, poisoning and procedural complications:** accidental falls, drug toxicity epicondylitis, road traffic accident, sunburn, tendon rupture. **Metabolism and nutrition disorders:** cachexia, decreased appetite, dehydration, diabetes mellitus, fluid retention, gout, hypercholesterolemia, hyperglycemia, hyperlipidemia, hyperuricemia, hypocalcemia, hypoglycemia, hypokalemia, hyponatremia, hypovitaminosis, increased appetite, metabolic alkalosis. **Musculoskeletal and connective tissue disorders:** bone pain, fasciitis, flank pain, intervertebral disc disorder, intervertebral disc protrusion, joint effusion, joint stiffness, joint swelling, monarthritides, muscle rigidity, muscle spasms, musculoskeletal stiffness, myopathy, myositis, nuchal rigidity, osteoarthritis, osteonecrosis, osteoporosis, polymyalgia, rheumatoid arthritis, shoulder pain, spinal osteoarthritis, tendonitis, tenosynovitis. **Neoplasms benign, malignant and unspecified:** abdominal neoplasm, adenocarcinoma, adenoma benign, basal cell carcinoma, bladder cancer, breast cancer, breast neoplasm, chronic lymphocytic leukemia, colon cancer, colorectal cancer, endometrial cancer, gallbladder cancer, gastric cancer, gastrointestinal neoplasm, hemangioma, hepatic neoplasm, hepatic neoplasm malignant, lip and/or oral cavity cancer, lung neoplasm malignant, lung cancer metastatic, lymphoma, malignant melanoma, melanocytic naevus, metastases to lung, multiple myeloma, oral neoplasm benign, neoplasm, neoplasm malignant, neoplasm prostate, neoplasm skin, neurofibroma, ovarian cancer, prostatic cancer, prostatic adenoma, pseudo lymphoma, renal neoplasm, skin cancer, skin papilloma, squamous cell carcinoma, thyroid neoplasm, uterine leiomyoma. **Nervous system disorders:** ageusia, akinesia, anticholinergic syndrome, aphasia, balance disorder, brain edema, carotid artery occlusion, carpal tunnel syndrome, cerebral artery embolism, cerebral hemorrhage, cerebral infarction, cerebral ischemia, chorea, cognitive disorder, coma, convulsion, coordination abnormal, dementia, depressed level of consciousness, disturbance in attention, dizziness postural, dysarthria, dysgraphia, facial palsy, grand mal convulsion, hemiplegia, hyperaesthesia, hyperkinesia, hyperreflexia, hyperreflexia, hypotonia, lethargy, loss of consciousness, memory impairment, migraine, muscle contractions involuntary, narcolepsy, neuralgia, neuropathy, nystagmus, parosmia, psychomotor hyperactivity, scintal, sedation, sensory disturbance, sleep phase rhythm disturbance, sleep talking, stupor, syncope vasovagal, tension headache. **Psychiatric disorders:** affect lability, aggression, agitation, bradyphrenia, bruism, suicide, delirium, delusional disorder persecutory type, disorientation, dissociation, emotional distress, euphoric mood, hallucination auditory, hallucination visual, initial insomnia, libido increased, mania, middle insomnia, mood altered, nightmare, obsessive thoughts, obsessive-compulsive disorder, panic reaction, parosmia, personality disorder, psychotic disorder, restlessness, sleep walking, suicidal ideation. **Renal and urinary disorders:** chromaturia, dysuria, glycosuria, hematuria, urgency, nephrolithiasis, neurogenic bladder, nocturia, oliguria, pollakiuria, proteinuria, renal artery stenosis, renal colic, renal cyst, renal failure, renal impairment, urinary retention. **Reproductive system and breast disorders:** amenorrhea, breast pain, dysmenorrhea, epididymitis, gynaecostasia, menopausal symptoms, menorrhagia, metrorrhagia, ovarian cyst, priapism, prostaticitis, sexual dysfunction, uterine hemorrhage, vaginal discharge, vaginal hemorrhage. **Respiratory, thoracic and mediastinal disorders:** apnea, aspiration, asthma, chronic obstructive pulmonary disease, dry throat, dysphonia, dyspnea external, epistaxis, hyperaesthesia, hiccups, hyperventilation, increased bronchial secretion, laryngospasm, nasal dryness, nasal polyps, obstructive airways disorder, pharyngolaryngeal pain, pleurisy, pneumonia aspiration, pneumothorax, postnasal drip, productive cough, pulmonary embolism, pulmonary edema, respiratory alkalosis, respiratory distress, respiratory failure, respiratory tract congestion, rhinitis allergic, rhinorrhoea, sinus congestion, sleep apnoea syndrome, sneezing, snoring, tachypnea, wheezing. **Skin and subcutaneous tissue disorders:** acne, alopecia, cold sweat, dermal cyst, dermatitis, dermatitis bullous, dermatitis contact, dry skin, ecchymosis, eczema, erythema, hyperkeratosis, livido reticularis, night sweats, periorbital edema, petechiae, photosensitivity allergic reaction, psoriasis, purpura, rash erythematous, rash maculo-papular, rash papular, rosacea, seborrhea, seborrheic dermatitis, skin burning sensation, skin discoloration, skin exfoliation, skin hyperpigmentation, skin hypertrophy, skin irritation, skin nodule, skin odor abnormal, skin ulcer, urticaria. **Vascular disorders:** aneurysm, angiopathy, arteriosclerosis, circulatory collapse, deep vein thrombosis, embolism, hematoma, hot flush, hypertensive crisis, lymphoedema, pallor, phlebitis, Raynaud's phenomenon, shock, thrombophlebitis, thrombosis, varicose vein.

Falling Asleep During Activities of Daily Living: Patients treated with MIRAPEX tablets 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, primarily in Parkinson's disease patients. 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 pramipexole tablets. Similar types of events were grouped into a smaller number of standardized categories using the MedDRA dictionary: abnormal behavior, abnormal dreams, accidents (including fall), blackouts, fatigue, hallucinations (all kinds), headache, hypotension (including postural hypotension), increased eating (including binge eating, compulsive eating, and hyperphagia), libido disorders (including increased and decreased libido, and hypersexuality), pathological gambling, pruritus, syncope, and weight increase.

DRUG ABUSE AND DEPENDENCE

Pramipexole is not a controlled substance. Pramipexole has not been systematically studied in animals or humans for its potential for abuse, tolerance, or physical dependence. However, in a rat model on cocaine self-administration, pramipexole had little or no effect.

OVERDOSAGE

There is no clinical experience with massive overdose. One patient, with a 10-year history of schizophrenia, took 11 mg/day of pramipexole for 2 days in a clinical trial to evaluate the effect of pramipexole in schizophrenic patients. No adverse events were reported related to the increased 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 overdose of a dopamine agonist. If signs of central nervous system stimulation are present, a phenothiazine or other butyrophenone neuroleptic agent may be indicated; the efficacy of such drugs in reversing the effects of overdose has not been assessed. Management of overdose may require general supportive measures along with gastric lavage, intravenous fluids, and electrocardiogram monitoring.

ANIMAL TOXICOLOGY

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 with pramipexole. These findings were first observed during week 76 and were dose dependent in animals receiving 2 or 8 mg/kg/day (plasma AUCs equal to 2.5 and 12.5 times the AUC in humans that received 1.5 mg TID). In a similar study of pigmented rats with 2 years' exposure to pramipexole at 2 or 8 mg/kg/day, retinal degeneration was not diagnosed. Animals given drug had thinning in the outer nuclear layer of the retina that was only slightly greater than that seen in control rats utilizing morphometry.

Investigative studies demonstrated that pramipexole reduced the rate of disk shedding from the photoreceptor rod cells of the retina in albino rats, which was associated with enhanced sensitivity to the damaging effects of light. In a comparative study, degeneration and loss of photoreceptor cells occurred in albino rats after 13 weeks of treatment with 25 mg/kg/day of pramipexole (54 times the highest clinical dose on a mg/m² basis) and constant light (100 lux) but not in pigmented rats exposed to the same dose and higher light intensities (500 lux). Thus, the retina of albino rats is considered to be uniquely sensitive to the damaging effects of pramipexole and light. Similar changes in the retina did not occur in a 2-year carcinogenicity study in albino mice treated with 0.3, 2, or 10 mg/kg/day (0.3, 2.2 and 11 times the highest clinical dose on a mg/m² basis). Evaluation of the retinas of monkeys given 0.1, 0.5, or 2.0 mg/kg/day of pramipexole (0.4, 2.2, and 8.6 times the highest clinical dose on a mg/m² basis) for 12 months and minipigs given 0.3, 1, or 5 mg/kg/day of pramipexole for 13 weeks also detected no changes. 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.

Fibro-ossous Proliferative Lesions in Mice: An increased incidence of fibro-ossous proliferative lesions occurred in the femurs of male mice treated for 2 years with 0.3, 2.0, or 10 mg/kg/day (0.3, 2.2, and 11 times the highest clinical dose on a mg/m² basis). Lesions occurred at a lower rate in control animals. Similar lesions were not observed in male mice or rats and monkeys of either sex that were treated chronically with pramipexole. The significance of this lesion to humans is not known.

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ABOUT MDS

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
Restless legs syndrome
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 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

ABOUT MDS

MDS OFFICERS (2007-2009)



President Anthony Lang, Canada	President-Elect Philip Thompson, Australia	Secretary Olivier Rascol, France	Secretary-Elect Matthew Stern, USA	Treasurer Yoshikuni Mizuno, Japan	Treasurer-Elect Oscar Gershanik, Argentina	Past-President Andrew Lees, United Kingdom
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 Shu-Leong Ho, China
 Karl Kieburz, USA
 Irene Litvan, USA
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 Philip Thompson, Australia
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 Ad-hoc: Serge Przedborski, USA
 Ad-hoc: Kathleen Shannon, USA

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 Co-Chair: Kathleen Shannon, USA
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 Tanya Simuni, USA
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ABOUT MDS

PAST-PRESIDENTS

2005-2006 Andrew Lees, United Kingdom
2003-2004 C. Warren Olanow, USA
2001-2002 Werner Poewe, Austria
1999-2000 Mark Hallett, USA
1997-1998 Eduardo Tolosa, Spain
1995-1996 Joseph Jankovic, USA
1991-1994 C. David Marsden, United Kingdom
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INTERNATIONAL MEDICAL SOCIETY FOR MOTOR DISTURBANCES PAST PRESIDENTS

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

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EDUCATION INFORMATION

To better fulfill its global mission of advancing the neurological sciences as they relate to Movement Disorders, MDS is expanding its educational program. This growing program offers an increasing variety of high caliber continuing medical education and continuing professional development opportunities in Movement Disorders.

VISITING PROFESSOR PROGRAM

This program provides excellent educational opportunities in Movement Disorders to regions of the world not adequately served by resources within that region. Applications will be considered by the Education Committee of the appropriate MDS regional section (MDS, MDS-AOS, or MDS-ES). For more details, visit www.movementdisorders.org/ or call Linda Caples at +1 414-276-2145.

SLIDE SETS

Members use of the slide sets will enable them to become familiar with the differential diagnosis and clinical features that define various common involuntary movements, as well as the course of treatment and complications of Movement Disorders. Slide Sets are available by logging into the Members Only area of the web site at www.movementdisorders.org/membersonly/

Current Slide Sets include:

- Ataxia (PPT) - *Jennifer G. Goldman, MD*
- Chorea (PPT) - *Kathleen M. Shannon, MD*
- The Diagnosis and Management of Dystonia (PPT) - *Steven J. Frucht, MD*
- Myoclonus: Diagnosis and Treatment (PPT) - *Steven J. Frucht, MD*
- Parkinsonism (PPT) - *Kathleen M. Shannon, MD*
- Restless Legs Syndrome (PPT)- *Charles H. Adler, MD, PhD*
- Tics and Tourette Syndrome (PPT) - *Jennifer G. Goldman, MD*

VIDEO LIBRARY

This library consists of video supplements from the *Movement Disorders Journal* since 1986. You may search the video library by keyword, by author, by volume and issue, or a combination of these fields. Search the Video Library after logging into www.movementdisorders.org/membersonly/

CASE OF THE MONTH

Case of the Month (COM) is the new MDS interactive online feature that presents unique and challenging movement disorder cases. MDS members are invited to answer questions after analyzing video and case history, and are provided with the expert's analysis. Please visit the MDS Web site to watch this month's case.

MDS is currently accepting submissions for Case of the Month. Case of the Month provides an opportunity for members to share interesting cases for educational purposes, in a forum dedicated to Movement Disorder experts. For information about submission requirements, including video format and patient consent forms, please visit the MDS Web site at www.movementdisorders.org/

11TH INTERNATIONAL CONGRESS - TEACHING COURSE SYLLABI

Currently available syllabi include:

- Current treatment of Parkinson's disease: Motor symptoms
- Intersection of sleep and Movement Disorders: Evaluation and treatment
- Current management of Parkinson's disease: Non-motor symptoms
- Pediatric Movement Disorders in an office setting: Diagnosis and treatment

As a result of reviewing this material, you 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.

Every 5 Seconds we help improve another life

Medtronic is proud to be a **Gold Supporter** of The Movement Disorder Society's 12th International Congress of Parkinson's Disease and Movement Disorders.

Elena

Activa[®] Deep Brain Stimulation
Therapy for Parkinson's disease

Stop by **Medtronic booth #213** for a **complimentary copy** of The Movement Disorder Society's **2008 Abstracts on CD-ROM** and see a demonstration of the new Activa DBS Patient Referral Advisor software for Parkinson's disease.

UC200805153 EN



Medtronic

Alleviating Pain · Restoring Health · Extending Life

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. The effectiveness of this device for this use has not been demonstrated. **Rx only**

November 8, 2006

EDUCATION INFORMATION

JOURNAL CME

Visit the MDS Web site's educational activities to view a list of available Journal CME articles. The *Movement Disorder Society* (MDS) is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. MDS designates this educational activity for a maximum of 1.0 *AMA PRA Category 1 Credit™*. Physicians should only claim credit commensurate with the extent of their participation in the activity. The latest articles include:

- March 2008: "Glial reactions in Parkinson's disease" Patrick L. McGeer, MD, PhD, and Edith G. McGeer, PhD
- February 2008: "The Role of Executive Function and Attention in Gait" Galit Yogev-Seligmann, MscPT, et al.
- January 2008: "A Systematic Review of Prevalence Studies of Depression in Parkinson's Disease" Jennifer S. A. M. Reijnders, MA, et al.
- January 2008: "Paradoxes of Functional Neurosurgery: Clues from Basal Ganglia Recordings"

AVAILABLE ONLINE

In alignment with our educational mission, The *Movement Disorder Society* is pleased to provide a variety of online activities. These activities aid in expanding the outreach of our educational offerings. The following online activities are now available:

- **Levodopa: The Gold Standard in the Treatment of Parkinson's Disease**
Available through November 1, 2008
At the conclusion of the activity, participants should be able to: know the pros and cons of initiating therapy early with levodopa; describe the late complications of levodopa therapy and their mechanisms of causation; know which symptoms of Parkinson's disease are dopa responsive and which require alternative therapeutic approaches.
- **Targeting A2A Receptors in Parkinson's Disease**
Available through August 1, 2008
At the conclusion of the activity, participants should be able to: describe the role of adenosine system in the basal ganglia in relation to Parkinson's disease; define the potential role of adenosine antagonists in the management of Parkinson's disease; discuss the current evidence for the use of adenosine antagonists in Parkinson's disease.

EDUCATION INFORMATION

- **Dopamine Transporter Imaging in Neurological Practice Web cast**
At the conclusion of this activity, participants should be able to: describe how dopamine transporter imaging is performed and discuss the science underlying the procedure; discuss the interpretation of dopamine transporter images; list the diseases/symptoms for which dopamine transporter imaging may be an appropriate investigative tool; explain how patients suitable for this procedure would be identified; and discuss the current uses, potential future uses, and limitations of dopamine transporter imaging in neurological clinical practice and research applications.

LIVE COURSES

DeNovo Parkinson's Disease

September 13, 2008 - JW Marriott, San Francisco, CA, USA

Parkinson's disease is a common disorder affecting the older population resulting in significant disability. In early stages, diagnosis can be difficult. Use of symptomatic and neuroprotective treatments remains controversial. Through lecture and small group sessions this course will address the issues of accurate diagnosis, assessment of progression and discuss optimizing treatment strategies.

Register now at: www.movementdisorders.org/

The Many Faces of Dystonia: A Frequently Misdiagnosed Disorder

November 1, 2008 – The Adolphus, Dallas, Texas, USA

This course will focus on increasing awareness of dystonia in the general neurology community by addressing topics related to the diagnosis and misdiagnosis of dystonia. Using a video case-based template, the course will highlight focal and generalized dystonia, demonstrating the spectrum of disease from mild to severe and the appropriate work-up. Treatment strategies will be summarized, but not highlighted.

Dopamine Transporter Imaging in Neurological Practice



October 31, 2008

Madrid, Spain



December 5, 2008

Toulouse, France



February 5, 2009

Glasgow, Scotland

This workshop is intended to introduce participants to the potential of dopamine transporter single photon emission computed tomography (SPECT) imaging in neurological practice. The workshop will answer some common questions, such as when it is appropriate for dopamine transporter imaging to be ordered by general practitioners, neurologists or Movement Disorder specialists.

So while some questions can be answered by a number of published dopamine transporter imaging studies of high scientific standard, answers to other questions are highly dependent upon expert opinion. The goal of the workshop will be to present a balanced view of the currently available information on dopamine transporter imaging studies. The scope of information to be presented and discussed has been chosen to not only identify the potential usefulness of dopamine transporter imaging in neurological practice, but also to guard against indiscriminate and injudicious use of dopamine transporter imaging, or erroneous interpretation of findings.

Register now at: www.movementdisorders.org/



De Novo Parkinson's Disease: Diagnosis and Treatment

September 13, 2008 – San Francisco, California

Course Director: Oksana Suchowersky, MD, FRCPC, FCCMG

Parkinson's disease is a common disorder affecting the older population resulting in significant disability. In early stages, diagnosis can be difficult. Use of symptomatic and neuroprotective treatments remains controversial. Through lecture and small group sessions this course will address the issues of accurate diagnosis, assessment of progression and discuss optimizing treatment strategies.

For more information, please visit the course website at www.movementdisorders.org/education/denovo/

MEMBERSHIP INFORMATION

MEMBERSHIP BENEFITS

- 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 live and online CME/CPD activities and reference material on topics in Movement Disorders.
- 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 regional programs, courses and workshops held each year.
- A print directory listing mailing addresses, telephone and fax numbers, and e-mail addresses for all members.
- Access to Members Only information on the MDS Web site at www.movementdisorders.org, including a searchable Membership Directory.
- A quarterly newsletter, entitled *Moving Along*, highlighting current news and views in the field of Movement Disorders.
- Participation in the election of international and regional section leadership representatives.

MEMBERSHIP CATEGORIES

Regular Membership - \$200 (USD) Annually
Clinicians, other healthcare professionals, researchers and policy makers in Movement Disorders.

Junior Membership - \$100 (USD) Annually
Residents, fellows, and those training in healthcare or scientific research. Status must be certified in writing by employer and submitted with payment.

Waived Dues Membership - \$10 (USD) Annually
MDS provides a reduced dues program specifically designed to enable those on a lower income to join the Society.

For more information or to apply online, please go to www.movementdisorders.org/membership/

NEW IN 2008 - NON-MEMBERS APPLYING FOR MEMBERSHIP

Those who have registered at the Non-Member rate will have the opportunity to apply for MDS membership at the International Congress for no additional fee with limited benefits through 2008, and full membership status, receiving the print journal, in 2009. Membership applications will be provided to all non-member attendees on site and must be returned to the MDS booth before the conclusion of the International Congress.

MDS AFFILIATE MEMBER SOCIETIES

The *Movement Disorder Society* (MDS) invites other neurological organizations and groups specializing in Movement Disorders to become Affiliate Members of MDS to encourage research and enhance the education of physicians and the public about Movement Disorders.

Being an Affiliate Member Society entitles your organization to:

- Announce MDS Affiliate Member status on your organization's letterhead and Web site.
- Receive "fast track" consideration of applications for sponsorship, support or endorsement of your organization's scientific meetings.
- Receive MDS mailings on future International Congresses and educational programs, as well as the official newsletter of the MDS, *Moving Along*.

To become an MDS Affiliate Member please submit a formal letter of application as well as the following supporting documents:

- A recent annual report of the activities of your organization
- An organizational mailing list, to include e-mail addresses if available
- A copy of your group's Constitution and Bylaws

Please note that in order to be considered for Affiliate Membership, all of the above documents must be received. Also, 15% of your group's members and all members of your executive committee must be current members of MDS.

No application fee is required to file for Affiliate Membership status, simply send the letter of application and supporting documentation to:

MDS International Secretariat
555 E. Wells Street, Suite 1100
Milwaukee, WI 53202-3823, USA

Fax: +1 414-276-3349

E-mail to pfierst@movementdisorders.org

AUDIO-VISUALS

The *Movement* Disorder Society publishes several audio-visuals available for sale from the MDS International Secretariat.

The titles that are currently available include:

Instructional Videotape for Motor Fluctuation Diaries in Parkinson's Disease

Authored by C.G. Goetz, M. Grobman, L. Blasucci, and G.T. Stebbins. This instructional videotape demonstrates the 3 states of Parkinson's disease, off, on, and on with dyskinesia, with the intent to assist patients in completion of their motor fluctuation diaries. This videotape is 15 minutes.

Toronto-Western Spasmodic Torticollis Rating Scale TWSTRS Training Videotape

Authored by C. Comella, S. Bressman, C.G. Goetz, and A. Lang. The video demonstrates the 10 categories in the TWSTRS scale, with verbal and visual examples of scoring in each category. This video is approximately 1 hour and 25 minutes.

Utility of an Objective Dyskinesia Rating Scale for Parkinson's Disease: (Rush Dyskinesia Rating Scale).

Goetz, et al. *Movement Disorders* Volume 9, Video Supplement. 2. This videotape provides guidelines and rating examples of the Rush Dyskinesia Rating Scale, a scale widely used for evaluating dyskinesias in Parkinson's disease. This videotape is approximately 17 minutes.

Unified Huntington's Disease Rating Scale Videotape
Movement Disorders, Volume 11, Issues 1-3, Videotape Supplement, The Unified Huntington's Disease Rating Scale: Reliability and Consistency. *Mov Disord* 1996;11:136-142. This video is approximately 1 hour and 59 minutes.

Unified Parkinson's Disease Rating Scale Training Videotape

Authored by C. G. Goetz, G.T. Stebbins, T. Chmura, S. Fahn, H. Klawans, and C. D. Marsden. This video demonstrates the different categories of the motor section of the UPDRS, with verbal and visual examples of scoring in each category. This videotape is approximately 1 hour in length.

Standardized Training Tools for the UPDRS Activities of Daily Living Scale" (UPDRS Part II).

Authored by C.G. Goetz, P.A. Lewitt, and M. Weidenman. *Movement Disorders* Volume 18, Video Supplement. 2. This videotape provides suggestions on the application and interview techniques for Part II of the UPDRS with patient examples and guidelines for raters. This videotape is approximately 1 hour and 15 minutes.

All materials are available in DVD or video format. Special reduced rates are available to MDS members. For more information or to place an order, go to www.movementdisorders.org/publications

SAVE the DATE for these Future MDS EDUCATIONAL PROGRAMS

DENOVO PARKINSON'S DISEASE: DIAGNOSIS AND TREATMENT

September 13, 2008 – San Francisco, CA, USA

DOPAMINE TRANSPORTER IMAGING IN NEUROLOGICAL PRACTICE

October 31, 2008 – Madrid, Spain

THE MANY FACES OF DYSTONIA: A FREQUENTLY MISDIAGNOSED DISORDER

November 1, 2008 – Dallas, TX, USA

DOPAMINE TRANSPORTER IMAGING IN NEUROLOGICAL PRACTICE

December 5, 2008 – Toulouse, France

2ND ASIAN AND OCEANIAN PARKINSON'S DISEASE AND MOVEMENT DISORDER CONGRESS

February 15-17, 2009 – New Delhi, India

2ND INTERNATIONAL CONFERENCE ON PSYCHOGENIC MOVEMENT DISORDERS AND OTHER CONVERSION DISORDERS

April 2-4, 2009 – Washington, D.C. USA

13TH INTERNATIONAL CONGRESS OF PARKINSON'S DISEASE AND MOVEMENT DISORDERS

June 7-11, 2009 – Paris, France

14TH INTERNATIONAL CONGRESS OF PARKINSON'S DISEASE AND MOVEMENT DISORDERS

June 13-17, 2010 – Buenos Aires, Argentina

For more information, visit www.movementdisorders.org or e-mail info@movementdisorders.org



A better life – for both of them

Research into Parkinson's disease is making progress towards more effective treatment methods.

Solvay Pharmaceuticals supports this research and actively contributes to its success. Our aim is to help improve the everyday life for patients with Parkinson's disease.

And when Parkinson's disease patients enjoy a better life, so do those close to them.



SOLVAY
PHARMACEUTICALS

INTERNATIONAL CONGRESS REGISTRATION AND VENUE

BADGES

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

Blue = Delegate	Green = Guest
Yellow = Exhibitor	Purple = Press
Orange = Exhibitor Delegate	Black = MDS Staff

DATES

Sunday, June 22, 2008 through Thursday, June 26, 2008

VENUE & HOTEL INFORMATION

Hilton Chicago – *Headquarters Hotel*

720 South Michigan Avenue

Chicago, IL 60605

United States

Tel. +1 (312) 922-4400

Fax +1 (312) 922-5240

All sessions and social events will be held at the Hilton Chicago

Holiday Inn Mart Plaza

350 West Mart Center Drive

Chicago, IL 60654

United States

Tel. +1 (312) 836-5000

Fax +1 (312) 222-9508

Sofitel Chicago Water Tower

20 East Chestnut Street

Chicago, IL 60611

United States

Tel. +1 (312) 324-4000

Fax +1 (312) 324-4206

REGISTRATION DESK

Location: Lower Level

Name badges, session tickets, guest passes and International Congress registration bags can be collected at the International Congress Registration Desk located on the Lower Level of the Hilton Chicago.

REGISTRATION DESK HOURS

Saturday, June 21	16:00 – 20:00
Sunday, June 22	9:00 – 19:30
Monday, June 23	7:00 – 18:00
Tuesday, June 24	7:00 – 18:00
Wednesday, June 25	7:00 – 18:00
Thursday, June 26	7:00 – 16:00

MDS HISTORY EXHIBIT AND HISTORY OF CHICAGO NEUROLOGY EXHIBIT

Location: Mobley Room, Lower Level

The MDS is proud to sponsor two History Exhibits which will be displayed throughout the duration of the International Congress.

History of Movement Disorders Exhibit

Continuing a tradition established by the MDS, an exhibit focusing on the “History of Movement Disorders” will be presented. This exhibit will trace the early development of Movement Disorders as a discipline, as well as the development of MDS as a preeminent International Society.

History of Chicago Neurology

New this year to the MDS History Exhibit Series is the addition of the “History of Chicago Neurology”. This exhibit will provide attendees with a look at the role Chicago played in US Neurology, local neurological societies, seminal Chicago figures and Chicago Neurology today.

Original books, manuscripts, letters, photographs, medical artifacts and instruments 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.

Hours of Operation:

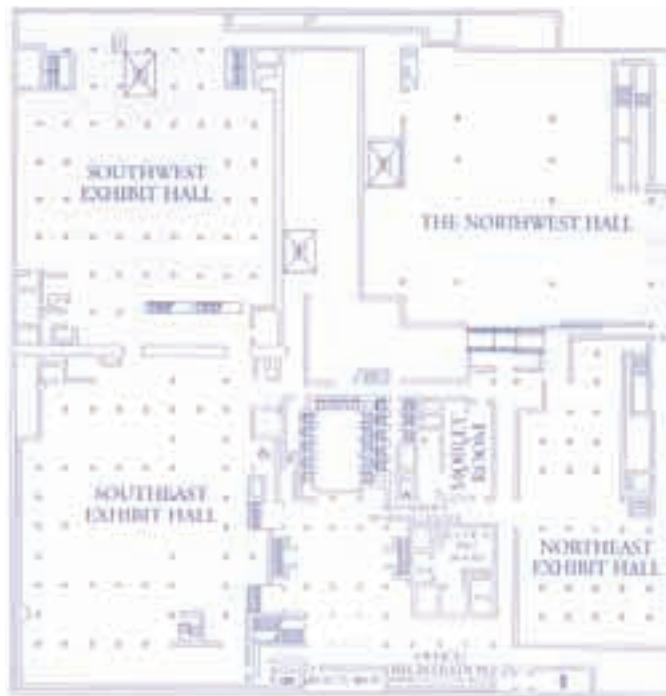
Sunday, June 22:	9:00 - 18:00
Monday, June 23:	7:00 - 18:00
Tuesday, June 24:	7:00 - 18:00
Wednesday, June 25:	7:00 - 18:00
Thursday, June 26:	7:00 - 16:00

Exhibit Organizers:

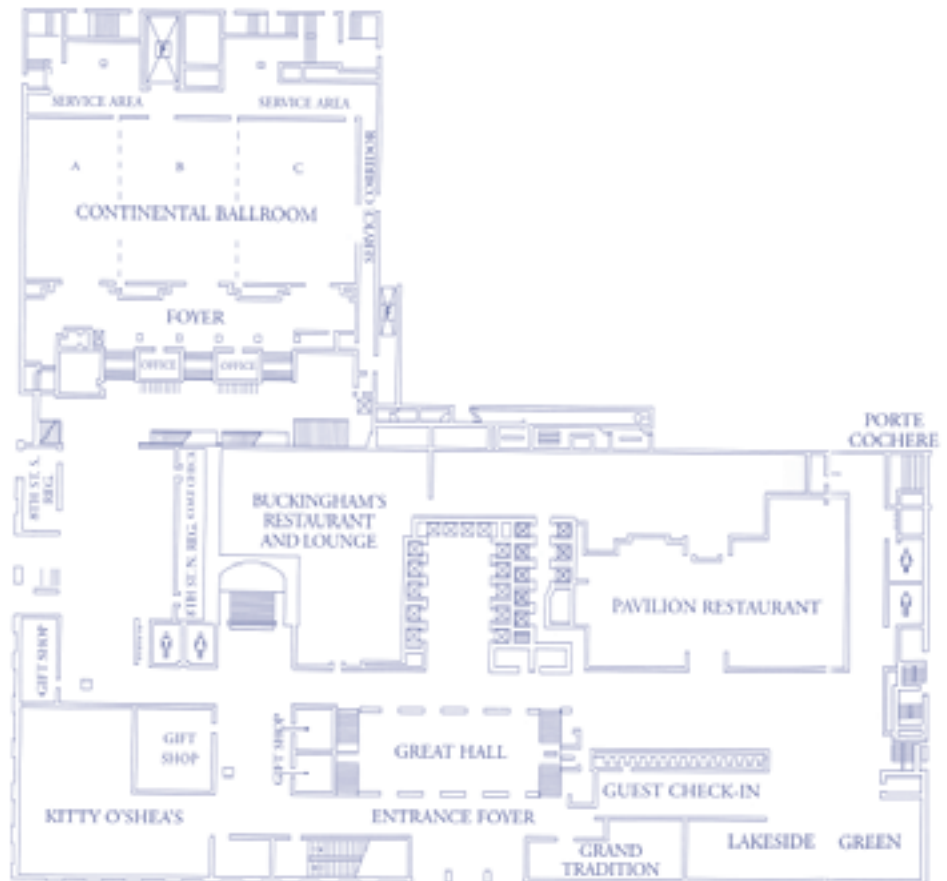
Christopher G. Goetz, MD - Exhibit Director
Douglas Lanska, MD - Associate Director
Teresa A. Chmura, BS - Exhibit Designer
Elena Goetz - Assistant Designer

HILTON FLOOR PLAN

LOWER LEVEL

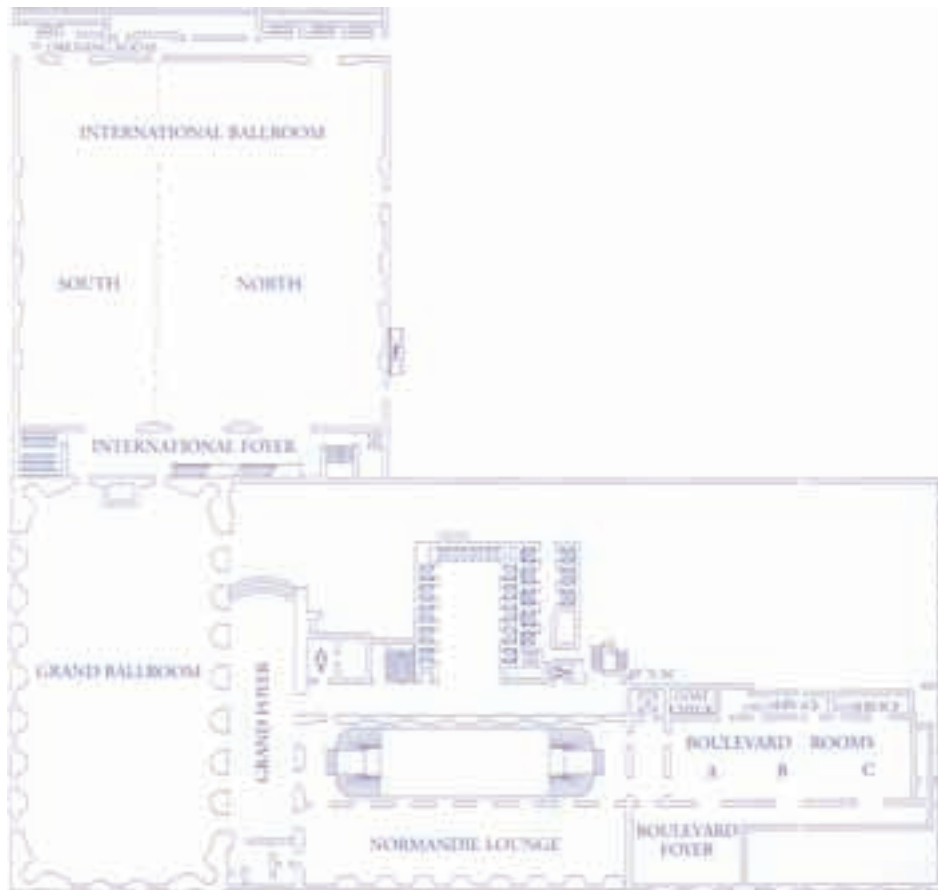


LOBBY LEVEL

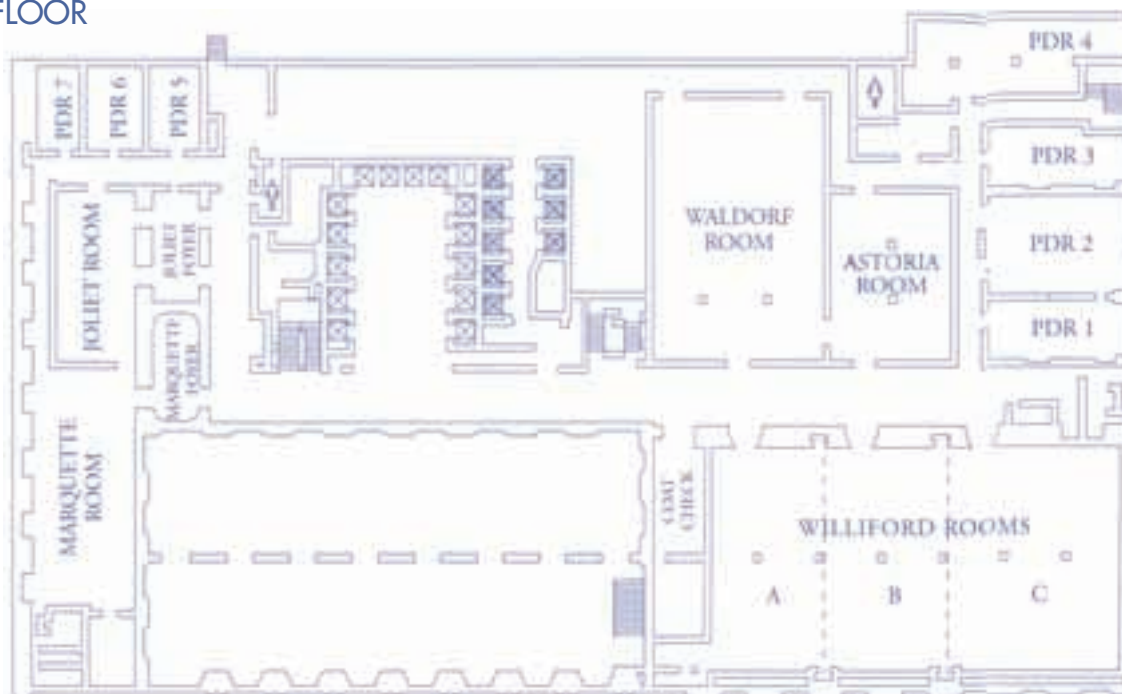


HILTON FLOOR PLAN

SECOND FLOOR



THIRD FLOOR



See what's taking shape



at Allergan booth #318

Allergan is proud to be a Gold Supporter of
The *Movement* Disorder Society's
12th International Congress of Parkinson's Disease
and Movement Disorders



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 will receive an additional copy with an upcoming MDS Journal issue.

ABSTRACTS-ON-CD-ROM

All abstracts published in the supplement to the MDS Journal are available by Abstracts-On-CD-ROM sponsored by MDS and supported by Medtronic, Inc. To obtain a copy, please visit the Medtronic Booth 213 and exchange the Medtronic Delegate bag insert.

CONTINUING MEDICAL EDUCATION

Please refer to page 33 for Continuing Medical Education information.

EVALUATIONS

Please take time to complete the evaluation forms provided for each session you attend. Your input and comments are essential in planning future educational programs for MDS.

When completed, evaluations may be returned to your meeting room attendants, the Speaker Ready Room (Astoria Room, Third Floor) or to the MDS Registration Desk.

INTERNET CAFÉ

Location: Northwest Exhibit Hall, Lower Level

Internet access is available to meeting attendees in the Northwest Exhibit Hall. Please limit your Internet use to 15 minutes to allow other attendees use of this service.

The Internet Café is supported by Pfizer Inc.

MDS EXHIBIT AND INFORMATION BOOTH

Location: Lower Level

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 on the Lower Level of the Hilton Chicago during the following hours:

Saturday, June 21	16:00 – 20:00
Sunday, June 22	9:00 – 19:30
Monday, June 23	7:00 – 18:00
Tuesday, June 24	7:00 – 18:00
Wednesday, June 25	7:00 – 18:00
Thursday, June 26	7:00 – 16:00

NO CAMERAS

Cameras are not permitted in any 12th International Congress educational sessions, exhibit hall or in the poster areas.

OPTIONAL TOURS DESK

Location: 8th Street Entrance, Lobby Level

Tours have been arranged by metroConnections.

Please visit the Tours Desk located near the 8th Street Entrance on the Lobby Level of the Hilton Chicago to collect your tickets. Additional tour tickets may be purchased at the desk, based on availability.

MetroConnections Tour Desk Hours:

Sunday, June 22, 2008	8:00 – 14:00
Monday, June 23, 2008	8:00 – 12:00

PRESS ROOM

Location: Private Dining Room #7

Members of the working media receive waived registration fees for the 12th International Congress. Journalists and writers should report to the Press Room with their credentials to register for the International Congress and wear their name badge for admittance into MDS sessions. The Press Room will be open during the following hours:

Sunday, June 22	8:00 – 17:00
Monday, June 23	8:00 – 17:00
Tuesday, June 24	8:00 – 17:00
Wednesday, June 25	8:00 – 17:00
Thursday, June 26	8:00 – 16:00

INTERNATIONAL CONGRESS INFORMATION

SCIENTIFIC SESSIONS

The 2008 Scientific Program incorporates Opening Symposia, Corporate Therapeutic Sessions, Plenary and Parallel Sessions, Teaching Courses, Video Sessions, How To Do It - Skills Workshops, Controversies and Guided Poster Tours.

Tickets are required for admission into all Parallel Sessions, Teaching Courses, Video Sessions and How To Do It - Skills Workshops. There is no additional fee for tickets to these sessions. Please check the Onsite Registration Desk for availability of these tickets.

ABSTRACT POSTER SESSIONS

Delegate feedback from past International Congresses has indicated great interest in Poster Sessions. All posters will be available for viewing from Tuesday, June 24 through Thursday, June 26. Poster Sessions with authors present are featured each day based upon the following schedule:

Poster Session 1

Posters: 1-438
Tuesday, June 24
Poster Viewing: 9:00 - 17:00
Authors Present: 12:30 - 14:30
Location: Southeast Exhibit Hall, Lower Level

Poster Session 2

Posters: 439-677
Wednesday, June 25
Poster Viewing: 9:00 - 17:00
Authors Present: 12:30 - 14:30
Location: Northeast Exhibit Hall, Lower Level

Poster Session 3

Posters: 678-1210
Thursday, June 26
Poster Viewing: 9:00 - 16:00
Authors Present: 12:30 - 14:30
Location: Southwest Exhibit Hall, Lower Level

GUIDED POSTER TOURS

Attendees may sign up for the Guided Poster Tours on Tuesday, June 24, 2008 from 7:30 to 17:00 at the MDS Booth located near Registration on the Lower Level of the Hilton Chicago. Space is limited; tours will be filled on a first-come, first-served basis.

The Guided Poster Tours will be led by members of the MDS faculty and the authors will be present to discuss the abstracts. There will be six Guided Poster Tours and each tour will feature abstracts on a specific topic.

There will be two tours per day from Tuesday, June 24, 2008 through Thursday, June 26, 2008 which will run simultaneously. Tours will meet each day at 12:15 at the MDS Booth located on the Lower Level of the Hilton Chicago.

TUESDAY, JUNE 24, 2008

12:30 – 14:00 Northeast Exhibit Hall, Lower Level
Guided Poster Tour 1 – Dystonia (Posters 475-494)
Tour Leaders: Kailash Bhatia and Marie Vidailhet
Guided Poster Tour 2 – Parkinson's disease: Clinical Trials (Posters 582-601)
Tour Leaders: Werner Poewe and Olivier Rascol

WEDNESDAY, JUNE 25, 2008

12:30 – 14:00 Southeast Exhibit Hall, Lower Level
Guided Poster Tour 3 – Genetics (Posters 83-102)
Tour Leaders: Christine Klein and *To be announced*
Guided Poster Tour 4 – Parkinson's disease: Cognition (Posters 257-276)
Tour Leaders: Bruno Dubois and *To be announced*

THURSDAY, JUNE 26, 2008

12:30 – 14:00 Southeast Exhibit Hall, Lower Level
Guided Poster Tour 5 – Surgical Therapy (Posters 315-334)
Tour Leaders: Michael Okun and Jerry Vitek
Guided Poster Tour 6 – Neuropharmacology (Posters 226-245)
Tour Leaders: David Standaert and *To be announced*

INTERNATIONAL CONGRESS INFORMATION

SHUTTLES

MDS will provide complimentary shuttles to all International Congress attendees and their guests. The shuttles will circulate every 30 minutes among the three hotels: the Hilton Chicago, the Holiday Inn Mart Plaza and the Sofitel Chicago Water Tower.

Shuttle Pickup Locations:

Hilton Chicago – Lobby Level, 8th Street Entrance
Holiday Inn Mart Plaza – Main Entrance, Orleans St.
Sofitel Chicago Water Tower – Main Entrance,
E. Chestnut St.

Shuttle Times:

Shuttles will run every 30 minutes based on the schedule below. Pickup/drop off times for the Hilton Chicago and the Sofitel Chicago Water Tower will be on each hour and half hour (:00 and :30), and at the Holiday Inn Mart Plaza at 15 minutes before and after each hour (:15 and :45).

Sunday, June 22:	9:00 – 24:00
Monday, June 23:	6:15 – 22:30
Tuesday, June 24:	6:15 – 20:00
Wednesday, June 25:	6:15 – 23:30
Thursday, June 26:	6:15 – 18:00

SPEAKER READY ROOM

Location: Astoria Room, Third Floor

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

Saturday, June 21	16:00 – 20:00
Sunday, June 22	7:00 – 17:00
Monday, June 23	7:00 – 17:00
Tuesday, June 24	7:00 – 17:00
Wednesday, June 25	7:00 – 17:00
Thursday, June 26	7:00 – 14:00



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CHICAGO MAP



*See us at the MDS
International Congress
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Booth 101*



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A silver zipper pull is shown against a background of a zipper. The zipper teeth are silver and run diagonally from the top right to the bottom left. The zipper pull is a trapezoidal shape with a circular hole on the left side. On the pull, there is a logo consisting of a stylized sunburst with five rays above the word "AZILECT" in a blue, serif font. Below "AZILECT" is the text "(rasagiline tablets)" in a smaller, blue, sans-serif font.

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(rasagiline tablets)

SOCIAL EVENTS

OPENING CEREMONY AND WELCOME RECEPTION

SUNDAY, JUNE 22, 2008

Grand Ballroom (Opening Ceremony)
International Ballroom (Welcome Reception),
Second Floor, Hilton Chicago

Opening Ceremony: 19:30-20:00
Welcome Reception: 20:00-24:00

All International Congress attendees are warmly invited to meet friends and colleagues during the traditional International Congress Opening Ceremony on Sunday evening, June 22, at the Hilton Chicago. Following the Opening Ceremony, there will be a Taste of Chicago themed Welcome Reception with a variety of food and entertainment acts. These events are open to all registered delegates. Guests are welcome to purchase a Social Event pass that will allow them to accompany a registered delegate to the Opening Ceremony and Welcome Reception. Please check at the Registration Desk for availability.

This event is supported by Boehringer Ingelheim Pharmaceuticals, Inc.

VIDEO OLYMPICS

WEDNESDAY, JUNE 25, 2008

Reception with hors d'oeuvres and drinks, Grand Ballroom:
19:00 – 20:00
Video Olympics, International Ballroom: 20:00- 23:00

Please join Masters of Ceremony Anthony Lang and Kapil Sethi on Wednesday evening, June 25, as they host a world-renowned panel of Movement Disorders experts in guiding participants through unique Movement Disorder cases. The cases will be presented by representatives from Movement Disorder Centers around the world, and experts will consider each case and engage the audience in discussion. The final diagnosis will then be provided by the case presenter. The goal of this session is for attendees to learn from a series of unusual, very interesting patients and see how senior experts approach these types of challenging cases.

The experts are:

Joseph Jankovic, *Houston, TX, USA*
Philip Thompson, *Adelaide, Australia*
Werner Poewe, *Innsbruck, Austria*
Niall Quinn, *London, United Kingdom*
Eduardo Tolosa, *Barcelona, Spain*

This social event is open to all registered delegates. Guests are welcome to purchase a Social Event Pass that will allow them to accompany a registered delegate to the Video Olympics. Please check at the registration desk for availability.

This event is supported by UCB, Inc.

SATELLITE SYMPOSIA

MENTORSHIP LUNCH SYMPOSIUM

“Pearls from junior and senior mentors for new investigators: Opportunities and obstacles”

Panel discussion

TUESDAY, JUNE 24, 2008
12:30-14:00

Marquette Room, Third Floor

This symposium is designed for fellows and new investigators to address issues related to the role of mentorship in establishing a research career. The panel discussants are international, and have served as mentors for new investigators across the globe. The panel will provide brief comments prior to open audience participation.

BRITISH MOVEMENT DISORDERS GROUP (BRITMODIS)

TUESDAY, JUNE 24, 2008

12:30 - 14:30
Williford C, Third Floor

AWARDS INFORMATION

HONORARY MEMBERSHIP AWARDS

The Honorary Membership Awards recognize individuals who have made extraordinary contributions to the field of Movement Disorders or otherwise to The *Movement Disorder Society*.

Sunday, June 22, 2008

Opening Ceremony

19:30 to 20:30

Location: Grand Ballroom, Second Floor



Alim L. Benabid,
MD PhD
Grenoble, France



Mahlon R. DeLong, MD
Atlanta, GA,
USA

PRESIDENT'S DISTINGUISHED SERVICE AWARD

The President's Distinguished Service Award is given in recognition of long and distinguished service to The *Movement Disorder Society*. The recipient may only receive this award once in their lifetime.

Sunday, June 22, 2008

Opening Ceremony

19:30 to 20:30

Location: Grand Ballroom

STANLEY FAHN AWARD LECTURE

Wednesday, June 25, 2008 *as part of the Presidential Lecture Plenary Session*

International Ballroom, Second Floor
8:00 – 8:30

Dystonia: Found in Translation

Stanley Fahn Lecturer
– Susan B. Bressman, MD



Susan Bressman, M.D., is the Chairman of the Department of Neurology at Beth Israel Medical Center in New York City, and Professor of Neurology and the Vice Chairman of the Department of Neurology at Albert Einstein College of Medicine.

Dr. Bressman attended Columbia University's College of Physicians and Surgeons and received her postgraduate

training in neurology at the Columbia Presbyterian Medical Center. After residency she was a Movement Disorders Fellow under Dr. Stanley Fahn and remained at Columbia until 1997, where she developed a genetics program in movement disorders. Her research has focused on identifying genes for dystonia and other movement disorders and characterizing their phenotypes.

Dr. Bressman serves on the scientific advisory boards of the Michael J. Fox Foundation for Parkinson's Research and Bachmann-Strauss Foundation for Dystonia, and Parkinson's Research. She is also a Director of the American Academy of Neurology, and the President of WE MOVE (Worldwide Education and Awareness of Movement Disorders). Dr. Bressman has more than 100 published articles in peer-reviewed journals and is the co-editor of two books.

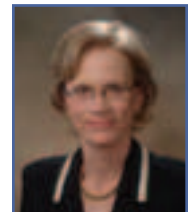
C. DAVID MARSDEN AWARD LECTURE

Wednesday, June 25, 2008 *as part of the Presidential Lecture Plenary Session*

International Ballroom, Second Floor
9:30 – 10:00

The Basal Ganglia: Their Mysterious Functions Revisited

C. David Marsden Lecturer
– Ann M. Graybiel, PhD



Ann M. Graybiel is the Walter A. Rosenblith Professor of Neuroscience and Investigator, McGovern Institute for Brain Research, at M.I.T. She was trained at Harvard and MIT and received her PhD from MIT. Research in the Graybiel Laboratory is focused on regions of the forebrain that influence movement, mood and motivation: the basal ganglia and neural pathways interconnecting the basal ganglia with the cerebral cortex. Dr. Graybiel and her group use methods ranging from multi-electrode recordings in awake behaving animals to genetic engineering to analyze these neural pathways. Central to many of these studies is work on brain mechanisms underlying habit formation and repetitive behaviors and understanding how such mechanisms can become dysfunctional in neurologic and neuropsychiatric disorders such as Parkinson's disease, Huntington's disease, obsessive-compulsive disorder, and in addictive states. On the basis of her work, Graybiel was elected to the National Academy of Sciences of the USA in 1988, the American Academy of Arts and Sciences in 1991, and the Institute of Medicine of the USA in 1994. She was awarded the National Medal of Science of the USA in 2002.

AWARDS INFORMATION

JUNIOR AWARDS

Two Junior Awards recipients have been selected based on their significant contribution to clinical and basic science research in the field of Movement Disorders. One award will be presented for excellence in clinical research, and another for excellence in basic research.

Wednesday, June 25, 2008

8:30 to 9:00

Location: International Ballroom, Second Floor

Chairs: Anthony E. Lang and Serge Przedborski

Clinical Research

Luke A. Massey

London, United Kingdom

Determining the anatomy of the subthalamic nucleus and substantia nigra on MRI using 9.4Tesla

L. Massey, M. Miranda, J. Thornton, O. Al-Helli, H. Parkes, P.-W. So, L. Mancini, A. Lees, T. Revesz, T. Yousry

Objective: Using high field MRI we aim to describe the anatomy of two key nuclei in Parkinsonism.

Background: In PD electrical stimulation of the STN is now part of routine practice, however the STN is not clearly identified on conventional MRI and thus has only a limited role in stereotactic surgical targeting. The STN is typically atrophied in PSP but this is not seen using conventional MRI techniques. The anatomy of the SN on conventional imaging is also controversial: there are few reports of direct comparison with pathological material reported in the literature to date. We have acquired high-field imaging at 9.4Tesla of post-mortem brain and compared this directly with pathological sections.

Methods: Brain tissue (4 controls, 3 PSP, 2 PD) were obtained from the Queen Square Brain Bank. Formalin-fixed tissue was cut to provide brainstem blocks which were then imaged with a multimodal MRI protocol including T1, T2 and T2* relaxometry and diffusion tensor imaging, using a Varian Inova 9.4Tesla system. Specimens were subsequently embedded in paraffin and stained with cresyl violet (for cell bodies) and luxol fast blue (for myelin) for conventional histological examination.

Results: T2-weighted images with an in-plane spatial resolution of 50 microns were obtained, with detailed demonstration of anatomy comparable to that from macroscopic pathology (see figure). Comparison of

9.4Tesla MRI and histological sections enabled clear identification of the boundaries of the STN, which are not apparent on clinical 1.5Tesla MRI. In PSP atrophy of the STN is seen and preliminary MRI measurements (see table) show shortening of T2* in the STN in PSP, and changes in ADC and FA. The anatomy of the SN is also demonstrated by this method. However, the histological correlate of a hyperintense band within the region of the SN on T2-weighted imaging is yet to be determined.[figure1]

Mean (range) MR values

Type	T1 (ms)	T2 (ms)	T2* (ms)	ADC (* 10-10mm ² /s)	FA
Control	786 (384)	15.75 (3)	7.25 (8)	3.85 (0.34)	0.77 (0.35)
PSP	1076 (477)	14 (3)	4.3 (4)	5.14 (4.06)	0.67 (0.30)
PD	917 (252)	14 (0)	9 (8)	4.82(*)	0.91 (*)

* DTI only available in 1 PD case

Conclusions: At 9.4T high resolution images enable accurate descriptions of small brainstem nuclei including the STN and SN. Direct comparison with histology has enabled more accurate definition of the MRI anatomy and preliminary anatomically specific MRI measurements in post mortem tissue are presented. Further work is needed particularly in the SN to delineate the anatomy more clearly.

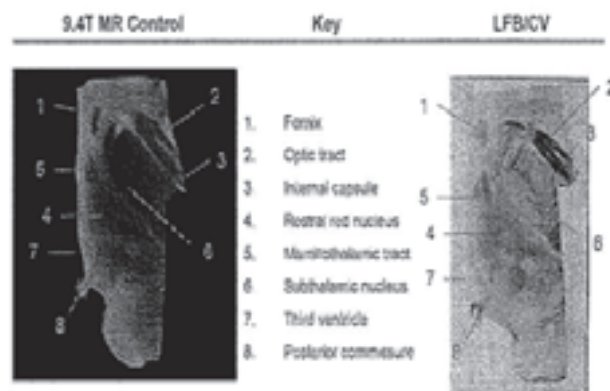


FIG. 1 (180).

AWARDS INFORMATION

Basic Science

Binith Cheeran

London, United Kingdom

Stimulation genomics: Identifying functional polymorphisms modulating LTP and LTD in human cerebral cortex and implications for levodopa induced dyskinesia in Parkinson's disease (PD)

B.J. Cheeran, P. Talelli, F. Mori, G. Koch, S.A. Schneider, A. Suppa, M. Edwards, H. Houlden, R. Greenwood, J.C. Rothwell, K.P. Bhatia (London, United Kingdom)

Objective: To pioneer a novel approach to screen common polymorphisms in key molecular regulators of LTP/LTD induction for a functional role in altered neuroplasticity in the human cortex.

Background: Disordered control of plasticity and homeostatic plasticity have been postulated to underlie susceptibility to Levo-dopa induced dyskinesia in Parkinson's Disease and dystonia. Building on work performed in animal models and brain slices, a number of noninvasive transcranial stimulation techniques are now available to probe plasticity in the human cortex, enabling us to rapidly screen candidate polymorphisms for a functional role in human neuroplasticity.

Methods: We investigated the induction and control of LTP/LTD-like changes in the primary motor cortex of healthy human volunteers with Brain Derived Neurotrophic Factor (BDNF) Val66Met polymorphism using a range of techniques (inhibitory and excitatory Theta Burst Stimulation, Paired Associative Stimulation and Transcranial Direct Current Stimulation (TDCS) preconditioned 1 Hz stimulation).

Results: We report for the first time that the induction and control of LTP/LTD-like changes in the primary motor cortex of healthy human volunteers is significantly influenced by a common polymorphism (BDNF Val66Met) in the BDNF gene.

Conclusions: The results suggest that the reduced plasticity and homeostatic plasticity of subjects who carry a BDNF met allele could be an important contributing factor to the occurrence of symptoms in conditions thought to be caused by abnormal neuroplasticity such as dystonia and levo-dopa induced dyskinesia in Parkinson's disease. Also several previous studies using rTMS as a therapeutic intervention (e.g. in dyskinesia in PD) may benefit from a re-analysis of data based on this common functional polymorphism. We conclude that, like Imaging Genomics, TMS paradigms can offer a unique insight into the physiological consequences of functional human polymorphisms.

The poster features a logo of a stylized human figure with arms raised inside a circle, set against a background of cherry blossoms and the US Capitol building. The text on the poster includes the title of the conference, the dates and location, and the names of the organizing committee members.

The Movement Disorder Society

• 2nd INTERNATIONAL CONFERENCE •

Psychogenic Movement Disorders

and Other Conversion Disorders

APRIL 2-4, 2009 • WASHINGTON, DC

Organizing committee:

Mark Hallett, Chair
Stanley Fahn
Joseph Jankovic
Anthony Lang
C. Robert Manning
Peter Halligan
Valerie Voon

Visit www.movementdisorders.org
for more information!

AWARDS INFORMATION

2008 TRAVEL GRANTS

Annu Aggarwal <i>Mumbai, India</i>	Ana Djarmati <i>Lübeck, Germany</i>	Helen Ling <i>Bangkok, Thailand</i>
Zeshan Ahmed <i>Jacksonville, FL, USA</i>	Jon Doan <i>Lethbridge, AB, Canada</i>	Praween Lolekha <i>Bangkok, Thailand</i>
Imtiaz Ahmed <i>London, United Kingdom</i>	Andre Felicio <i>Sao Paulo, Brazil</i>	Ignacio Obeso <i>London, United Kingdom</i>
Roy Alcalay <i>New York, NY, USA</i>	Erin Foster <i>St. Louis, MO, USA</i>	Jing Pan <i>Shanghai, China</i>
Phalguni Alladi <i>Bangalore, India</i>	Xiang Gao <i>Boston, MA, USA</i>	Santiago Perez-Lloret <i>Buenos Aires, Argentina</i>
Chrystalina Antoniadis <i>Cambridge, United Kingdom</i>	Criscey Go <i>Manila, Philippines</i>	David Peterson <i>La Jolla, CA, USA</i>
Benedicte Ballanger <i>Toronto, ON, Canada</i>	Justus Groen <i>Amsterdam, The Netherlands</i>	Ilse Pienaar <i>Cape Town, South Africa</i>
Cynthia Bedeschi <i>Sao Paulo, Brazil</i>	Petra Havrankova <i>Prague, Czech Republic</i>	Kathleen Poston <i>New York, NY, USA</i>
Daniela Besong-Agbo <i>Marburg, Germany</i>	Anna Hotter <i>Innsbruck, Austria</i>	Cauchy Pradhan <i>Bangalore, India</i>
Tomas Bjorklund <i>Lund, Sweden</i>	Yue Huang <i>Sydney, Australia</i>	Mona Ragothaman <i>Bangalore, India</i>
Matteo Bologna <i>Rome, Italy</i>	Natalie Ives <i>Birmingham, United Kingdom</i>	Antonio Rodrigues <i>Marburg, Germany</i>

Norbert Brüggemann <i>Lübeck, Germany</i>	Tom Johnston <i>Toronto, ON, Canada</i>	Ola Sader-Mazbar <i>Nazareth, Israel</i>
Meghan Campbell <i>St. Louis, MO, USA</i>	Natlada Kanjanasut <i>Chulalongkorn, Thailand</i>	Gurdal Sahin <i>Lund, Sweden</i>
Tamine Capato <i>Sao Paulo, Brazil</i>	Siddharth Kaul <i>Springfield, IL, USA</i>	Susanne Schneider <i>London, United Kingdom</i>
Robert Caslake <i>Aberdeen, Scotland, UK</i>	Renju Kuriakose <i>Toronto, ON, Canada</i>	Lauren Schrock <i>San Francisco, CA, USA</i>
Binith Cheeran <i>Manchester, United Kingdom</i>	Elli Kyrtzi <i>Athens, Greece</i>	Saima Siddiqui <i>New Delhi, India</i>
Yun Ju (Christine) Song <i>Randwick, Australia</i>	Tserensodnom Bayasgalan <i>Ulaanbaatar, Mongolia</i>	Tereza Vogiatzi <i>Athens, Greece</i>
Vimal Stanislaus <i>Lidcombe, Australia</i>	Sandra Van Der Salm <i>Amsterdam, The Netherlands</i>	Harrison Walker <i>Birmingham, AL, USA</i>
Yen Tai <i>London, United Kingdom</i>	Xavier Vasques <i>St. Andre DeSangonis, France</i>	Dakshitha Wickramasinghe <i>Borella, Sri Lanka</i>
Kanya Temkiatvises <i>Siriraj, Thailand</i>	Naomi Visanji <i>Toronto, ON, Canada</i>	Ravi Yadav <i>New Delhi, India</i>

CME INFORMATION

PURPOSE

The purpose of the MDS International Congress is to offer a forum for clinical and basic discussion on a variety of Movement Disorder topics, including presentations of current research and available treatments.

LEARNING OBJECTIVES

Through state-of-the-art lectures, hot topic reviews, controversy debates, Teaching Courses, How To Do It - Skills Workshops and Video Sessions, participants will 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.

CONTINUING MEDICAL EDUCATION

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

CREDIT DESIGNATION

The *Movement* Disorder Society designates this educational activity for a maximum of 34 *AMA PRA Category 1 Credits*™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

TARGET AUDIENCE

The target audience of the 12th International Congress of Parkinson's Disease and Movement Disorders includes clinicians, researchers, post-doctoral fellows, medical residents, medical students and other healthcare professionals with an interest in the current research and approaches for the diagnosis and treatment of Movement Disorders.

FINANCIAL DISCLOSURE

It is the policy of The *Movement* Disorder Society (MDS) to ensure balance, independence, objectivity, and scientific rigor in all MDS sponsored educational activities. All persons in control of content in any MDS sponsored activity are required to disclose to MDS and the activity audience all financial relationships with any commercial interest. Such persons include, but are not limited to: planning committee members, faculty, medical writers, joint sponsor staff (when applicable), co-sponsor staff (when applicable) and MDS staff.

Relationships are defined as financial relationships in any amount occurring within the past 12 months from the signing date of the attestation. This pertains to relationships with pharmaceutical companies, biomedical device manufacturers, or other corporations whose products or services are related to the field of Movement Disorders. This disclosure will be provided to all participants prior to the beginning of the CME activity. Individuals must inform MDS of any changes in financial relationships that occur between the signing date of the attestation and the date of the activity. For an individual with no financial relationships to disclose, the learners must be informed that no financial relationships exist.

The intent of this policy is not to prevent a speaker with a conflict of interest from making a presentation. It is merely intended that any potential conflict should be identified and resolved prior to the activity, so that there is no commercial bias present in the presentation. Failure by any faculty or planning committee member to disclose all such relationships will result in their inability to participate in the CME activity.

Faculty financial disclosure information will be provided to participants onsite in Chicago.

CLAIMING CME CREDIT

Physicians may claim their CME Certificates from their home or office upon the completion of the MDS 12th International Congress. Simply visit www.movementdisorders.org/congress/congress08/cme after July 1 and use your Reference Number (found in the upper right of your registration confirmation form) to log in and claim your credits. You will be able to print or save a PDF of your credit award from your own computer.



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12TH INTERNATIONAL CONGRESS PROGRAM-AT-A-GLANCE

	June 22 (Sunday)	June 23 (Monday)	June 24 (Tuesday)	June 25 (Wednesday)	June 26 (Thursday)
7:00		Committee meetings			
8:00		Opening Symposia 8:00-13:00	Plenary Session 1	Presidential Lectures	Plenary Session 5
9:00	Committee Meetings				
10:00			Break		
11:00	Corporate Therapeutic Session 10:30-12:00		Plenary Session 2	Plenary Session 4	Controversies
12:00	Lunch		Lunch and Posters	Lunch and Posters	MDS Business Meeting <i>(All delegates are encouraged to attend)</i>
13:00	Opening Symposia 13:00-19:00	Lunch	AOS General Assembly Meeting 13:30-14:15 Lunch and Posters	Parallel Sessions	Lunch and Posters
14:00		Opening Symposia 13:30-16:00			Parallel Sessions
15:00			Break	Break	
16:00		Corporate Therapeutic Sessions 16:30-20:30	How To Do It - Skills Workshops and Video Sessions	How To Do It - Skills Workshops and Video Sessions	
17:00					
18:00					
19:00				Video Olympics 19:00-23:00	
20:00	Opening Ceremony and Welcome Reception 19:30-24:00				
21:00		Dinner 20:30			

SESSION DEFINITIONS

Controversies:

This Plenary Session is designed to bring together a large audience by incorporating all International Congress attendees. Content is prepared to stimulate interest and debate among a panel of experts. Views from several angles will be addressed as discussion of pre-selected “hot” topics will be open for deliberation among the panelists.

Corporate Therapeutic Sessions:

These company-based informational sessions will provide attendees with non-CME educational opportunities to learn the latest in therapeutics.

How To Do It – Skills Workshops:

These clinic-based training sessions provide an educational illustration of clinical techniques and treatment procedures through demonstrations utilizing patient videotapes and proper equipment to further develop practitioners' skills and knowledge of the treatment of Movement Disorders.

Opening Symposia:

These sessions will provide the latest information regarding the scientific and clinical evidence supporting treatment options for Parkinson's disease and other Movement Disorders. Planned by a subcommittee of the Congress Scientific Program Committee, this series is supported through unrestricted educational grants from industry supporters and offer Continuing Medical Education credits.

Parallel Sessions:

These concurrent sessions are designed to provide an in-depth report of the latest research findings, state-of-the-art treatment options, as well as involve a discussion of future strategies. Sessions will have evidence-based components and incorporate the “hot” issues in Parkinson's disease and other Movement Disorders.

Plenary Sessions:

Designed to incorporate all International Congress attendees, these sessions will provide a broad overview of the latest clinical and basic science research findings and state-of-the-art information.

Poster Sessions:

Poster Sessions give each delegate an opportunity to view their colleagues' posters on the most current research in the field of Movement Disorders. Authors will be present for two hours each day to explain their work and answer questions. Guided Poster Tours will also run each day to give a small group of delegates an opportunity to hear discussion on a select group of abstracts in each of six sub-categories. *Please see page 23 for more information on the Guided Poster Tours.*

Teaching Courses:

These sessions are designed as educational programs to provide up-to-date information focused on a single topic. The sessions highlight both clinical and basic sciences of topics of relevance to Movement Disorders specialists. The sessions are unique in providing a syllabus that includes a review of the topic and the presentation slides. In addition, these programs provide ample time for questions and a discussion period at the conclusion of the presentations.

Video Sessions:

Designed to provide a broad overview of related Movement Disorders, the video sessions will focus on the phenomenology covering the many different kinds of Movement Disorders affecting the population today.



= Ticket required for entry. Please check the Registration Desk for ticket availability.

DAILY SCHEDULE SUNDAY, JUNE 22, 2008

Corporate Therapeutic Sessions:

These company-based information sessions will provide attendees with non-CME educational opportunities to learn the latest in therapeutics.

Teva Neurosciences

Location: Grand Ballroom, Second Floor
10:30-12:00

Issues and Controversies in Parkinson's disease

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

Search for Diagnostic Tools and pre-motor diagnosis of Parkinson's disease

Matthew Stern
Philadelphia, PA, USA

Trial Design and changing strategies

Karl Kiebertz
Rochester, NY, USA

Review of Azilect early and adjunct

Oliver Rascol
Toulouse, France

Following this session, lunch will be provided in the Grand Ballroom Foyer compliments of Teva Neurosciences.

2010 Opening Symposium

Issues in the diagnosis and early treatment of Parkinson's disease

Location: Continental Ballroom, Lobby Level
13:00 to 16:00

Chairs: Anthony E. Lang
Toronto, Canada

Kapil D. Sethi
Augusta, GA, USA

13:00 Diagnosing Parkinson's disease - The role of functional neuroimaging

David J. Brooks
London, United Kingdom

13:30 Two decades of neuroprotection - Lessons learned and a peek into the future

Ira Shoulson
Rochester, NY, USA

14:00 When to start symptomatic treatment for Parkinson's disease? A reappraisal

Anthony H.V. Schapira
London, United Kingdom

15:00 How and what to start? Tailoring treatments for individual patients - Art or science?

Olivier Rascol
Toulouse, France

2010 Opening Symposium - continued

At the conclusion of this session, participants should be better able to:

1. Describe the role of functional imaging in the diagnosis of Parkinson's disease
2. Explain the status of neuroprotection in PD
3. Indicate the various factors that need to be considered in deciding when and how to initiate therapy in PD

2011 Opening Symposium

Managing the late stage Parkinson's disease patient

Location: Continental Ballroom, Lobby Level
16:00 to 19:00

Chairs: Eduardo Tolosa
Barcelona, Spain

Ray L. Watts
Birmingham, AL, USA

16:00 Motor fluctuations and dyskinesias - Magnitude of the problem and underlying mechanisms

José A. Obeso
Pamplona, Spain

16:30 Managing motor complications in the clinic

Matthew B. Stern
Philadelphia, PA, USA

17:00 Daytime sleepiness and nocturnal problems in Parkinson's disease

William Ondo
Houston, TX, USA

17:30 Neuropsychiatric manifestations of advanced Parkinson's disease - The range

Hubert Henry Fernandez
Gainesville, FL, USA

18:30 Management of cognitive problems and dementia in Parkinson's disease

Murat Emre
Istanbul, Turkey

At the conclusion of this session, participants should be better able to:

1. Recognize how to manage the most important motor complications associated to levodopa treatment
2. Identify how to manage the sleep disturbances and nocturnal problems most commonly occurring in Parkinson disease
3. Describe the pathophysiology and management of the neuropsychiatric and cognitive problems occurring in advanced Parkinson disease

DAILY SCHEDULE SUNDAY, JUNE 22, 2008 AND MONDAY, JUNE 23, 2008

Opening Ceremony

Location: Grand Ballroom, Second Floor
19:30-20:00

Welcome Reception

Location: International Ballroom, Second Floor
20:00-2400

DAILY SCHEDULE MONDAY, JUNE 23, 2008

3012 Opening Symposium

Infusion therapies and surgery for Parkinson's disease

Location: International Ballroom, Second Floor
8:00 to 11:00

Chairs: Andrew J. Lees
London, United Kingdom

José A. Obeso
Pamplona, Spain

8:00 Continuous dopaminergic stimulation - Where are we?
Fabrizio Stocchi
Roma, Italy

8:30 Apomorphine infusion and intrajejunal levodopa
Angelo Antonini
Monza, Italy

9:00 Deep brain stimulation - Exploring new targets
Robert E. Gross
Atlanta, GA, USA

10:00 An update on cell-based and gene therapy in Parkinson's disease
C. Warren Olanow
New York, NY, USA

3013 Opening Symposium

The twists and turns of dystonia

Location: Grand Ballroom, Second Floor
11:00 to 13:00

Chairs: Susan B. Bressman
New York, NY, USA

Daniel Tarsy
Boston, MA, USA

11:00 Dystonia classification, differential diagnosis and genetics
Alberto Albanese
Milano, Italy

11:45 AM Treatment of dystonia including botulinum toxin treatment and surgery - Impact on quality of life
Giovanni Abbruzzese
Genova, Italy

3013 Opening Symposium - continued

At the conclusion of this session, participants should be better able to:

1. Explain the classification of dystonia
2. Recognize the more common genetic types of dystonia
3. Discuss the treatment of the different types of dystonia

3014 Opening Symposium

Restless legs syndrome and periodic limb movements in sleep: Diagnosis, co-morbidities, basic science and treatment

Location: International Ballroom, Second Floor
13:30 to 16:00

Chairs: Cynthia L. Comella
Chicago, IL, USA

Arthur S. Walters
Highland Park, NJ, USA

13:30 Diagnosis and co-morbidities
Arthur S. Walters
Highland Park, NJ, USA

14:00 New genetic discoveries, role of iron and other basic science
David B. Rye
Atlanta, GA, USA

14:30 Treatment
Claudia M. Trenkwalder
Kassel, Germany

15:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Recognize the diagnostic features of restless legs syndrome (RLS), its associated co-morbidities, and how to distinguish it from RLS mimics
2. Explain the new genetic discoveries in RLS, the role of iron deficiency in pathogenesis and other basic science discoveries relative to RLS.
3. Discuss the treatment options for RLS

Corporate Therapeutic Sessions:

These company-based information sessions will provide attendees with non-CME educational opportunities to learn the latest in therapeutics.

EMD Serono Inc.

Location: Grand Ballroom, Second Floor
16:30 - 17:30

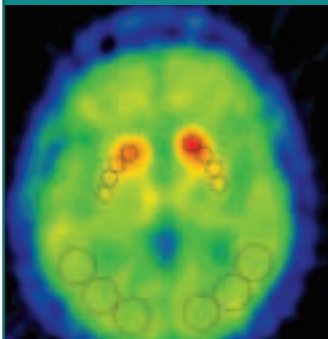
Behind the mask of Parkinson's disease

Chair: Paolo Barone
Napoli, Italy

Chair: Anthony H.V. Schapira
London, United Kingdom



XVIII WFN
World Congress on
*Parkinson's Disease
and Other Movement Disorders*



Miami Beach, FL, USA, December 13-16, 2009

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DAILY SCHEDULE MONDAY, JUNE 23, 2008

Corporate Therapeutic Sessions: - continued

Is it time to re-think Parkinson's disease?

Anthony H.V. Schapira
London, United Kingdom

The meaning of cognitive impairment and related disorders
in the understanding of Parkinson's disease progression

Paolo Barone
Napoli, Italy

Assessing drug effects in Parkinson's disease: a multifaceted
approach beyond UPDRS

Jaime Kulisevsky
Barcelona, Spain

Solvay Pharmaceuticals

Location: Continental Ballroom, Lobby Level
16:30-17:30

**Continuous intra-duodenal infusion of levodopa/
carbidopa in the treatment of advanced
Parkinson's disease**

Chair: Jens Volkmann
Kiel, Germany

Current concepts on the role of dopamine in the normal
basal ganglia: implications for Parkinson's disease therapy

C. Warren Olanow
New York, NY, USA

Duodenal levodopa infusion: from theory to clinical practice

Angelo Antonini
Monza, Italy

Break Appetizers and drinks

Location: Normandie Lounge, Second Floor and
Continental Ballroom Foyer, Lobby Level
17:30 – 18:00

Boehringer Ingelheim Pharmaceuticals Inc.

Location: Grand Ballroom, Second Floor
18:00-19:00

**The Continuum of Parkinson's disease Treatment
and Effect on Activities of Daily Living**

Chair: Matthew B. Stern
Philadelphia, PA, USA

Current Concepts in Early Treatment of Parkinson's Disease

Anthony H.V. Schapira
London, United Kingdom

Treatment of Advanced Parkinson's Disease

Werner Poewe
Innsbruck, Austria

The Evolution of Disability in Parkinson's Disease

Lisa M. Shulman
Baltimore, MD, USA

Corporate Therapeutic Sessions: - continued

Vernalis Pharmaceuticals, Inc.

Location: Continental Ballroom, Lobby Level
18:00-19:00

Clinical and Practical Considerations in the Acute
Management of "Off" Episodes in Advanced Parkinson's
Disease

Chairman: Kapil Sethi
Augusta, GA, USA

Clinical Considerations in the acute management of "off"
episodes

Stewart A. Factor
Augusta, GA, USA

Practical Considerations in the acute management of "off"
episodes

William Ondo
Houston, TX, USA

Break Appetizers and drinks

Location: Normandie Lounge, Second Floor and
Continental Ballroom Foyer, Lobby Level
19:00 – 19:30

GlaxoSmithKline

Location: Continental Ballroom, Lobby Level
19:30-20:30

**Advances in the Management and Treatment of
Parkinson's Disease**

Chair: Ray Watts
Birmingham, AL, USA

Lecture - Current Trends and Medical Developments

Panel Discussions - Patient Case Studies

Rajesh Pahwa
Kansas City, KS, USA

Fabrizio Stocchi
Roma, Italy

Mark Stacy
Durham, NC, USA

Bonnie Hersh
Chestnut Hill, MA, USA

Dinner

Location: International Ballroom, Second Floor
20:30



= Ticket required for entry. Please check the
Registration Desk for ticket availability.

DAILY SCHEDULE TUESDAY, JUNE 24, 2008**4101 Plenary Session I****Neuronal death in Parkinson's disease: How, why when and where?**

Location: International Ballroom, Second Floor

8:00 to 10:00Chairs: Etienne C. Hirsch
*Paris, France*Yoshikuni Mizuno
*Tokyo, Japan***8:00 The molecular underpinning of the selective neuronal vulnerability in Parkinson's disease**D. James Surmeier
*Chicago, IL, USA***8:30 Value of genetic models in understanding the cause and mechanisms of Parkinson's disease**Ted M. Dawson
*Baltimore, MD, USA***9:00 Lesson from basic science to devise therapeutic strategies for Parkinson's disease**Jonathan M. Brotchie
*Toronto, ON, Canada***9:30 Panel discussion**

At the conclusion of this session, participants should be better able to:

1. List the mechanisms underlying differential neuronal vulnerability in Parkinson's disease (PD)
2. Explain the interest of genetic analysis for the understanding of cell death mechanisms in PD
3. Define the strategies for the development of neuroprotective drugs

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Visit www.movementdisorders.org/congress/congress08/cme after July 1 to obtain your CME Certificate for the MDS 12th International Congress.

For detailed information, please see the back inside cover of this program.

4102 Plenary Session II**Cognitive impairment in Parkinson's disease**

Location: International Ballroom, Second Floor

10:30 to 12:30Chairs: Murat Emre
*Istanbul, Turkey*Christopher G. Goetz
*Chicago, IL, USA***10:30 Clinical spectrum of cognitive impairment in Parkinson's disease**Bruno Dubois
*Paris, France***11:00 Anatomical and molecular pathology underlying the cognitive impairment in Parkinson's disease**Glenda M. Halliday
*Randwick, Australia***11:30 Imaging in cognitive impairment: Lessons from other forms of dementia**Nick Fox
*London, United Kingdom***12:00 Panel discussion**

At the conclusion of this session, participants should be better able to:

1. Discuss the clinical characteristics of cognitive impairment in PD, assessment methods and current operative criteria for PD-dementia
2. Define the anatomical structures, pathological features, and biochemical deficits associated with cognitive impairment in PD
3. Recognize the role of neuroimaging techniques in understanding structural/functional alterations that underlie cognitive decline in PD and their contribution to diagnosis

Poster Presentations**Poster Session 1**

Location: Southeast Exhibit Hall, Lower Level

Poster Viewing: 9:00 to 17:00**Authors Present: 12:30 to 14:30****Poster Numbers: 1-438****Guided Poster Tours****Guided Poster Tour 1 – Dystonia (475-494)**

Tour Leaders: Kailash Bhatia and Marie Vidailhet

Guided Poster Tour 2 – Parkinson's disease: Clinical Trials (Posters 582-601)

Tour Leaders: Werner Poewe and Olivier Rascol

Location: Northeast Exhibit Hall, Lower Level

Time: 12:30 to 14:00

(Attendees will meet at 12:15 at the MDS Booth, Lower Level)
Please refer to page 70 for abstract details.

DAILY SCHEDULE TUESDAY, JUNE 24, 2008

AOS General Assembly Meeting

Location: Willford Room, Third Floor
All delegates from Asia and Oceania are encouraged to attend.
13:30

4201 Parallel Session 

Parkinson's disease: Epidemiological issues

Location: Grand Ballroom, Second Floor
14:30 to 16:30

Chairs: G. Webster Ross
Honolulu, HI, USA
Caroline M. Tanner
Sunnyvale, CA, USA

14:30 Parkinson's disease risk factors - An overview
Alexis Elbaz
Paris, France

15:00 Methodologic issues
Beate Ritz
Los Angeles, CA, USA

15:30 Future directions: PD RFQ, MDS Epidemiology Task Force, multicenter risk factor studies
Caroline M. Tanner
Sunnyvale, CA, USA

16:00 Panel discussion
At the conclusion of this session, participants should be better able to:

1. Explain the current status of epidemiological research on PD etiology, progression and complications of therapy
2. Describe methodological approaches and measurement tools for ascertainment and risk factor investigation in PD epidemiological research
3. Recognize the role of collaborative research

4202 Teaching Course 

Dysautonomia in Parkinson's disease: Spectrum, evaluation and treatment

Location: Continental A, Lobby Level
14:30 to 16:30

Chairs: Carlo Colosimo
Rome, Italy
Christopher Mathias
London, United Kingdom

14:30 Gastrointestinal dysfunction
Ronald F. Pfeiffer
Memphis, TN, USA

4202 Teaching Course - continued 

15:00 Genito-urinary dysfunction
Ryuji Sakakibara
Chiba, Japan

15:30 Cardiovascular dysfunction
Christopher Mathias
London, United Kingdom

16:00 Panel discussion
At the conclusion of this session, participants should be better able to:

1. Describe the main clinical features of dysautonomia in Parkinson's disease (PD)
2. Indicate the most valuable diagnostic tests for gastrointestinal, genitourinary and cardiovascular disorders in PD
3. Discuss treatment options for these disorders

4203 Teaching Course 

Neuropsychiatry in Parkinson's disease: Beyond dementia

Location: Continental B, Lobby Level
14:30 to 16:30

Chairs: Dag Aarsland
Stavanger, Norway
Bruno Dubois
Paris, France

14:30 Depression and anxiety
Anette Schrag
London, United Kingdom

15:00 Apathy and fatigue
Laura Marsh
Baltimore, MD, USA

15:30 Hallucinations and psychosis
Dag Aarsland
Stavanger, Norway

16:00 Panel discussion
At the conclusion of this session, participants should be better able to:

1. List the symptoms of depression and anxiety, their diagnosis, ratings and treatment in PD
2. Describe how anxiety and depression overlap but are distinct disorders in PD
3. Explain the features of fatigue and apathy in PD, the diagnostic criteria, rating tools available and treatment recommendations based on current evidence
4. Define how fatigue and apathy overlap, but are distinct disorders in PD

DAILY SCHEDULE TUESDAY, JUNE 24, 2008**4203 Teaching Course - continued**

- Review hallucinations and psychosis in PD, their clinical characteristics, pathophysiology and treatment
- Distinguish the typical patterns of hallucinations and psychosis in PD and specifically their differences from hallucinations and psychosis in other medical or psychiatric conditions

4204 Parallel Session**Motor control studies in Movement Disorders**

Location: International Ballroom North, Second Floor
14:30 to 16:30

Chairs: Mark Hallett
Bethesda, MD, USA

Pietro Mazzoni
New York, NY, USA

14:30 Why don't we move faster? Parkinson's disease, movement vigor and implicit motivation

Pietro Mazzoni
New York, NY, USA

15:00 Learning to predict the future: The cerebellum adapts feedforward movement

Amy J. Bastian
Baltimore, MD, USA

15:30 Maladaptive organization of motor cortex in dystonia

Angelo Quartarone
Messina, Italy

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

- Describe the role of dopamine in motor motivation
- Explain the role of the cerebellum in adapting movement
- Discuss the role of plasticity in dystonia

4205 Parallel Session**Gender issues in Movement Disorders**

Location: Continental C, Lobby Level
14:30 to 16:30

Chairs: David G. Standaert
Birmingham, AL, USA

Marie Vidailhet
Paris, France

14:30 Male/female differences in phenotypes of Parkinson's disease and drug responses

Lisa M. Shulman
Baltimore, MD, USA

4205 Parallel Session - continued**15:00 Estrogen issues**

Walter A. Rocca
Rochester, MN, USA

15:30 Non-estrogen issues

David G. Standaert
Birmingham, AL, USA

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

- Discuss the new subtleties of management of the disease, including medications, hormone replacement therapy, pregnancy, etc.
- Explain the arguments in favor of a protective effect of the oestrogens in PD
- Discuss the potential role of the hormonal and non-hormonal factors in the vulnerability to Parkinson's disease

4206 Parallel Session**Nursing roles in Movement Disorders: New horizons**

Location: Boulevard Room, Second Floor
14:30 to 16:30

Chairs: Jeana A. Jaglin
Chicago, IL, USA

Orna Moore
Tel-Aviv, Israel

14:30 The role of the Nurse in medical management

Orna Moore
Tel-Aviv, Israel

15:00 The role of the Nurse in surgical management

Susan Heath
San Francisco, CA, USA

15:30 Expanding the vision of the Movement Disorders Nurse

Cathi Thomas
Boston, MA, USA

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

- Discuss the role and limitations of the nurse/nurse clinician in medical management of Movement Disorder patients
- Review the specialized skills of the nurse/nurse clinician in dealing with the surgical management of Movement Disorder patients.
- Define the core competencies of PD/MDS nurse specialist.
- Recognize the full scope of practice of nursing in Movement Disorders
- Discuss potential ways/areas to enhance the nurse's role in the setting of the participant's own center

DAILY SCHEDULE TUESDAY, JUNE 24, 2008

4207 Parallel Session 

FXTAS

Location: Waldorf Room, Third Floor

14:30 to 16:30

Chairs: Maureen A. Leehey
Aurora, CO, USA

Henry L. Paulson
Ann Arbor, MI, USA

14:30 FXTAS overview: fMRI genotypes, clinical scope and presentation, natural history and videos of patients

Maureen A. Leehey
Aurora, CO, USA

15:00 The molecular underpinnings of FXTAS

Paul Hagerman
Davis, CA, USA

15:30 The epidemiology, diagnosis and treatment of FXTAS

Pierre R. Burkhard
Geneva, Switzerland

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Recognize patients in clinic that are appropriate to test for FXTAS
2. Discuss the genetic risks, clinical manifestations, and therapy of FXTAS
3. Describe the major molecular underpinnings of FMR-1 gene expansion related disease, especially FXTAS

4208 Parallel Session 

Update on dystonia

Location: International Ballroom South, Second Floor

14:30 to 16:30

Chairs: Ryuji Kaji
Tokushima City, Japan

Sabine O. Meunier
Paris, France

14:30 Paired associative stimulation in dystonia

Sabine O. Meunier
Paris, France

15:00 Neuroplasticity at corticostriatal synapses in dystonia models

Paolo Calabresi
Rome, Italy

15:30 Molecular dissection and anatomical basis of dystonia

Satoshi Goto
Tokushima City, Japan

16:00 Panel discussion

4208 Parallel Session - continued 

At the conclusion of this session, participants should be better able to:

1. List arguments derived from human studies and from slice experiments in animals that suggest cortical and /or corticostriatal plasticity is abnormal in dystonia
2. Discuss whether abnormal plasticity is part or consequence of dystonic movements in humans
3. Discuss possible anatomical basis of dystonia

4301 How To Do It - Skills Workshop 

How to examine mental function in patients with movement disorders

Location: Boulevard Room, Second Floor

17:00 to 19:00

David John Burn
Newcastle Upon Tyne, United Kingdom

Murat Emre
Istanbul, Turkey

At the conclusion of this session, participants should be better able to:

1. Describe how to take an informative history utilizing patient and family members
2. Describe a general mental status examination
3. Describe a focused mental status examination pertinent to patients with specific movement disorders, eg. Parkinson's disease, levodopa psychosis, diffuse Lewy body disease, Huntington's disease, etc.

4302 How To Do It - Skills Workshop 

Recognizing, understanding and managing side effects of deep brain stimulation

Location: International Ballroom North, Second Floor

17:00 to 19:00

Marwan I. Hariz
London, United Kingdom

Leo Verhagen
Chicago, IL, USA

At the conclusion of this session, participants should be better able to:

1. Identify hardware problems and their management
2. Recognize the most frequent stimulation-related side effects, including the underlying mechanisms and their management
3. Discuss medication-stimulation interactions and drug management

DAILY SCHEDULE TUESDAY, JUNE 24, 2008**4303 How To Do It - Skills Workshop****Electrophysiological evaluation of patients with movement disorders**

Location: Continental A, Lobby Level
17:00 to 19:00

Peter Brown
London, United Kingdom

Alfredo Berardelli
Roma, Italy

At the conclusion of this session, participants should be better able to:

1. Describe clinically available tools for electrophysiological studies of movement disorders
2. Define electrophysiological findings in different movement disorders
3. Identify psychogenic movement disorders

4304 How To Do It - Skills Workshop**Transcranial sonography (TCS): Clinical research and applications**

Location: Continental B, Lobby Level
17:00 to 19:00

Daniela Berg
Tübingen, Germany

Johann M. Hagenah
Lübeck, Germany

Mark Hallett
Bethesda, MD, USA

At the conclusion of this session, participants should be better able to:

1. Describe the relevant anatomy which is investigated by TCS in patients with movement disorders
2. Identify practice applications of TCS in patients with movement disorders
3. Recognize research applications of TCS in patients with movement disorders

4305 How To Do It - Skills Workshop**How to prepare a video of a movement disorders patient and how to train patients to take videos that are useful**

Location: Williford C, Third Floor
17:00 to 19:00

Janis M. Miyasaki
Toronto, ON, Canada

Carol Brown Moskowitz
New York, NY, USA

4305 How To Do It - Skills Workshop - continued

At the conclusion of this session, participants should be better able to:

1. Discuss the technical issues associated with taking videos of patients
2. Explain how to train Movement Disorders patients to take videos that are useful
3. Describe protocols for testing individual disorders

4401 Video Session**Psychogenic movement disorders**

Location: Grand Ballroom, Second Floor
17:00 to 19:00

David E. Riley
Cleveland Heights, OH, USA

Victor Fung
Sydney, Australia

At the conclusion of this session, participants should be better able to:

1. Describe the clinical characteristics of psychogenic movement disorders
2. Identify additional features that assist in the diagnosis of psychogenic movement disorders
3. Distinguish psychogenic movement disorders from other disorders encountered in the attendee's own clinical practice

4402 Video Session**Dystonic disorders: Primary and secondary**

Location: Continental C, Lobby Level
17:00 to 19:00

Marie Vidailhet
Paris, France

Ryuji Kaji
Tokushima City, Japan

At the conclusion of this session, participants should be better able to:

1. Differentiate the different types of dystonia from other movement disorders
2. Describe the most appropriate means of therapy for each type of dystonia
3. Identify the pros and cons for using deep brain stimulation for treating dystonia

= Ticket required for entry. Please check the Registration Desk for ticket availability.

DAILY SCHEDULE TUESDAY, JUNE 24, 2008 AND WEDNESDAY, JUNE 25, 2008

4403 Video Session



Atypical parkinsonism

Location: International Ballroom South, Second Floor

17:00 to 19:00

Carlo Colosimo
Rome, Italy

Gregor K. Wenning
Innsbruck, Austria

At the conclusion of this session, participants should be better able to:

1. Identify salient features of different types of Atypical parkinsonism
2. Recognize unusual presentations of Atypical parkinsonism
3. Discuss recent advances in Neuroimaging to aid in the differential diagnosis of Atypical parkinsonism

4404 Video Session



Toxic, immune-mediated, infectious movement disorders

Location: Waldorf Room, Third Floor

17:00 to 19:00

Francisco Eduardo C. Cardoso
Belo Horizonte, Brazil

Regina Katzenschlager
Vienna, Austria

At the conclusion of this session, participants should be better able to:

1. Recognize toxic, immune-mediated, and infectious movement disorders
2. Describe how to test for toxic, immune-mediated, and infectious movement disorders
3. Discuss how to treat toxic, immune-mediated, and infectious movement disorders

DAILY SCHEDULE WEDNESDAY, JUNE 25, 2008

5101 Plenary Session III

Presidential Lectures

Location: International Ballroom, Second Floor

8:00 to 10:00

Chairs: Anthony E. Lang
Toronto, Canada

Serge Przedborski
New York, NY, USA

5101 Plenary Session III - continued

8:00 Stanley Fahn Lecture
Dystonia: Found in Translation
Susan B. Bressman
New York, NY, USA



8:30 Junior Award Lecture – Basic Science
Binith Cheeran
London, United Kingdom

8:45 Junior Award Lecture – Clinical Science
Luke A. Massey
London, United Kingdom

9:00 C. David Marsden Lecture
The Basal Ganglia:
Their Mysterious Functions Revisited
Ann M. Graybiel
Cambridge, MA, USA



9:30 Panel discussion

5102 Plenary Session IV

From bench to bedside: What's new in hyperkinetic disorders

Location: International Ballroom, Second Floor

10:30 to 12:30

Chairs: Andrew J. Lees
London, United Kingdom

Kathleen M. Shannon
Chicago, IL, USA

10:30 Ataxias
Thomas Klockgether
Bonn, Germany

11:00 Non-Huntington's disease choreas
Sarah Tabrizi
London, United Kingdom

11:30 Myoclonus/Startle
José A. Obeso
Pamplona, Spain

12:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Discuss the basic science associated with each disease
2. Indicate the clinical phenotypic description of each disease
3. Explain how basic science has helped to understand clinical manifestations, current management and future therapies of each disease

DAILY SCHEDULE WEDNESDAY, JUNE 25, 2008

Poster Presentations

Poster Session 2

Location: Northeast Exhibit Hall, Lower Level
Poster Viewing: 9:00 to 17:00
Authors Present: 12:30 to 14:30
Poster Number: 439-677

Guided Poster Tours

Guided Poster Tour 3 – Genetics (Posters 83-102)

Tour Leaders: Christine Klein and *To be announced*

Guided Poster Tour 4 – Parkinson's disease: Cognition (Posters 257-296)

Tour Leaders: Bruno Dubois and *To be announced*

Location: Southeast Exhibit Hall, Lower Level
Time: 12:30 – 14:30

(Attendees will meet at 12:15 at the MDS Booth, Lower Level)
 Please refer to page 71-72 for abstract details.

5201 Parallel Session

Genetics of Parkinson's disease: Dominant, recessive and complex associations including Gaucher's disease

Location: International Ballroom North, Second Floor
14:30 to 16:30

Chairs: Nobutaka Hattori
Tokyo, Japan

Christine Klein
Lübeck, Germany

14:30 The dominant
 Alexis Brice
Paris, France

15:00 The recessive
 Christine Klein
Lübeck, Germany

15:30 The complex
 Demetrius M. Maraganore
Rochester, MN, USA

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Discuss the genetics of monogenic parkinsonism
2. Recognize the role of genetics in sporadic parkinsonism
3. Describe implications of genetic testing and clinical care

5202 Parallel Session

Clinical trials in Parkinson's disease

Location: International Ballroom South, Second Floor
14:30 to 16:30

Chairs: Karl D. Kieburtz
Rochester, NY, USA

Cristina Sampaio
Lisboa, Portugal

14:30 Clinical outcome measures and surrogate markers
 Karl D. Kieburtz
Rochester, NY, USA

15:00 Design Issues
 Barbara C. Tilley
Charleston, SC, USA

15:30 What's in the pipeline?
 Anthony E. Lang
Toronto, ON, Canada

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. List the common outcome measures in PD clinical trials
2. Identify novel therapeutic agents under study in PD that may have potential future use
3. Identify the interventions currently studied for PD treatment

5203 Parallel Session

Frontotemporal lobar degeneration

Location: Continental B, Lobby Level
14:30 to 16:30

Chairs: Dennis W. Dickson
Jacksonville, FL, USA

Christine Van Broeckhoven
Antwerpen, Belgium

14:30 Clinical spectrum
 Bradley F. Boeve
Rochester, MN, USA

15:00 Genetics of frontotemporal lobar degenerations
 Christine Van Broeckhoven
Antwerpen, Belgium

15:30 Neuropathology of frontotemporal degenerations
 Ian Mackenzie
Vancouver, BC, Canada

16:00 Panel discussion

DAILY SCHEDULE WEDNESDAY, JUNE 25, 2008

5203 Parallel Session - *continued*



At the conclusion of this session, participants should be better able to:

1. Define the clinical spectrum of frontotemporal lobar degeneration
2. Indicate the genetics of frontotemporal lobar degenerations
3. Describe the neuropathology of frontotemporal lobar degenerations

5204 Teaching Course



Tics and stereotypies

Location: Continental C, Lobby Level

14:30 to 16:30

Chairs: Jonathan W. Mink
Rochester, NY, USA

Teresa Temudo
Porto, Portugal

14:30 Tourette's syndrome

Roger M. Kurlan
Rochester, NY, USA

15:00 Movement disorders in autistic children

Paul E. Greene
New York, NY, USA

15:30 Rett's syndrome

Teresa Temudo
Porto, Portugal

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Review the clinical spectrum of tics and stereotypies, tics, and related disorders
2. Discuss the genetics and pathophysiology of these disorders
3. Discuss recent advances in treatment including surgical interventions

5205 Parallel Session



Deep brain stimulation surgery meets neuropsychiatry

Location: Marquette Room, Third Floor

14:30 to 16:30

Chairs: Marwan I. Hariz
London, United Kingdom

Andres M. Lozano
Toronto, ON, Canada

14:30 Abnormal behavior related to DBS

Pierre Pollak
Grenoble, France

5205 Parallel Session - *continued*



15:00 Obsessive compulsive disorder and Tourette's syndrome

Luc Mallet
Paris, France

15:30 Depression

Andres M. Lozano
Toronto, ON, Canada

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Recognize acute side effects induced by STN DBS
2. Discuss what they tell us about the physiology of the STN and the role of basal ganglia in normal and abnormal behavior
3. Describe the effects of surgery in different targets on OCD
4. Identify the neuronal networks that are implicated in OCD
5. Describe the effects of surgery on different targets in depression and the lessons for the understanding of the biology of depression

5206 Parallel Session



Recent insights into Huntington's disease

Location: Continental A, Lobby Level

14:30 to 16:30

Chairs: Christopher A. Ross
Baltimore, MD, USA

Paul Muchowski
San Francisco, CA, USA

14:30 Transcriptional dysregulation in Huntington's disease

Dimitri Krainc
Charlestown, MA, USA

15:00 Protein quality control and degradation

Paul Muchowski
San Francisco, CA, USA

15:30 What observational studies (PHAROS and PREDICT) have told us about the course of HD and early features

Jane Paulsen
Iowa City, IA, USA

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Describe pathogenic mechanisms in HD and related disorders
2. State importance of early clinical changes and biomarkers in HD
3. Discuss potential routes to therapy for HD and related disorders

DAILY SCHEDULE WEDNESDAY, JUNE 25, 2008**5207 Parallel Session****Speech and swallowing disorders in Parkinson's disease**

Location: Boulevard Room, Second Floor
14:30 to 16:30

Chairs: Ronald F. Pfeiffer
Memphis, TN, USA

Emily Wang
Chicago, IL, USA

14:30 Diagnosis and treatment of speech impairments in Parkinson's disease

Lorraine Ramig
Boulder, CO, USA

15:00 Recent findings on pharmacological and surgical management of Parkinson's disease on speech motor function

Emily Wang
Chicago, IL, USA

15:30 Diagnosis and treatment of swallowing disorders in Parkinson's disease

David H. McFarland
Montreal, QC, Canada

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Recognize the signs and symptoms of voice and speech disorders occurring in 90% of individuals with PD
2. State the significant role of effective speech and voice treatment throughout the course of disease progression
3. Identify and describe the physiological, acoustic and perceptual measurements that are frequently used to document speech outcomes associated with pharmacological and surgical management of PD
4. Recognize the role of speech treatment in managing surgical complications in speech
5. Recognize the signs and symptoms of swallowing disorders in PD
6. Describe the current approaches to diagnose and treat swallowing disorders in PD

5208 Teaching Course**Vascular and post-hypoxic movement disorders**

Location: Williford C, Third Floor
14:30 to 16:30

Chairs: Vladimir Kostic
Belgrade, Serbia and Montenegro

Werner Poewe
Innsbruck, Austria

5208 Teaching Course - continued**14:30 Vascular parkinsonism and gait disorders**

Nir Giladi
Tel Aviv, Israel

15:00 Hyperkinetic movement disorders: Acute and delayed

Vladimir Kostic
Belgrade, Serbia and Montenegro

15:30 Movement disorders due to global hypoxia/ischemia

Uday B. Muthane
Bangalore, India

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Recognize the clinical spectrum of movement disorders in the context of vascular encephalopathies
2. Discuss the lesion patterns and pathophysiology underlying vascular movement disorders
3. Differentiate features of vascular parkinsonism from those of neurodegenerative forms of parkinsonism
4. Explain the difference between movement disorders induced by cerebral microangiopathy vs. those caused by local ischemia or large brain infarcts.
5. Describe the clinical phenotype and diagnostic work-up of patients with gait disorders caused by subcortical arteriosclerotic encephalopathy.
6. Explain the principles of therapeutic management in patients with vascular movement disorders

5301 How To Do It - Skills Workshop**Assessment of sleep disorders in the clinical practice of Movement Disorders**

Location: Williford B, Third Floor
17:00 to 19:00

Alex Iranzo
Barcelona, Spain

Birgit Högl
Innsbruck, Austria

At the conclusion of this session, participants should be better able to:

1. Discuss the sleep scales that are available to screen for sleep disturbances in Movement disorders
2. Explain the appropriate applications of each
3. Explain the indications for referral of a movement disorders patient for a sleep laboratory assessment
4. Describe the different features of sleep that are evaluated by polysomnography, MSLT and MWT

DAILY SCHEDULE WEDNESDAY, JUNE 25, 2008

5302 How To Do It - Skills Workshop 

Botulinum toxin: Specialized issues and applications

Location: Continental A, Lobby Level
17:00 to 19:00

Marie Hélène Marion
London, United Kingdom

Marco Onofri
Pescara, Italy

At the conclusion of this session, participants should be better able to:

1. Recognize phenotypes of spasticity
2. Explain how to treat spasticity and drooling with botulinum toxin
3. Identify truncal axial dystonias and evaluate methods of treatment

5303 How To Do It - Skills Workshop 

How to recognize normal and abnormal movement in children

Location: Williford C, Third Floor
17:00 to 19:00

Nardo Nardocci
Milano, Italy

Terence D. Sanger
Stanford, CA, USA

At the conclusion of this session, participants should be better able to:

1. Identify the primary features of major categories of pediatric movement disorders, including dystonia, chorea, myoclonus, tremor, and tics
2. Recognize typical features of psychogenic movement disorders in children
3. Distinguish between movement disorders and certain normal movements in young children

5304 How To Do It - Skills Workshop 

The MDS-UPDRS: How to apply the new UPDRS in practice and research settings

Location: Waldorf Room, Third Floor
17:00 to 19:00

Chairs: Christopher G. Goetz
Chicago, IL, USA

Glenn T. Stebbins
Chicago, IL, USA

5304 How To Do It - Skills Workshop - continued 

17:00 MDS-UPDRS
Christopher G. Goetz
Chicago, IL, USA

17:30 Clinimetric testing program results
Stephanie R. Shaftman
Charleston, SC, USA

18:00 Application of the MDS-UPDRS: Rating patient examples
Glenn T. Stebbins
Chicago, IL, USA

At the conclusion of this session, participants should be better able to:

1. Explain the background, development, new features, and caveats of MDS-UPDRS
2. Describe the clinimetric testing program completed so far and the next steps envisioned
3. Perform ratings of patients using the MDS-UPDRS
4. Compare the ratings with those from the original UPDRS

5305 How To Do It - Skills Workshop 

Examining the ataxic patient

Location: Marquette Room, Third Floor
17:00 to 19:00

Tanja Schmitz-Hubsch
Bonn, Germany

S.H. Subramony
Galveston, TX, USA

At the conclusion of this session, participants should be better able to:

1. Describe the limb movement and body posture disorders associated with cerebellar dysfunction
2. Discuss the properties of the different ataxia scales, including ICARS, FARS and SARA
3. Discuss the proper use of the different ataxia scales, including ICARS, FARS and SARA

5401 Video Session 

Uncommon hyperkinetic movement disorders

Location: Continental B, Lobby Level
17:00 to 19:00

Kailash P. Bhatia
London, United Kingdom

Yoshikazu Ugawa
Fukushima, Japan

DAILY SCHEDULE WEDNESDAY, JUNE 25, 2008 AND THURSDAY, JUNE 26, 2008

5401 Video Session - continued 

At the conclusion of this session, participants should be better able to:

1. Recognize the clinical phenomenology of uncommon hyperkinetic movement disorders
2. Describe the pathophysiology underlying uncommon hyperkinetic movement disorders
3. Classify uncommon hyperkinetic movement disorders and the differential diagnosis from classical syndromes

5402 Video Session 

Metals and movement disorders

Location: Williford A, Third Floor
17:00 to 19:00

Kapil D. Sethi
Augusta, GA, USA

Neeraj Kumar
Rochester, MN, USA

At the conclusion of this session, participants should be better able to:

1. Discuss how abnormal environmental exposure to heavy metals, such as iron and manganese, are involved in neurologic disease
2. Interpret the toxicokinetics of metals of interest and the preferred medium for analysis for each
3. Describe the pathogenesis of how free radical formation induced by excess heavy metals impacts the basal ganglia

5403 Video Session 

Movement disorder look-alikes: The great pretenders

Location: Continental C, Lobby Level
17:00 to 19:00

Aikaterini Kompoliti
Chicago, IL, USA

Philip D. Thompson
North Terrace, Adelaide, Australia

At the conclusion of this session, participants should be better able to:

1. Recognize phenocopies of common movement disorders in patients with a variety of underlying conditions
2. Identify disorders that can assemble classical movement disorder syndromes
3. Utilize differential diagnostic tests to distinguish between classical movement disorder syndromes and their look-alikes

5404 Video Session 

Gait disorders

Location: Boulevard Room, Second Floor
17:00 to 19:00

Bastiaan R. Bloem
Nijmegen, Netherlands

John G. Nutt
Portland, OR, USA

At the conclusion of this session, participants should be better able to:

1. Differentiate various gait disorders
2. Explain the investigation of various gait disorders
3. Discuss the treatment of various gait disorders

Video Olympics
Reception with hors d'oeuvres and drinks
Grand Ballroom, Second Floor
19:00-20:00

Video Olympics
International Ballroom, Second Floor
20:00-23:00

Masters of Ceremony:
Anthony Lang
Kapil Sethi

Panel of Experts:
Joseph Jankoric
Philip Thompson
Werner Poewe
Niall Quinn
Eduardo Olosa

DAILY SCHEDULE THURSDAY, JUNE 26, 2008

6101 Plenary Session V

Hot topics in sleep and Movement Disorders

Location: International Ballroom, Second Floor
8:00 to 10:00

Chairs: Cynthia L. Comella
Chicago, IL, USA
Alex Iranzo
Barcelona, Spain

8:00 New concepts in anatomy and physiology of sleep
Clifford B. Saper
Boston, MA, USA

8:30 Excessive daytime sleepiness in Parkinson's disease and related disorders
Jerry Siegel
North Hills, CA, USA

DAILY SCHEDULE THURSDAY, JUNE 26, 2008

6101 Plenary Session V - continued

9:00 **New developments in restless legs syndrome**

Juliane Winkelmann
Munich, Germany

9:30 **Panel discussion**

At the conclusion of this session, participants should be better able to:

1. Describe the anatomy and pathophysiology of normal sleep, and the implications for sleep disorders
2. Describe the underlying causes and pathogenesis of excessive daytime sleepiness in Parkinson's disease
3. Explain the new advances in the genetics and pathophysiology of RLS and periodic limb movements of sleep

6151 Controversies in Movement Disorders

Location: International Ballroom, Second Floor
10:30 to 12:30

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

Eduardo Tolosa
Barcelona, Spain

10:30 **EMBR: Help or hindrance to clinical management?**

Help
Cristina Sampaio
Lisboa, Portugal

10:45 **Hindrance**

William J. Weiner
Baltimore, MD, USA

DBS for Parkinson's disease will be passé in 5 years:

11:00 **Yes**
Stanley Fahn
New York, NY, USA

11:15 **No**
Paul Krack
Grenoble, France

Is sporadic Parkinson's disease a genetic disorder?

11:30 **Yes**
Vincenzo Bonifati
Rotterdam, Netherlands

11:45 **No**
J. William Langston
Sunnyvale, CA, USA

6151 Controversies in Movement Disorders - continued

12:00 **Essential tremor: Multi-system disorder?**

Yes
Elan D. Louis
New York, NY, USA

12:15 **No**
Günther Deuschl
Kiel, Germany

At the conclusion of this session, participants should be better able to:

1. Indicate if EBMR is a help or hindrance in clinical management
2. Explain if DBS will be passé in 5 years
3. Explain whether or not sporadic PD is a genetic disorder
4. Describe whether essential tremor is a multi-system disorder or narrow condition

MDS Business Meeting

Location: International Ballroom, Second Floor
Open to all delegates.
12:30-13:30

Poster Presentations

Poster Session 3

Location: Southwest Exhibit Hall, Lower Level
Poster Viewing: 9:00 to 16:00
Authors Present: 12:30 to 14:30
Poster Numbers: 678-1210

Guided Poster Tours

Guided Poster Tour 5 – Surgical Therapy (Posters 315-334)
Tour Leaders: Michael Okun and Jerry Vitek
Guided Poster Tour 6 – Neuropharmacology (Posters 226-245)

Tour Leaders: David Standaert and *To be announced*
Location: Southeast Exhibit Hall, Lower Level
Time: 12:30 – 14:00

(Attendees will meet at 12:15 at the MDS Booth, Lower Level)
Please refer to page 73-74 for abstract details.

6201 Parallel Session

Update on molecular pathogenesis and protein interactions in Parkinson's disease

Location: Waldorf Room, Third Floor
14:30 to 16:30

Chairs: Un Jung Kang
Chicago, IL, USA
Leonidas Stefanis
Papagou, Greece

DAILY SCHEDULE THURSDAY, JUNE 26, 2008

6201 Parallel Session - continued 

- 14:30 Alpha-Synuclein - Molecular pathogenesis**
Leonidas Stefanis
Papagou, Greece
- 15:00 Normal and pathological function of LRRK2**
Takeshi Iwatsubo
Tokyo, Japan
- 15:30 Update on PINK1 biology**
Ming Guo
Los Angeles, CA, USA
- 16:00 Panel discussion**
At the conclusion of this session, participants should be better able to:
1. Discuss the biology of α -synuclein, its aberrant conformations and their role in the pathogenesis of Parkinson's disease
 2. Describe the role of LRRK2, phosphorylation activity and GTPase activity and cytotoxicity
 3. Recognize the role of PINK1 and its interactors in the pathogenesis of Parkinson's disease

6202 Parallel Session 

- Update on DBS for Parkinson's disease and dystonia**
Location: International Ballroom South, Second Floor
14:30 to 16:30
- Chairs: Joachim K. Krauss
Hannover, Germany
Pierre Pollak
Grenoble, France
- 14:30 STN vs. GPi**
Michael S. Okun
Gainesville, FL, USA
- 15:00 Pedunculopontine nucleus DBS for the treatment of gait problems in Parkinson's disease**
Elena Moro
Toronto, ON, Canada
- 15:30 DBS in primary and secondary dystonia**
Joachim K. Krauss
Hannover, Germany
- 16:00 Panel discussion**

6202 Parallel Session - continued 

- At the conclusion of this session, participants should be better able to:
1. Discuss the rationale for choosing the STN or the GPi for DBS in patients with Parkinson's disease
 2. Explain the rationale for considering the PPN as a target for DBS in Parkinson's disease
 3. Define the status of DBS for treatment of dystonia and what remains to be accomplished


6203 Parallel Session 

- Electrical oscillatory correlates of parkinsonism**
Location: Continental A, Lobby Level
14:30 to 16:30
- Chairs: Hagai Bergman
Jerusalem, Israel
John C. Rothwell
London, United Kingdom
- 14:30 Link between akinesia and abnormal cortical electrical oscillation**
Hagai Bergman
Jerusalem, Israel
- 15:00 Is rigidity related to slow variations of electrical oscillation in the basal ganglia?**
Peter Brown
London, United Kingdom
- 15:30 Tremor and dysfunction of the cortico-basal ganglia electrical network**
Jens Volkmann
Kiel, Germany
- 16:00 Panel discussion**

- At the conclusion of this session, participants should be better able to:
1. Explain range of physiological oscillations in cortico-basal ganglia – thalamo-cortical networks
 2. Discuss relationship of pathological oscillations with akinesia, rigidity and tremor
 3. Explain therapeutic implications of network oscillation

6204 Teaching Course 

- PSP and CBD**
Location: Continental B, Lobby Level
14:30 to 16:30
- Chairs: Oscar S. Gershanik
Buenos Aires, Argentina
Lawrence I. Golbe
New Brunswick, NJ, USA

 = Ticket required for entry. Please check the Registration Desk for ticket availability.

DAILY SCHEDULE THURSDAY, JUNE 26, 2008

6204 Teaching Course - *continued*

- 14:30 Clinical diagnosis and differential diagnosis of PSP and CBD**
Lawrence I. Golbe
New Brunswick, NJ, USA
- 15:00 Pathology and role of genetics**
Dennis W. Dickson
Jacksonville, FL, USA
- 15:30 Therapeutic interventions: Current and future**
Irene Litvan
Louisville, KY, USA
- 16:00 Panel discussion**

At the conclusion of this session, participants should be better able to:

1. Differentiate PSP and CBD from other parkinsonian and dementing disorders in the clinic
2. Identify experimental diagnostic modalities
3. Describe tau pathology in PSP and CBD with respect to cell type and neuroanatomical distribution of neurodegeneration
4. Compare biochemical and genetic similarities and differences between PSP and CBD
5. Discuss the available palliative treatments and management strategies
6. Discuss experimental therapeutics at various points in the development pipeline

6206 Parallel Session

Hot topics in experimental therapeutics for Parkinson's disease

Location: International North, Second Floor
14:30 to 16:30

Chairs: Shinichi Muramatsu
Shimotsuke, Japan

Clive N. Svendsen
Madison, WI, USA

14:30 Gene therapy
Jeffrey H. Kordower
Chicago, IL, USA

15:00 Cell-based therapies
Olle Lindvall
Lund, Sweden

15:30 Infusion therapies
Werner Poewe
Innsbruck, Austria

16:00 Panel discussion

6206 Parallel Session - *continued*

At the conclusion of this session, participants should be better able to:

1. Describe the rationale for gene therapy in Parkinson's disease and the challenges for this field
2. Discuss why cellular transplants have not yet provided a complete cure for Parkinson's disease
3. Identify important experiments that need to be done to move the field forward
4. Explain the challenges of infusion therapies and how they may be used to treat movement disorders

6207 Teaching Course

Impulse control disorders

Location: Continental C, Lobby Level

14:30 to 16:30

Chairs: Andrew H. Evans
North Fitzroy, Australia

Mark A. Stacy
Durham, NC, USA

14:30 Clinical spectrum (relationship/differences between ICDs and DDS); Relationship to addiction

Andrew H. Evans
North Fitzroy, Australia

15:00 Pathogenesis: Anatomy, role of dopamine, genetic factors, imaging

Antonio P. Strafella
Toronto, ON, Canada

15:30 Assessment, clinical pharmacology and management

Daniel Weintraub
Philadelphia, PA, USA

16:00 Panel discussion

At the conclusion of this session, participants should be better able to:

1. Recognize behaviors associated with impulse control difficulties in PD and other movement disorders
2. Define dopamine dysregulation syndrome, punning and addictive behaviors
3. List and define the differences between the nigrostriatal, mesolimbic and mesocortical pathways, and dopamine receptor stimulation in PD
4. Identify the important differences in the direct and indirect pathway stimulation in respect to dyskinesias, impulse control disorders and the dopamine dysregulation syndrome
5. Describe the relationship between disease progression and increasing dopaminergic therapy in PD
6. Discuss risk factors and treatment options for patients with impulse control behaviors

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Education



Research



Advocacy

UCB is proud to join The *Movement* Disorder Society in the commitment to creating a better future for patients and caregivers living with movement disorders.



EXHIBITOR INFORMATION

GENERAL INFORMATION AND EXHIBIT HOURS

Please allow adequate time in your daily schedule to visit the Exhibit Hall, located in the Northwest Hall which is on the Lower Level of the Hilton Chicago. The exhibition is an integral component of your International Congress experience, offering you the opportunity to speak with representatives of companies providing services or marketing products directly related to Movement Disorders. Delegates may visit the Exhibit Hall during the following hours:

Monday, June 23	9:00 – 17:00
Tuesday, June 24	9:00 – 17:00
Wednesday, June 25	9:00 – 17:00
Thursday, June 26	9:00 – 16:00

EXHIBITOR REGISTRATION

Location: Inside Northwest Hall, Lower Level

Exhibitors must register at the Exhibitor Registration Desk during the following hours:

Sunday, June 22	9:00 – 19:00
Monday, June 23	7:00 – 17:30
Tuesday, June 24	7:00 – 17:30
Wednesday, June 25	7:00 – 17:30
Thursday, June 26	7:00 – 16:30

EXHIBITOR BADGE POLICY

Admission to the Exhibit Hall will be by name badge or Exhibit Hall Pass only. Security guards will monitor Exhibit Hall entrances for proper identification. Exhibit stand personnel must show an official MDS exhibitor name badge in order to gain access to the Exhibit Hall during installation, show, or dismantlement hours. Independent contractor personnel, hired by an exhibitor to install and dismantle their display, should register onsite at the GES Service Desk located within the Northwest Exhibit Hall, for a temporary name badge valid for only installation and dismantlement hours.

Exhibitor Badge (Yellow):

Allows admittance to the exhibit hall area only.

Exhibitor Delegate Badge (Orange)

Allows the delegate to enter the Exhibit hall as an exhibitor and attend scientific sessions (some requiring a ticket) and Social Events.

ENDORSEMENT DISCLAIMER

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

Back by popular demand,

Dr. Christopher Goetz has organized another intriguing MDS History Exhibit, as well as the History of Chicago Neurology. These stimulating displays recount the history of the Movement Disorders subspecialty, the contributions of James Parkinson, as well as the legacy of local Chicago hospitals, universities and organizations to the field of Movement Disorders.

Visit the History Exhibit in the
Mobley Room on the Lower Level
of the Hilton Chicago.



EXHIBITOR DIRECTORY

ALLERGAN

2525 Dupont Drive
Irvine, CA 92612
USA
Telephone: +1 714-246-6337
Fax: +1 714-246-6587
Web site: www.allergan.com

Booth #: 318

Allergan is a world leader in neuromodulator therapy and neurosciences. For nearly two decades, we have been committed to the research and clinical development of BOTOX (botulinum toxin type A), one of the world's most versatile medicines, to improve the physical well-being and quality of life for people around the world who suffer from a variety of serious or debilitating disorders. BOTOX is currently available in more than 75 countries with 20 approved indications.

AMERICAN PARKINSON DISEASE ASSOCIATION

135 Parkinson Avenue
Staten Island, NY 10305
USA
Telephone: +1 800-223-2732
Fax: +1 718-981-4399
Web site: www.apdaparkinson.org

Table #: 1

The American Parkinson Disease Association (APDA) mission is "to ease the burden, to find the cure" through research, patient and caregiver support and education. Educational materials will be provided from the exhibit.

BENIGN ESSENTIAL BLEPHAROSPASM RESEARCH FOUNDATION, INC.

P.O. Box 12468
Beaumont, TX 77726-2468
USA
Telephone: +1 409-832-0788
Fax: +1 409-832-0890
Web site: www.blepharospasm.org

Table #: 7

The Benign Essential Blepharospasm Research Foundation, founded in 1981, is the only organization dedicated solely to finding the cause and cure for Blepharospasm, Meige and related disorders. BEBREF, a volunteer directed, nonprofit organization, has support groups nationwide, promotes awareness and research, distributes educational material to patients and physicians and serves as a referral clearing house.

BOEHRINGER INGELHEIM PHARMACEUTICALS, INC.

900 Ridebury Road
Ridgefield, CT 06877
USA
Telephone: +1 203-798-9988
Fax: +1 203-798-4674
Web site: us.boehringer-ingelheim.com

Booth #: 201

Boehringer Ingelheim Pharmaceuticals, Inc., the US subsidiary of Boehringer Ingelheim, headquartered in Germany, operates globally in 47 countries with approximately 39,800 employees. The company is committed to researching, developing, manufacturing and marketing novel products of high therapeutic value for human and veterinary medicine.

For more information please visit us.boehringer-ingelheim.com.

CLEVEMED

4415 Euclid Ave., 4th Floor
Cleveland, OH 44103
USA
Telephone: +1 800-CleveMed
Fax: +1 216-791-6739
Web site: www.clevemed.com

Booth #: 332

Kinesia, developed by CleveMed, is a compact user-worn system that uses motion sensors and electromyography to quantify the severity of upper extremity movement disorder symptoms, such as tremor and bradykinesia. Parkinson's disease or essential tremor specialists can use Kinesia to track symptoms during an exam or at a patient's home.

CURE PSP/SOCIETY FOR PSP

EP III, 11350 McCormick Rd. Suite 906
Hunt Valley, MD 21031
USA
Telephone: +1 800-457-4777
Fax: +1 757-838-6086
Web site: www.curepsp.org

Table #: 10

Cure and prevent PSP and CBD (progressive supranuclear palsy and corticobasal degeneration). Cure PSP exists to increase awareness of PSP and CBD, fund research toward a cure and prevention, education healthcare professionals and provide support, education and hope for persons and families with PSP and CBD.

EXHIBITOR DIRECTORY

ELSEVIER

927 Leverenz Rd.
Naperville, IL 60565
USA
Telephone: +1 630-420-0756
Fax: +1 630-523-7712
Web site: www.elsevier.com

Booth #: 327

Elsevier, a world leader in publishing, will have on display all their books and journals related to Parkinson's disease and Movement Disorders. Featured items will be the journal *Parkinsonism and Related Disorders* and two new books, Fahn: *Principles and Practice of Movement Disorders: Text and DVD* and Koller: *Parkinson's Disease and Related Disorders: Volume 1 and 2*.

EMD SERONO INC.

1 Technology Place
Rockland, MA 02339
USA
Telephone: +1 781-982-9000
Fax: +1 781-681-2932
Web site: www.merckserono.net

Booth #: 407

Headquartered in Geneva, Switzerland, Merck Serono* discovers, develops, manufactures and markets innovative small molecules and biopharmaceuticals to help patients with unmet medical needs. Merck Serono has a long-term commitment to the development of innovative treatments to help manage neurological disorders such as Multiple Sclerosis (MS) and Parkinson's disease (PD).

*Merck Serono S.A. is an affiliate of Merck KGaA, Darmstadt, Germany. The division Merck Serono operates in North America under the name EMD Serono.

GE HEALTHCARE LTD.

Pollards Wood
Nightingales Lane
Chalfont, St Giles, Bucks HP8 4SP
United Kingdom
Telephone: + 44 1494 544 000
Fax: + 44 1494 498 234
Web site: www.gehealthcare.com

Booth #: 402

GE Healthcare is dedicated to helping you transform healthcare delivery by driving critical breakthroughs in biology and technology. Our expertise in medical imaging is enabling healthcare professionals around the world discover new ways to detect disease earlier, access more information and intervene earlier with more targeted treatments, so they can help their patients live their lives to the fullest.

GLAXOSMITHKLINE

5 Moore Drive
Research Triangle Park, NC 27709
USA
Telephone: +1 800-366-8900
Fax: +1 919-315-6049
Web site: www.gsk.com

Booth #: 221

GlaxoSmithKline is a leading research-based pharmaceutical company with a powerful combination of skills to discover and deliver innovative medicines. We offer a number of programs to support effective health management strategies and improve patient care.

Please visit our exhibit to learn more about our products.

IN-STEP MOBILITY

8027 N. Monticello
Skokie, IL 60076
USA
Telephone: +1 847-676-1275
Fax: +1 847-676-1202
Web site: www.ustep.com

Booth #: 227

Advanced Mobility aids for Parkinson's. Two main products: U-Step walking stabilizer-an advanced walker that is excellent for fall prevention. The second product is the LaserCane for Parkinson's freezing.

EXHIBITOR DIRECTORY

INTERNATIONAL ESSENTIAL TREMOR FOUNDATION

P.O. Box 14005
 Lenexa, KS 66285-4005
 USA
 Telephone: +1 913-341-3880
 Fax: +1 913-341-1296
 Web site: www.essentialtremor.org

Table #: 5

The IETF distributes educational information to the public, patients, and physicians around the world who are affected by ET. The IETF works to: (1) increase public awareness about the disorder; (2) create support groups in local communities throughout the United States; (3) financially support research in ET; (4) provide patients access to specialist who treat the condition; (5) publish newsletters that; and, (6) support community education programs to improve quality of life for those affected.

IPSEN

42 Rue du Dr Blanche
 Paris 75016
 France
 Telephone: +33 1 44 30 4315
 Fax: +33 1 44 30 4200
 Web site: www.ipsen.com

Booth #: 131

Ipsen is an innovation driven international specialty pharmaceutical group with over 20 products on the market. The company's development strategy is based on a combination of products in targeted therapeutic areas (oncology, endocrinology and neuromuscular disorders) which are growth drivers, and primary care products which contribute significantly to its research financing. The location of its four Research and Development centers (Paris, Boston, Barcelona and London) gives the Group a competitive edge in gaining access to leading university research teams and highly qualified personnel.

LEWY BODY DEMENTIA ASSOCIATION

P.O. Box 451429
 Atlanta, GA 31145
 USA
 Telephone: +1 404-935-6444
 Fax: +1 480-422-5434
 Web site: www.lbda.org

Table #: 3

LBDA is a leading resource for those affected by Lewy body dementias (LBD). LBD is a progressive brain disease and the second most common cause of neurodegenerative dementia after Alzheimer's disease. Patients with LBD have similar problems to those with Parkinson's disease with dementia.

LIPPINCOTT WILLIAMS & WILKINS A WOLTERS KLUWER BUSINESS

1578 Fordham Street
 Bolingbrook, IL 60490
 USA
 Telephone: +1 630-776-7108
 Fax: +1 630-771-0941
 Web site: www.lww.com

Booth #: 432

Publisher of the latest books and journals available in neurology.

MEDTRONIC, INC.

710 Medtronic Parkway
 Minneapolis, MN 55432
 USA
 Telephone: +1 800-707-0933
 Fax: +1 763-514-0050
 Web site: www.medtronic.com

Booth #: 213

Medtronic is the world leader in medical technology providing lifelong solutions for people with chronic disease. Each year, 6 million patients benefit from Medtronic's technology. Activa® DBS Therapy has been used in more than 40,000 patients for the treatment of three movement disorders; Parkinson's disease, essential tremor and dystonia.

EXHIBITOR DIRECTORY

MERZ PHARMACEUTICALS GMBH

Eckenheimer Landstrasse 100
Frankfurt 60318
Germany
Telephone: +49-69-1503-290
Fax: +49-69-1503-722
Web site: www.merz.com

Booth #: 401

Merz Pharmaceuticals is a research-based innovative pharmaceutical company with focus on unmet needs in neurology and aesthetic dermatology. Merz proudly developed memantine, the first drug worldwide for the treatment of moderate to severe Alzheimer's disease, and introduced Xeomin[®], the first and only Botulinum Toxin Type A, free of complexing proteins.

MOUSE SPECIFICS INC.

8 Faneuil Hall, 3rd Floor
Boston, MA 02109
USA
Telephone: +1 617-973-5009
Fax: +1 617-973-6469
Web site: www.mousespecifics.com

Booth #: 331

Mouse Specifics provides instrumentation to describe gait and autonomic nervous system disturbances in animal models of Parkinson's disease. DigiGait is able to discriminate basal ganglia from motorneuron influences on walking. ECGenic rapidly reports the effects of neurotoxins on the heart.

NATIONAL PARKINSON FOUNDATION

1501 NW 9th Ave.
Miami, FL 33136
USA
Telephone: +1 305-243-6666
Fax: +1 305-243-5595
Web site: www.parkinson.org

Table #: 4

The National Parkinson Foundation (NPF), chartered in 1957, is dedicated to supporting research and providing education, services, support, advocacy, and outreach to the Parkinson community. With a national and international presence, NPF is the largest organization in the world that serves people with Parkinson's disease and their families.

NATIONAL SPASMODIC DYSPHONIA ASSOCIATION

300 Park Boulevard, Suite 415
Itasca, IL 60143
USA
Telephone: +1 800-795-6732
Fax: +1 630-250-4505
Web site: www.dysphonia.org

Table #: 8

The mission of the National Spasmodic Dysphonia Association is to advance medical research into the causes of and treatments for spasmodic dysphonia, promote physician and public awareness of the disorder, and provide support to those affected by spasmodic dysphonia. A new research grant program was established in 2007.

NATIONAL SPASMODIC TORTICOLLIS ASSOCIATION

9920 Talbert Avenue
Fountain Valley, CA 92708
USA
Telephone: +1 714-378-9837
Web site: www.torticollis.org

Table #: 6

The National Spasmodic Torticollis Association is a non-profit organization dedicated to providing information and support to ST patients, educating the public and medical community about ST, advocating for the rights of those with ST, and promoting research on ST.

NOVARTIS PHARMACEUTICALS

One Health Plaza
East Hanover, NJ 07936
USA
Telephone: +1 862-778-8300
Web site: www.novartis.com

Booth #: 326

Novartis Pharmaceuticals Corporation is dedicated to discovering, developing, manufacturing and marketing prescription drugs that help meet our customers' medical needs and improve their quality of life. Please visit the Novartis Pharmaceuticals Corporation exhibit where our Account Professionals will be available to discuss our products and answer your questions.

Please visit the combined exhibition of Novartis and Orion.

EXHIBITOR DIRECTORY

ORION CORPORATION ORION PHARMA

Orionintie 1
 FI-02101 Espoo
 Finland
 Telephone: + 358-10-4261
 Fax: +358-10-426-3815
 Web site: www.orion.fi/english

Booth #: 326

Orion Corporation is a Finnish listed company which is dedicated to treating and preventing disease by discovery and developing innovative medicinal treatments. Orion is the originator of Stalevo® (levodopa, carbidopa, entacapone) for Parkinson's disease.

Please visit the combined exhibition of Novartis and Orion.

PARKINSON'S DISEASE FOUNDATION

1359 Broadway, Ste. 1509
 New York, NY 10018
 USA
 Telephone: +1 212-923-4700
 Fax: +1 212-923-4778
 Web site: www.pdf.org

Booth #: 125

The Parkinson's Disease Foundation offers a wide variety of materials and quarterly-published newsletters for the ongoing education of persons with PD and their families and friends. Visit our booth to view these materials and request supplies for your office or clinic. For scientists involved in PD research, visit us to learn of our research grant programs.

PRESTWICK PHARMACEUTICALS, INC.

1825 K St NW, Suite 1475
 Washington, DC 20006
 USA
 Telephone: +1 202-296-1400
 Fax: +1 202-296-2169
 Web site: www.prestwickpharma.com

Booth #: 334

Prestwick Pharmaceuticals, Inc. is a late-stage product-based biopharmaceutical company engaged in the development and marketing of drugs for diseases of the central nervous system (CNS). Our portfolio of drugs addresses significant unmet medical needs in such CNS disorders as Huntington's disease, Parkinson's disease and Schizophrenia.

SENSONICS, INC.

125 White Horse Pike
 Haddon Heights, NJ 08035
 USA
 Telephone: +1 856-547-7702
 Fax: +1 856-547-5665
 Web site: www.sensonics.com

Booth #: 431

Sensonics, Inc., manufactures and distributes quality, quantitative smell and taste tests. The Smell Identification Test™ is the standard means for assessing olfactory function throughout the world. Visit www.sensonics.com for more information about our products and services.

SOLSTICE NEUROSCIENCES, INC.

40 General Warren Blvd., Suite 160
 Malvern, PA 19355
 USA
 Telephone: +1 267-620-8000
 Fax: +1 267-620-8190
 Web site: www.solsticeneuro.com

Booth #: 232, 333

Solstice Neurosciences, Inc. is focused on the development, manufacturing, sales and marketing of specialty biopharmaceutical products. Solstice's first product, Myobloc® (Botulinum Toxin Type B) Injectable Solution, represents the only botulinum toxin type B currently available worldwide. MYOBLOC® is indicated to reduce the severity of abnormal head position and neck pain associated with cervical dystonia.

EXHIBITOR DIRECTORY

SOLVAY PHARMACEUTICALS GMBH

Hans-Böckler-Allee 20
Hannover 30173
Germany
Telephone: +49 511-857-3159
Fax: +49 511-857-2294
Web site: www.solvaypharmaceuticals.com

Booth #: 115

Solvay Pharmaceuticals is a global player in selected disease target areas. A strong focus concentrates research and development efforts into clinical indications where doctors and patients want new and better therapies to choose from. The same focus in sales and marketing teams gives us a strong presence in segments like neurology. Solvay Pharmaceuticals is spreading quickly from Europe, USA and Canada into other countries like Brazil, Australia, China and Mexico today.

TEVA NEUROSCIENCE, INC.

901 East 104th St., Suite 900
Kansas City, MO 64131
USA
Telephone: +1 816-508-5000
Fax: +1 816-508-5010
Web site: www.tevaneuroscience.com

Booth #: 101, 111

Azilect® (rasagiline tablets) is indicated for the treatment of the signs and symptoms of idiopathic Parkinson's disease as initial monotherapy and as adjunct therapy to levodopa. Azilect® is marketed in the United States by Teva Neuroscience, Inc.

TEVA PHARMACEUTICAL INDUSTRIES, LTD.

P.O. Box 3190
Petach Tikva 49131
Israel
Telephone: +972-3-9267152
Fax: +972-3-9267852
Web site: www.tevapharm.com

Booth #: 101, 111

H. LUNDBECK A/S

Ottiliavej 9
DK-2500 Valby
Denmark
Telephone: +45-36-43-29-30
Fax: +45-36-43-8900
Web site: www.lundbeck.com

Booth #: 101, 111

THE MOVEMENT DISORDER SOCIETY

International Secretariat
555 East Wells Street, Suite 1100
Milwaukee, WI 53202-3823
USA
Telephone: +1 414-276-2145
Fax: +1 414-276-3349
Web site: www.movementdisorders.org

Booth: Located on the Lower Level

The *Movement* Disorder Society 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. Visit our exhibit booth to learn more about MDS and membership opportunities.

TREMOR ACTION NETWORK

P.O. Box 5013
Pleasanton, CA 94566-0513
USA
Telephone: +1 925-462-0111
Fax: +1 925-369-0485
Web site: www.tremoraction.org

Table #: 9

TREMORACTION.ORG connects the bench to Tremor patients through awareness, advocacy, and research. Please stop by our booth to discuss the services we provide. TAN Spikes and Spasms newsletter, DVD, and informative resources are available. Visit our Web site at www.tremoraction.org.

UCB INC.

1950 Lake Park Drive
Smyrna, GA 30080
USA
Telephone: +1 770-970-8788
Fax: +1 770-970-8991
Web site: ucb-group.com

Booth #: 413, 312

UCB, Inc., with U.S. headquarters in Smyrna, Georgia, is a leading biopharmaceutical company, specializing in the fields of central nervous system disorders, allergy and respiratory disease, immune and inflammatory disorders and oncology. Please visit our booth to learn more about our products.

EXHIBITOR DIRECTORY

VALEANT PHARMACEUTICALS

One Enterprise
Aliso Viejo, CA 92656
USA
Telephone: +1 949-461-6000
Fax: +1 949-461-6609
Web site: www.valeant.com

Booth #: 419

Valeant Pharmaceuticals International (NYSE: VRX) is a multi-national, specialty pharmaceutical company that develops, manufactures and markets a broad range of prescription and non-prescription pharmaceutical products primarily in the areas of neurology, dermatology and infectious disease. Please stop by the Valeant booth to learn more about our Parkinson's disease product line.

VERNALIS PHARMACEUTICALS INC.

1140 Headquarters Plaza
2nd Floor, West Tower
Morristown, NJ 07960
USA
Telephone: +1 973-867-5555
Fax: +1 973-867-5524
Web site: www.vernalis.com

Booth #: 425

Vernalis is a specialty bio-pharmaceutical company with two marketed products: Apokyn[®] (apomorphine hydrochloride injection) and Frova[®] (frovatriptan). The company has a broad development pipeline focused on Neurology and Central Nervous System disorders.

WE MOVE

204 W. 84th Street
New York, NY 10024
USA
Telephone: +1 212-875-8312
Fax: +1 212-875-8389
Web site: www.mdvu.org

Booth #: 127

WE MOVE is a not-for-profit organization providing Movement Disorder information and education to healthcare professionals, patients, and families. At www.mdvu.org, healthcare 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.

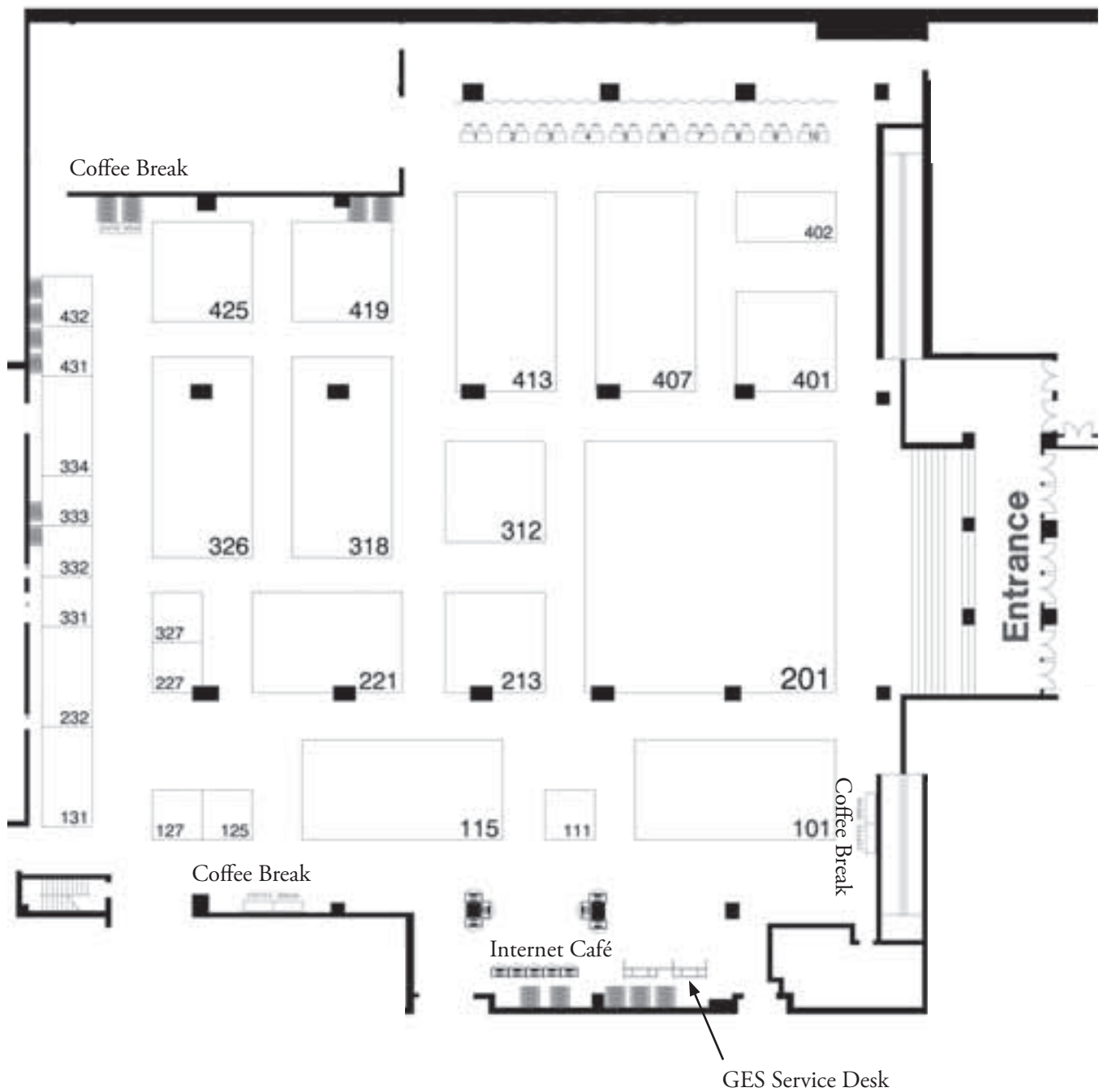
WILEY-BLACKWELL

111 River Street
Hoboken, NJ 07030 USA
Telephone: +1 201-748-6000
Fax: +1 201-748-6088
Web site: www.wiley.com

Table#: 2

Wiley-Blackwell, the scientific, technical, medical and scholarly publishing business of John Wiley & Sons, is the world's leading society publisher and offers peer-reviewed primary research and evidence-based medicine across thousands of online journals, books, reference works and databases.

EXHIBIT HALL FLOOR PLAN





C H A N G I N G T H E W O R L D

Imagine what it would be like if we could find a cure for cancer. Or an effective vaccination for HIV and AIDS. Or a medicine that could protect against heart disease or stroke.

Companies such as GlaxoSmithKline have already made breakthroughs that have saved millions of lives and hundreds of thousands more are living longer and living healthier.

So when we say our goal as a company is to help people **'do more, feel better, live longer,'** it means a lot more than just another advertising slogan or corporate mission statement.

The work we've done in the past has led to some of today's most effective treatments; the research we do now and in the future could find the new medicines for tomorrow's cures.

GUIDED POSTER TOURS

GUIDED POSTER TOUR 1

Dystonia

Tuesday, June 24, 2008

Northeast Exhibit Hall, Lower Level, Hilton Chicago

12:30 - 14:00

Poster numbers (475-494)

- 475 Plasticity of sensorimotor circuits in patients with SWEDDs resembles the pattern seen in dystonia and differs from Parkinson's disease
P. Schwingenschuh, D. Ruge, C. Terranova, S.A. Schneider, P. Mir, J.C. Rothwell, K.P. Bhatia, M.J. Edwards (London, United Kingdom)
- 476 Simple and complex hand movements in patients with writer's cramp: An event-related fMRI study
P. Havránková, R. Jech, N.D. Walker, G. Operto, J. Vymazal, E. Ruzicka (Prague, Czech Republic)
- 477 Premotor-motor inhibition exhibits task-specificity in patients with focal hand dystonia
S. Pirio Richardson, S. Beck, B. Bliem, M. Hallett (Albuquerque, NM)
- 478 Gray matter abnormalities in spasmodic dysphonia
K. Simonyan, C.L. Ludlow (Bethesda, MD)
- 479 Indications and results for globus pallidus internus (GPI) stimulation in perinatal hypoxic injury
N. Burger, F. Vergani, L. Cif, B. Biolsi, H. El Fertit, S. Gil Robles, X. Vasques, P. Coubes (Montpellier, France)
- 480 Myeloradiculopathy secondary to cervical dystonia
D. Riicard, E. Roze, J.-F. Lepeintre, S. Thobois, M. Anheim, A. Elbaz, D. Grabli, S. Leu, J. Xie, S. Yaici, C. Tranchant, C. Mazel, P. Burbaud, P. Krack, P. Pollak, E. Broussolle, M. Vidailhet (Paris, France)
- 481 Physiology of the subthalamic nucleus in patients with primary dystonia
L.E. Schrock, S. Shimamoto, J.L. Ostrem, R.S. Turner, P.A. Starr (San Francisco, CA)
- 482 A phase II, double blind randomised controlled crossover trial of dronabinol for the treatment of cervical dystonia
C. Zadikoff, P. Wadia, A. Asante, S.H. Fox (Toronto, ON, Canada)
- 483 Neurophysiological evidence for cerebellar dysfunction in primary focal dystonia
J.T.H. Teo, B.P.C. van de Warrenburg, S.A. Schneider, J.C. Rothwell, K.P. Bhatia (London, United Kingdom)
- 484 Clinical course of DYT1 dystonia patients treated with deep brain stimulation: A three to ten year-follow-up
L. Cif, X. Vasques, V. Gonzalez, S. Gavarini, B. Biolsi, G. Collod-Beroud, H. Elfertit, S. Gil Robles, P. Coubes (Montpellier, France)
- 485 Bilateral pallidal stimulation in generalised dystonia related to post-anoxic birth injury: Multicentre study
M. Vidailhet, C. Lagrange, M.-L. Welter, P. Krack, P. Krystkowiak, P. Burbaud, J. Xie, V. Fraix, D. Grabli, S. Thobois, P. Cornu, S. Navarro, A.-L. Benabid, S. Chabardes, E. Seigneuret, S. Blond, E. Cuny, P. Mertens, A. Destee, E. Broussolle, P. Pollak (Paris, France)
- 486 The syndrome of cervical dystonia-cerebellar ataxia (DYTCA): Dystonia without loss of cortical inhibition
P. Talelli, B.P.C. van de Warrenburg, S.A. Schneider, M.J. Edwards, P. Giunti, N.W. Wood, J.C. Rothwell, K.P. Bhatia (London, United Kingdom)
- 487 Differentiation of botulinum toxin non-responders in idiopathic cervical dystonia utilizing diffusion tensor imaging
J.M. Johnson, J. Boyd, D.D. Duane, C. Filippi (Burlinton, VT)
- 488 Association of focal dystonia and a common SNP in the DYT1 gene
N. Brüggemann, N. Kock, A. Rakovic, I. König, J. Hagenah, A. Schmidt, K. Lohmann, A. Münchau, E. Altenmüller, H.-C. Jabusch, H. Siebner, C. Klein (Lubeck, Schleswig-Holstein, Germany)
- 489 Parkinsonism with dystonia caused by the illicit use of ephedrone
M. Selikhova, L. Fedoryshyn, Y. Matviyenko, I. Komnatska, M. Kyrlychuk, L. Krolicki, A. Friedman, A. Taylor, H.R. Jager, A. Lees, Y. Sanotsky (London, United Kingdom)
- 490 Dystonia and progressive supranuclear palsy: A closer relation than expected
I. Avelés-Olmos, E. López-Valdes, S. Fanjul, M. Toledo, M.D. Castro, V. Sánchez-Cruz (Leganes, Spain)
- 491 Temporal discrimination threshold (TDT) and spatial discrimination threshold (SDT) – comparing endophenotypes in adult-onset primary torsion dystonia (AOPTD)
D. Bradley, R. Whelan, R. Walsh, R. Reilly, M. Hutchinson (Dublin, Ireland)
- 492 Adult onset dystonia can cause tremulous pseudoparkinsonism and is one cause of SWEDDs: Clinical description of 30 cases
P. Schwingenschuh, C. Terranova, F. Carrillo, S. Schneider, G. Kagi, M.J. Edwards, L. Silveira-Moriyama, J. Dickson, A.J. Lees, P. Mir, N.P. Quinn, K.P. Bhatia (London, United Kingdom)
- 493 Hyperactivation of the putamen during a discrimination task in unaffected relatives of patients with familial primary torsion dystonia
R. Whelan, D. Bradley, R. Walsh, R.B. Reilly, M. Hutchinson (Dublin, Ireland)
- 494 Behavioural and neurophysiological effects of proprioceptive training in musician's dystonia
K. Rosenkranz, K. Butler, C. Cordivari, A. Lees, A. Williamon, J.C. Rothwell (London, United Kingdom)

GUIDED POSTER TOUR 2

Parkinson's disease: Clinical Trials

Tuesday, June 24, 2008

Northeast Exhibit Hall, Lower Level, Hilton Chicago

12:30 - 14:00

Poster numbers (582-601)

- 582 Unified dyskinesia rating scale: Presentation and clinimetric profile
C.G. Goetz, J.G. Nutt, G.T. Stebbins (Chicago, IL)
- 583 Parkwatch: Analysis of response fluctuations in Parkinson's disease using a digital wrist watch
P.C.G. Nijssen (Tilburg, Netherlands)
- 584 Effect of investigator perception of treatment efficacy on outcome measures in a clinical trial of neuroprotective agents in Parkinson disease
O. Suchowersky, P. Huang, R. Elble, W. Weiner (Calgary, AB, Canada)

GUIDED POSTER TOURS

- 585 The PARS study: Recruitment of a cohort at risk for Parkinson disease
D. Jennings, A. Siderowf, M. Stern, K. Marek, PARS Study Investigators (New Haven, CT)
- 586 ADAGIO: A prospective, double-blind, delayed-start study to examine the potential disease-modifying effect of rasagiline in early Parkinson's disease (PD)
C.W. Olanow, O. Rascol, for the ADAGIO Investigators (New York, NY)
- 587 PROUD: The impact of early vs. delayed treatment with pramipexole on new onset Parkinson's disease
A.H. Schapira, H.H. Hsu, K. Scrine, M.F. Gordon, K.L. Marek (London, United Kingdom)
- 588 Pardopruxon (SLV308) in patients with early stage Parkinson's disease – a double-blind, placebo-controlled, multi-center study by the Bruegel study group
J. Bronzova, C. Sampaio, R.A. Hauser, A. Lang, O. Rascol, A. Theeuwes, S. van de Witte, G. van Scharrenburg (Weesp, Netherlands)
- 589 A double-blind, randomised, placebo-controlled trial to investigate the efficacy and safety of nebicapone in levodopa-treated Parkinson's disease patients with motor fluctuations
J.J. Ferreira, O. Rascol, W. Poewe, C. Sampaio, F. Rocha, T. Nunes, L. Almeida, P. Soares-da-Silva, Nebicapone 202 Study Group (Lisbon, Portugal)
- 590 Safety of istradefylline as adjunctive therapy in Parkinson's disease: Pooled analysis of 5 placebo-controlled 12- to 16-week studies
J. Williams, R. Ballerini, N.M. Sussman, K. Allenby, A. Mori (Princeton, NJ)
- 591 Duodenal levodopa infusion for advanced Parkinson's disease: 30-month treatment outcome
F. Mancini, M. Canesi, G. Pezzoli, R. Zangaglia, C. Pacchetti, M. Zibetti, L. Lopiano, M. Dal Fante, L. Manfredi, A. Antonini (Milan, Italy)
- 592 Transdermal delivery of a levodopa prodrug; a pilot clinical trial
M. Kushnir, A. Yaar, A. Reichman, E. Heldman (Rehovot & Ness Ziona, Israel)
- 593 Long-term improvement of motor fluctuations and health-related quality of life with levodopa/carbidopa gel
P.L.A. Odin, H. Honig, T. Fox, A. Rüssmann, S. Leimbach, K. Fox (Bremerhaven, Germany)
- 594 Comparison of adjunctive ropinirole 24-hour prolonged release and ropinirole immediate release in patients with advanced Parkinson's disease: A per-protocol analysis of the PREPARED study
A.H.V. Schapira, F. Stocchi, B. Hunter, L. Giorgi (London, United Kingdom)
- 595 Long-term safety and tolerability of transdermal rotigotine in advanced Parkinson's disease
W. Poewe, O. Rascol, N. Quinn, E. Tolosa, W.H. Oertel, N. Giladi, B. Boroojerdi (Innsbruck, Austria)
- 596 The long-term antidyskinetic effect of amantadine therapy in Parkinson's disease patients
E. Wolf, K. Seppi, R. Katzenschlager, G. Hochschorner, G. Ransmayr, P. Schwingenschuh, I. Kloiber, D. Haubenberger, W. Poewe (Innsbruck, Tirol, Austria)
- 597 Best medical therapy vs. deep brain stimulation for PD: Six month results from a multi-site randomized trial
F.M. Weaver, VA CSP #468/NINDS Study Group (Hines, IL)
- 598 5-year update of the safety and efficacy of unilateral intrastriatal implantation of Spheramine®
R.L. Watts, N.P. Stover, A. Freeman, M. DeLong, R.A.E. Bakay, E. Reissig (Birmingham, AL)
- 599 Treating festinating speech with altered auditory feedback in Parkinson's disease – the first report of a clinical trial
E.Q. Wang, L. Verhagen Metman (Chicago, IL)
- 600 Randomised controlled trial of memantine for dementia associated with Parkinson's disease
I. Leroi, R. Overshott, E. Daniai, E.J. Byrne, A. Burns (Manchester, Lancashire, United Kingdom)
- 601 Design of a randomized, placebo-controlled trial of pramipexole in patients with Parkinson's disease and depressive symptoms
P. Barone, A.H.V. Schapira, C.D. Debieuvre, D. Massey (Napoli, Italy)

GUIDED POSTER TOUR 3

Genetics

Wednesday, June 25, 2008

Southeast Exhibit Hall, Lower Level, Hilton Chicago

12:30 - 14:00

Poster numbers (83-102)

- 83 A novel function of a UCHL1 polymorphic variant as a potent antioxidant: Implications in Parkinson's disease
E. Kyratzi, M. Pavlaki, L. Stefanis (Athens, Greece)
- 84 New phenotypic presentation of benign hereditary chorea caused by a novel mutation in the thyroid transcription factor-1 (TTF-1) gene
A. Glik, I. Vuillaume, D. Devos, R. Inzelberg (Ramat Gan, Israel)
- 85 A heterozygous frameshift mutation in the PRKRA (DYT16) gene associated with generalized dystonia in a German patient
A. Djarmati, P. Seibler, B. Langpap, J. Hagenah, A. Schmidt, N. Brüggemann, H. Siebner, H.-C. Jabusch, E. Altenmüller, A. Münchau, K. Lohmann, C. Klein (Luebeck, Germany)
- 86 The spectrum of dystonia in Mohr-Tranebjaerg syndrome in three Australian kindreds
K.L. Parratt, K. Ng, D.B. Rowe, J.G.L. Morris, M.W. Hayes, C.M. Sue, L. Tranebjaerg, V.S.C. Fung (Sydney, NSW, Australia)
- 87 Familial hand dystonia with DYT1 mutation and the effect of thalamotomy
M.J. Kim, S.J. Chung, S.R. Kim, J.K. Lee, M.C. Lee (Seoul, Korea)
- 88 α -synuclein (SNCA) variants and Parkinson's disease susceptibility
S.M. Goldman, C.M. Tanner, M. Korell, G.S. Bhudhikanok, B. Schuele, J.A. Hoppin, L. Sterling, D.M. Umbach, G.W. Ross, A. Blair, J.W. Langston, F. Kamel (Sunnyvale, CA)
- 89 Cigarette smoking, CYP2A6 gene variants, and Parkinson disease: A case-control study
M.F. Facheris, N.K. Schneider, T.G. Lesnick, M. de Andrade, L.J. Kost, J.M. Cunningham, W.A. Rocca, D.M. Maraganore (Rochester, MN)
- 90 Assessment of Parkinson's disease in the Swedish Twin Registry: Lack of concordance in twins over 50 years of age
H. Widner, K. Wirdefeldt, M. Gatz, S.L. Bakaysa, A. Fiske, M. Flensburg, G.M. Petzinger, M.F. Lew, M. Welsh, N.L. Pedersen (Lund, Sweden)

GUIDED POSTER TOURS

- 91 Clinical characteristics of patients with Parkinson's disease who are carriers of severe GBA mutations
N. Giladi, A. Orr-Urtreger, Z. Gan-Or, O. Moore, A. Bar Shira, T. Gurevich (Tel Aviv, Israel)
- 92 Glucocerebrosidase mutations are an important risk factor for Lewy body inclusions in Alzheimer's disease patients of Ashkenazi Jewish ancestry
L.S. Libow, P.G. Frisina, L. Edelman, K. Hirschhorn, M. Grace, S. Scott, C. Tarshish, V. Haroutunian (New York, NY)
- 93 Longer REP1 repeat lengths are associated with both essential tremor (ET) and Parkinson disease (PD)
C.M. Testa, L. Miyatake, M. Wilson, M. Bouzyk, S. Factor (Atlanta, GA)
- 94 Clinical characteristics of PD patients carrying homozygotic and compound heterozygotic GBA mutations
E.L. Ash, A. Bar-Shira, N. Giladi, O. Moore, A. Orr-Urtreger, T. Gurevich (Tel Aviv, Israel)
- 95 Genotype distribution in familial and sporadic spastic paraplegia
L. Schöls, C. Beetz, K. Karle, S. Klebe, S. Klimpe, S. Otto, R. Schüle (Tübingen, Germany)
- 96 AAV2-neurturin (CERE-120) for Parkinson's disease: 24-month follow-up from the phase I clinical trial
W.J. Marks, Jr., J.L. Ostrem, L. Verhagen, P.A. Starr, P.S. Larson, R.A.E. Bakay, C.W. Olanow, R.T. Bartus (San Francisco, CA)
- 97 Functional studies in subjects carrying glucocerebrosidase (GBA) mutations
O. Goker-Alpan, G. Lopez, K. Berman, E. Sidransky (Bethesda, MD)
- 98 Mood and anxiety disorders in an Ashkenazi Jewish (AJ) population with LRRK2 G2019S Parkinson's disease (PD)
V.L. Shanker, M. Groves, S.M. Hailpern, L.J. Ozelius, R. Saunders-Pullman, A. Hunt, M. Sethi, J. Soto-Valencia, S.B. Bressman (New York, NY)
- 99 Expanded study of NURR1 gene expression in patients with Parkinson's disease
W. Le, T. Pan, M. Huang, P. Xu, X. Zhang, J. Jankovic (Houston, TX)
- 100 TDP-43 proteinopathy in progressive supranuclear palsy (PSP)
L. Massey, T. Lashley, S.S. O'Sullivan, R. Phadke, L. Moriyama-Silveira, A. Lees, J. Holton, T. Revesz (London, United Kingdom)
- 101 Familial cortical myoclonic tremor with epilepsy: A new Italian pedigree linked to chromosome 2p11.1-q12.2
A. Suppa, F. Brancati, M. Marianetti, G. Barrano, C. Mina, A. Pizzuti, G. Sideri, A. Berardelli (Rome, Italy)
- 102 Distinct mechanisms of MPTP resistance revealed by transcriptome mapping in mouse striatum
R. Pattarini, Y. Rong, C. Qu, J.I. Morgan (Memphis, TN)

GUIDED POSTER TOUR 4

Parkinson's disease: Cognition

Wednesday, June 25, 2008

Southeast Exhibit Hall, Lower Level, Hilton Chicago

12:30 - 14:00

Poster numbers (257-276)

- 257 Prevalence and profile of mild cognitive impairment in early, untreated Parkinson's disease – a community-based study
D. Aarsland, K. Bronnick, J.P. Larsen, O.B. Tysnes, G. Alves (Stavanger, Norway)
- 258 Cognitive impairment in early and untreated Parkinson's disease: Associations with cerebrospinal fluid levels of β -amyloid1-42, total tau, and phosphorylated (181p) tau protein
D. Aarsland, E. Mulugeta, K. Bronnick, O.B. Tysnes, J.P. Larsen, G. Alves (Stavanger, Norway)
- 259 Clinical factors associated with age-related variability in cognitive test performance in Parkinson's disease
J.R. Williams, S.M. Testa, J. Brandt, L. Marsh (Baltimore, MD)
- 260 Comparison of cognitive function in early-onset versus late-onset Parkinson's disease
I. Galazky, C.I. Higginson, V.L. Wheelock, C.T.E. Pappas, K.A. Sigvardt (Magdeburg, Germany)
- 261 Cognitive changes without dementia in PD: Evidence for neuropsychological subtypes
J.B. Leverenz, M. Glisky, C.P. Zabetian, D.W. Tsuang, A. Griffith, P. Agarwal, K. Olson, J. Shaw, S. Millard, G.S. Watson (Seattle, WA)
- 262 Relevance of new Movement Disorders Task Force recommendations for Parkinson Disease Dementia diagnosis (PD-D).
M. Kiesmann, J.-B. Chanson, T. Vogel, I.-J. Namer, G. Kaltenbach, M. Berthel (Strasbourg, France, Metropolitan)
- 263 Impact of mild cognitive deficits on daily functioning in Parkinson's disease
E. Rosenthal, L. Brennan, J. Milber, H. Hurtig, D. Weintraub, A. Siderowf (Philadelphia, PA)
- 264 Impaired curve negotiation in drivers with Parkinson's disease
E.Y. Uc, M. Rizzo, E. Dastrup, S.W. Anderson, J. Sparks, R.L. Rodnitzky, J.D. Dawson (Iowa City, IA)
- 265 Switching between motor representations in Parkinson's disease – an fMRI study
R.C. Helmich, E. Aarts, F.P. de Lange, B.R. Bloem, I. Toni (Nijmegen, Netherlands)
- 266 Brain metabolic pattern (BMP) of cognitive decline in Parkinson's disease (PD)
M.C. Rodriguez-Oroz, D. Garcia Garcia, P. Clavero, I. Lamet, C. Irurzun, P. Martinez-Lage, J. Arbizu, E. Prieto, J.A. Obeso (Pamplona, Navarra, Spain)
- 267 Relationship of cortical Pittsburgh compound B (PIB) binding and clinical features in Parkinson disease dementia
M.A. Burack, M.C. Campbell, E.R. Foster, N. Golchin, J. Hartlein, T. Hershey, J.S. Perlmutter (St. Louis, MO)
- 268 Relationship between neuropsychological functioning and PIB binding in Parkinson disease
M.C. Campbell, M.A. Burack, E.R. Foster, N. Golchin, A.N. Goulding, J. Hartlein, T. Hershey, J.S. Perlmutter (St. Louis, MO)

GUIDED POSTER TOURS

- 269 Combined use of 3T proton spectroscopy, DTI and VBM for capturing cortical changes in Parkinson's disease with cognitive dysfunction: A preliminary study
J. Pagonabarraga, G. Llebaria, J. Kulisevsky, B. Pascual-Sedano, M. Martinez-Corral, B. Gomez-Anson, R. Rotger, J. Acosta-Cabronero, P.J. Nestor, J. Rusalleda, M. Delfino (Barcelona, Spain)
- 270 Implicit motor sequence learning in patients with Parkinson's disease depends on the stage of disease
M.A. Stephan, S. Weber Zaugg, A. Kaelin-Lang (Bern, Switzerland)
- 271 Effect of dopaminergic therapy on the fronto-striatal patterns of activity observed in patients with Parkinson's disease during the execution of a cognitive task
L. Monetta, T. Jubault, A. Strafella, A.-L. Lafontaine, M. Panisset, A. Ptito, C. Gauthier, O. Monchi (Montreal, QC, Canada)
- 272 Changes in cerebral glucose metabolism in patients with Parkinson's disease dementia after cholinesterase inhibitor therapy
S.W. Yong, I.S. Joo, P.H. Lee (Suwon, Republic of Korea)
- 273 Medication improves executive function in tremor-dominant subtype of idiopathic Parkinson disease
A.J. Hood, S.C. Amador, M.C. Schiess, A.B. Sereno (Houston, TX)
- 274 White matter disease as a risk factor for memory decline following deep brain stimulation surgery
M. Sharland, J. Bobholz, S. Winstanley, S. Gremley, D. Lee, B. Hiner, K. Blindauer, S. Hung, B. Kopell (Milwaukee, WI)
- 275 A longitudinal study of cognitive dysfunction in patients affected by Parkinson's disease with and without freezing of gait
M. Amboni, A. Cozzolino, K. Longo, M. Picillo, P. Barone (Napoli, Italy)
- 276 Visual hallucinations in Parkinson's disease, preliminary fMRI results
A.M. Meppelink, B.M. de Jong, R. Renken, K.L. Leenders, R. Jacobs, T. van Laar (Groningen, Netherlands)
- 319 Immediate and sustained effect produced by changing the stimulated electrode contact in STN DBS for PD
I. Martinez-Torres, L. Zrinzo, S. Perez-Hoyos, E. Tripoliti, M.I. Hariz, P. Limousin (London, United Kingdom)
- 320 Long-term gait deterioration after bilateral STN DBS is not due to the natural progression of Parkinson's disease
C.E. Martin, I. Barnaure, R.L. Alterman, M. Tagliati (New York, NY)
- 321 The effect of 60 Hz STN-DBS on gait and speech in patients with Parkinson's disease
H. Brozova, I. Barnaure, R.L. Alterman, M. Tagliati (New York, NY)
- 322 The effect of subthalamic nucleus deep brain stimulation on autonomic nervous system in Parkinson's disease
K. Sumi, T. Obuchi, T. Otaka, T. Kano, K. Kobayashi, H. Oshima, C. Fukaya, T. Yamamoto, Y. Katayama (Tokyo, Japan)
- 323 Chronic subthalamic deep brain stimulation improves pain in Parkinson disease
H.-J. Kim, S.-H. Paik, J.-W. Cho, H.-J. Kim, B.S. Jeon (Goyang-si, Gyeonggi-do, Korea)
- 324 PPN-DBS effects on non-motor functions in Parkinson's disease patients: Two years follow-up
M. Pierantozzi, A. Stefani, A. Peppe, R. Ceravolo, L. Brusa, A. Romigi, S. Galati, P. Stanzione (Rome, Italy)
- 325 Experience with MRI safety and DBS: Data from the National Parkinson Foundation Centers of Excellence
M. Tagliati, J. Jankovic, A.M. Koss, F. Pagan, M.S. Okun, National Parkinson Foundation DBS Working Group (New York, NY)
- 326 Occipital pseudoaneurysm as a complication of extension channel placement for DBS in Parkinson's disease
A. Castrioto, N. Tambasco, C. Menichetti, M. Hamam, V. Rossi, C. Castrioto, P. Calabresi, A. Rossi (Perugia, Italy)
- 327 A simple non-invasive method to detect lead fractures in DBS
F. Alesch, H. Lanmueller (Vienna, Austria)
- 328 Safety and efficacy of deep brain stimulation in mildly demented Parkinson's disease patients. A multiple case study
C.M. Buetefisch, M. Parsons, M.W. Haut, S.R. Goldstein, D.M. Whiting, M.Y. Oh (Morgantown, WV)
- 329 Decisions regarding deep brain stimulation (DBS) for Parkinson's disease (PD) when hypersexuality co-exists
J. Bourke, M. Samuel, A. Costello, N. Hulse, C. Clough, R. Selway, K. Ashkan, J. Moriarty (London, United Kingdom)

GUIDED POSTER TOUR 5

Surgical Therapy

Thursday, June 26, 2008

Southeast Exhibit Hall, Lower Level, Hilton Chicago

12:30 - 14:00

Poster numbers (315-334)

- 315 The STN beta band profile in Parkinson's disease is stationary and shows prolonged attenuation after deep brain stimulation
H.M. Bronte-Stewart, C. Barberini, M. Miller Koop, B. Hill, J.M. Henderson, B. Wingeier (Stanford, CA)
- 316 Next generation deep brain stimulation therapy: Modeling field steering in the brain with segmented electrodes
G.C. Miyazawa, D. Stone, G.F. Molnar (Minneapolis, MN)
- 317 Three dimensional visualization of the subthalamic nucleus
O.S. Klepitskaya, Z. Kim, M.D. Richardson, S.G. Ojemann (Aurora, CO)
- 318 Subthalamic neuronal activity is altered by contralateral subthalamic deep brain stimulation in Parkinson disease
H.C. Walker, B.L. Guthrie, S.L. Guthrie, N.P. Stover, D.G. Standaert, R.L. Watts (Birmingham, AL)
- 320 Long-term gait deterioration after bilateral STN DBS is not due to the natural progression of Parkinson's disease
C.E. Martin, I. Barnaure, R.L. Alterman, M. Tagliati (New York, NY)
- 321 The effect of 60 Hz STN-DBS on gait and speech in patients with Parkinson's disease
H. Brozova, I. Barnaure, R.L. Alterman, M. Tagliati (New York, NY)
- 322 The effect of subthalamic nucleus deep brain stimulation on autonomic nervous system in Parkinson's disease
K. Sumi, T. Obuchi, T. Otaka, T. Kano, K. Kobayashi, H. Oshima, C. Fukaya, T. Yamamoto, Y. Katayama (Tokyo, Japan)
- 323 Chronic subthalamic deep brain stimulation improves pain in Parkinson disease
H.-J. Kim, S.-H. Paik, J.-W. Cho, H.-J. Kim, B.S. Jeon (Goyang-si, Gyeonggi-do, Korea)
- 324 PPN-DBS effects on non-motor functions in Parkinson's disease patients: Two years follow-up
M. Pierantozzi, A. Stefani, A. Peppe, R. Ceravolo, L. Brusa, A. Romigi, S. Galati, P. Stanzione (Rome, Italy)
- 325 Experience with MRI safety and DBS: Data from the National Parkinson Foundation Centers of Excellence
M. Tagliati, J. Jankovic, A.M. Koss, F. Pagan, M.S. Okun, National Parkinson Foundation DBS Working Group (New York, NY)
- 326 Occipital pseudoaneurysm as a complication of extension channel placement for DBS in Parkinson's disease
A. Castrioto, N. Tambasco, C. Menichetti, M. Hamam, V. Rossi, C. Castrioto, P. Calabresi, A. Rossi (Perugia, Italy)
- 327 A simple non-invasive method to detect lead fractures in DBS
F. Alesch, H. Lanmueller (Vienna, Austria)
- 328 Safety and efficacy of deep brain stimulation in mildly demented Parkinson's disease patients. A multiple case study
C.M. Buetefisch, M. Parsons, M.W. Haut, S.R. Goldstein, D.M. Whiting, M.Y. Oh (Morgantown, WV)
- 329 Decisions regarding deep brain stimulation (DBS) for Parkinson's disease (PD) when hypersexuality co-exists
J. Bourke, M. Samuel, A. Costello, N. Hulse, C. Clough, R. Selway, K. Ashkan, J. Moriarty (London, United Kingdom)
- 330 DBS in Tourette syndrome (TS): A two-year open label experience in two patients
J.L. Juncos, R. Gross, M.R. DeLong (Atlanta, GA)
- 331 Influence of thalamic DBS on gait in patients with advanced essential tremor
A. Fasano, F. Rose, J. Volkmann, G. Deuschl, J. Herzog (Kiel, Germany)
- 332 Optimal pallidal stimulation frequency for dystonia may vary with age
M. Tagliati, C.E. Martin, R.L. Alterman (New York, NY)
- 333 Long-term outcome predictors of pallidal stimulation in patients with primary dystonia: The role of disease duration and speech involvement
I.U. Isaias, J. Volmann, R.L. Alterman, M. Mehdorn, M. Pinsker, R. Reese, G. Deuschl, M. Tagliati (New York, NY)

GUIDED POSTER TOURS

- 334 How should we measure outcome of deep brain stimulation (DBS) in childhood dystonia?
H. Gimeno, R. Mahoney, K. Tustin, S. Jupp, T. Kerr, M. Kaminska, R. Selway, J.-P. Lin (London, United Kingdom)

GUIDED POSTER TOUR 6

Neuropharmacology

Thursday, June 26, 2008

Southeast Exhibit Hall, Lower Level, Hilton Chicago

12:30 - 14:00

Poster numbers (226-245)

- 226 Neurological effects with recombinant human erythropoietin in Friedreich's ataxia
S.M. Boesch, B.N. Sturm, S. Hering, B. Scheiber-Mojdehkar, H. Steinkellner, H. Goldenberg, W. Poewe (Innsbruck, Austria)
- 227 A detailed behavioural, neurochemical and histological characterization of an α -synuclein transgenic mouse line as a model of Parkinson's disease
T.K. Murray, S.N. Mitchell, J. Cooper, K.R. Bales, C.V. Cella, D.L. Czilli, P.J. Collins, C. Evans, M.A. Ward, K.M. Merchant, M.J. O'Neill (Windlesham, Surrey, United Kingdom)
- 228 Motor and non-motor behavioral impairments associated to decreased expression of tyrosine hydroxylase after intracerebral administration of lactacystin
M.S. García-Gutiérrez, E. García-Payá, C. de Cabo, M. Galindo, C. Leiva, J. Manzanares (San Juan de Alicante, Spain)
- 229 Administration of the cannabinoid receptor agonist CP-55,940 reduced motor impairment and tyrosine hydroxylase expression loss in 6-hydroxydopamine-lesioned mice
E. García-Payá, M.S. García-Gutiérrez, M. Alvarez-Sauco, M. Galindo, C. Barcia, M.T. Herrero, C. Leiva, J. Manzanares (San Juan de Alicante, Alicante, Spain)
- 230 Gardenosides increase astrocyte's neuroprotective effect on dopaminergic neurons by inhibiting lipopolysaccharide-induced secretion of inflammatory factors
X.z. Li, L.-m. Bai, Y.-p. Yang, K.-y. Liu, C.-j. Mao, C.-f. Liu (Suzhou, China)
- 231 Glucocorticoid-mediated and cytokine-mediated inflammatory responses in MPTP-treated monkeys. Implications in the progressive degeneration process
M.-T. Herrero, C. Barcia, D. Aguado, M.A. Carrillo, V. de Pablos, E. Fernandez-Villalba (Murcia, Spain)
- 232 Migration of type A botulinum toxin in vivo is not related to the size of the toxin complex
A.M. Pickett (Wrexham, United Kingdom)
- 233 The relationship between exposure to aripiprazole and development of parkinsonism
L.L.L. Lua, L. Zhang (Sacramento, CA)
- 234 Efficacy of NT 201 (Xeomin®) in focal dystonia
W. Jost, S. Grafe, C. Georg (Wiesbaden, Germany)
- 235 Disabling alien limb phenomena improved with clonazepam and botulinum toxin injections
I.U. Haq, I. Malaty, H.H. Fernandez, M.S. Okun, R.R. Rodriguez (Gainesville, FL)
- 236 Case-control study of plasma uric acid in Parkinson's disease
F. Moisan, M.-A. Lorient, A. Le Floch, M. Vidailhet, I. Ceballos, J.-L. Perignon, C. Tzourio, A. Elbaz (Paris, France)
- 237 Striatal histone post-translational modifications in animal models of levodopa-induced dyskinesia
A.P. Nicholas, F.D. Lubin, P.J. Hallett, P. Vatter, P. Ravenscroft, E. Bezard, S. Zhou, S.H. Fox, J.M. Brotchie, J.D. Sweatt, D.G. Standaert (Birmingham, AL)
- 238 [¹¹C]raclopride PET imaging demonstrates correlation between correction of dopamine neurotransmission and behavioral recovery following gene therapy
T. Bjorklund, L. Leriche, N. Breyse, M.-C. Grégoire, T. Carlsson, F. Dollé, R.J. Mandel, N. Déglon, P. Hantraye, D. Kirik (Lund, Sweden)
- 239 Innovative options for the conservative treatment of Tourette syndrome (TS): The role of tetrabenazine (TBZ) on the basis of a selected cohort of 120 patients treated at the AIST Milan
M. Porta, M. Sassi, A. Brambilla, S. Defendi, D. Servello (Milan, Italy)
- 240 The role of dopaminergic medication doses in impulse control disorders (ICD) in Parkinson's disease (PD)
J. Jimenez-Shahed, K. Baker, A. Davidson, J. Jankovic (Houston, TX)
- 241 Coenzyme Q10 in Parkinson's disease (PD)
S.N. Siddiqui, C.C. Soundararajan, M. Manral, S. Vivekanandhan, M. Behari (New Delhi, India)
- 242 Does intolerance to a diagnostic acute L-dopa challenge in parkinsonian syndromes differentiate MSA from IPD?
S. Estévez, S. Perez-Lloret, M. Merello (Cdad. Aut. Bs. As., Argentina)
- 243 Glutamate neurotransmission in dyskinetic Parkinson's disease: An 11C-CNS 5161 PET study
I. Ahmed, A. Hammers, S. Bose, F. Turkheimer, G. Hotton, N. Quinn, D.J. Brooks (London, United Kingdom)
- 244 A pharmacokinetic-pharmacodynamic model for duodenal levodopa infusion
J. Westin, T. Willows, T. Groth, M. Dougherty, M. Karlsson, D. Nyholm, S. Pålhagen (Borlange, Sweden)
- 245 Applying the UK PD Brain Bank criteria to SWEDDs (scans without evidence of dopaminergic deficit)
V.K. Gontu, J. Birchall, N. Bajaj (Derby, United Kingdom)



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Chicago, IL, USA
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ABSTRACTS

POSTER SESSION 1

Tuesday, June 24, 2008 – 12:30 to 14:30

Southeast Exhibit Hall, Lower Level, Hilton Chicago

Poster Viewing: 9:00 to 17:00

Authors Present: 12:30 to 14:30

Poster Numbers: 1-438

Basic Science

Poster numbers 1-78

- 1 Programmed cell death-2 isoform 1 is ubiquitinated by parkin and increased in autosomal recessive Parkinson's disease
J. Fukae, S. Sato, K. Shiba, K. Sato, H. Mori, P.A. Sharp, Y. Mizuno, N. Hattori (Tokyo, Japan)
- 2 A zebrafish model of tauopathy
Q. Bai, E.A. Burton (Pittsburgh, PA)
- 3 Effects of partial 6-OHDA lesions on time preparation and response selection during the performance of a stimulus-response compatibility task in the rat
T.G. Hasbroucq, J. Hardouin, B. Burle, F. Vidal, N. Turle-Lorenzo, M. Amalric, C. Alain (Marseille, France)
- 4 Predominant release of lysosomal enzymes by microglia after LPS treatment revealed by proteomic studies
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- 5 Effects of transcranial random noise stimulation (tRNS) on cortical excitability
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- 6 Rasagiline is neuroprotective in a transgenic mouse model of multiple system atrophy
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- 7 A neuroprotective role of lysosomal enzyme cathepsin D against α -synuclein pathogenesis
L. Qiao, S. Hamamichi, K.A. Caldwell, G.A. Caldwell, S. Wilson, T. Yacoubian, Z.-l. Xie, L. Speake, R. Parks, D. Crabtree, S. Crimmins, Y. Uchiyama, Y. Zhou, L. Peng, Y. Lu, D.G. Standaert, K.C. Walls, J.J. Shacka, K. Roth, J. Zhang (Birmingham, AL)
- 8 D3 dopamine receptor antagonists prevent the development of L-DOPA-induced dyskinesia in Parkinson's disease
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- 9 Neuroprotective effect of the iron chelator desferoxamine on myelomonocytes from sporadic ALS patients subjected to hypoxic stress: Implications for therapy
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- 10 Striatal transplantation improves L-Dopa response in multiple system atrophy: Experimental evidence
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- 11 Stereotactic model of the electrical distribution within the internal globus pallidus during deep brain stimulation
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- 12 Localization and densities of PD associated proteins and cytokines after lipopolysaccharide (LPS) treatments
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A. Friedman, J. Galazka-Friedman, E.R. Bauminger, D. Dickson, Z. Wszolek (Warsaw, Poland)
- 14 Comparison of the sensitivity to MPTP and/or ovariectomy in heterozygous double mutated human α -synuclein mice
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- 15 A novel moderate-affinity metal binding ligand confers neuroprotection against 6-hydroxydopamine mediated chronic cell death and improves rotational behavior in a mouse model of Parkinson's disease
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- 24 Potential interactors of the familial Parkinson's disease protein LRRK2
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- 34 Striatal spine loss in parkinsonism: An early dopamine-dependent pathology in the MPTP-treated nonhuman primate model of Parkinson's disease
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- 35 Deep brain stimulation of the subthalamic nucleus: A neuroanatomical study on inhibitory interneurons of the basal ganglia in the rat 6-hydroxydopamine Parkinson model
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- 36 Serine 129 phosphorylation of α -synuclein induces unfolded protein response-mediated cell death
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- 37 Parkin dysfunction results in defective depolarization-induced exocytosis
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- 38 Lesions of the entopeduncular nucleus in rats restore deficient prepulse inhibition of the acoustic startle response induced by selective breeding, a model of Tourette syndrome
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- 39 Possible localization of DJ-1 on the plasma membrane in the mouse brain and cultured cell lines
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- 40 Brain uncoupling protein-2 mediates neuronal survival by leptin against mitochondrial dysfunction and ATP deficiency
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- 41 Subcellular localization of ATP13A2
F. Sato, S.-i. Kubo, S. Imai, Y. Mizuno, N. Hattori (Tokyo, Japan)
- 42 Distribution of LRRK2 protein in the central nervous system of human brain – pathological and normal brain
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- 43 Alpha-synuclein expression in LRRK2 mutant primary cells
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- 44 Axonal α -synuclein (α S) aggregates herald centripetal degeneration of cardiac sympathetic nerve in Parkinson disease (PD)
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- 45 Neuropathological characterization of naturally occurring autoantibodies against α -synuclein
F. Neff, F. Seitz, C. Binder, D. Besong Agbo, M. Bacher, P. Kahle, R. Dodel (Marburg, Hesse, Germany)
- 46 Neuroprotective potential of systemic inosine administration in the 6-hydroxydopamine model of Parkinson's disease
B. Terpstra, J. Lipton, C. Sortwell (Cincinnati, OH)
- 47 Modeling long-term STN deep brain stimulation in parkinsonian rats
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- 48 Stimulation genomics: Identifying functional polymorphisms modulating LTP and LTD in human cerebral cortex and implications for levodopa induced dyskinesia in Parkinson's disease (PD)
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- 49 Equivalent potency of Xeomin® and Botox®
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- 50 Influence of aging on motor function and striatum dopamine transporter of rats
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- 51 Study of mitochondrial function in a PSI-induced model of Parkinson's disease
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- 52 Developmental alterations of LRRK2 in the mouse brain
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- 55 Safinamide: Long-term treatment effects on dopamine metabolism in motor and non-motor regions of monkey brain
C. Caccia, L. Girola, P. Salvati, S. Rossetti, R. Anand (St. Moritz, Switzerland)
- 56 Safinamide: Modulation of dopaminergic and glutamatergic systems
C. Caccia, P. Salvati, S. Rossetti, R. Anand (St. Moritz, Switzerland)
- 57 Rapamycin rescues lactacystin-induced dopaminergic neuron injury in vivo
T. Pan, W. Xie, J. Jankovic, W. Le (Houston, TX)
- 58 Dopamine peroxidation: A colour reaction from human midbrain analyzed by mass spectrometry
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- 59 Gene transfer of pleiotrophin provides morphological neuroprotection and functional neurorestoration to the parkinsonian rat
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- 60 One-third of elderly men without a history of Parkinson's disease or dementia with Lewy bodies have Lewy pathology in the olfactory bulb
J.E. Duda, J.V. Noorigian, H. Petrovitch, L.R. White, G.W. Ross (Philadelphia, PA)
- 61 Xeomin® is stable without refrigeration and is not affected by short-term temperature stress
S. Grein, G.J. Mander, H.V. Taylor (Frankfurt am Main, Germany)
- 62 Effect of different patterns of GPI DBS on bradykinesia in the non-human primate model of Parkinson's disease
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- 63 Supranormal gait in mice following brief exposure to isoflurane
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- 64 Sex differences in the MPTP mouse model of Parkinson's disease
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- 65 Dopamine regulates alpha-synuclein expression through epigenetic mechanism
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- 66 The neuroprotective effects of stable overexpression of mitochondrial uncoupling protein 5 (UCP5) in neuronal cells exposed to MPP+ and dopamine-induced cytotoxicity
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- 67 Association of olfactory dysfunction in the elderly with Lewy pathology in the olfactory bulb
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- 68 The potential of minocycline for neuroprotection in nigra of zitter mutant rat
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- 69 Individual dopaminergic neurons show raised iron levels in Parkinson disease
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- 70 Diminished tyrosine hydroxylase positive neurons in the substantia nigra in canine multiple system degeneration
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- 71 Altered glutamate (AMPA) and dopamine (D2) receptor expression with treadmill exercise in the 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine-lesioned mouse model of basal ganglia injury
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- 72 Differential modulation of motorcortical plasticity and excitability in early and late phases of human motor learning
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- 73 Pigment epithelium-derived factor is neuroprotective in in vitro models of Parkinson's disease
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- 74 Putative neuroanatomical substrates underlying mood disorders in Parkinson's disease
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- 75 Lewy pathology in the olfactory bulb is associated with decreased neuron density in the substantia nigra
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- 78 Incidental Lewy bodies in various neurodegenerative disorders
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- 81 Nonclinical and clinical development of ProSavin, an EIAV-based gene therapy for Parkinson's disease
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- 82 Modulation of the K⁺ channel Kir2.3 by a tissue-specific viral vector as a potential new gene therapy for Parkinson's disease
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E. Kyrtzi, M. Pavlaki, L. Stefanis (Athens, Greece)
- 84* New phenotypic presentation of benign hereditary chorea caused by a novel mutation in the thyroid transcription factor-1 (TTF-1) gene
A. Glik, I. Vuillaume, D. Devos, R. Inzelberg (Ramat Gan, Israel)
- 85* A heterozygous frameshift mutation in the PRKRA (DYT16) gene associated with generalized dystonia in a German patient
A. Djarmati, P. Seibler, B. Langpap, J. Hagenah, A. Schmidt, N. Brüggemann, H. Siebner, H.-C. Jabusch, E. Altenmüller, A. Münchau, K. Lohmann, C. Klein (Luebeck, Germany)

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- 86* The spectrum of dystonia in Mohr-Tranebjær syndrome in three Australian kindreds
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- 87* Familial hand dystonia with DYT1 mutation and the effect of thalamotomy
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- 88* α -synuclein (SNCA) variants and Parkinson's disease susceptibility
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- 89* Cigarette smoking, CYP2A6 gene variants, and Parkinson disease: A case-control study
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- 90* Assessment of Parkinson's disease in the Swedish Twin Registry: Lack of concordance in twins over 50 years of age
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- 91* Clinical characteristics of patients with Parkinson's disease who are carriers of severe GBA mutations
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- 92* Glucocerebrosidase mutations are an important risk factor for Lewy body inclusions in Alzheimer's disease patients of Ashkenazi Jewish ancestry
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- 93* Longer REP1 repeat lengths are associated with both essential tremor (ET) and Parkinson disease (PD)
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- 94* Clinical characteristics of PD patients carrying homozygotic and compound heterozygotic GBA mutations
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- 95* Genotype distribution in familial and sporadic spastic paraplegia
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- 96* AAV2-neurturin (CERE-120) for Parkinson's disease: 24-month follow-up from the phase I clinical trial
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- 100* TDP-43 proteinopathy in progressive supranuclear palsy (PSP)
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- 101* Familial cortical myoclonic tremor with epilepsy: A new Italian pedigree linked to chromosome 2p11.1-q12.2
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- 102* Distinct mechanisms of MPTP resistance revealed by transcriptome mapping in mouse striatum
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- 108 Polymorphism of the angiotensin converting enzyme gene increased risk of levodopa-induced psychosis in Parkinson's disease for older female
J.-J. Lin, K.-C. Yueh (Nantou, Taiwan)
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- 110 Role of D216H polymorphism in DYT1 Italian patients
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- 111 Spinocerebellar ataxia type12 was not found in Korean parkinsonian patients
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- 112 Novel mutations associated with autosomal recessive form of spastic paraplegia with thin corpus callosum
S. Bohlega, S.M. Wakil, H. Murad, B. Baz, R. AlAmr, S. Yamani, S.M. Al Wadaee, B.F. Meyer (Riyadh, Saudi Arabia)
- 113 The contribution of missense mutations in parkin to the molecular etiology of Parkinson's disease in South Africa
S. Bardien, R. Keyser, D. Lombard, J. Carr (Cape Town, South Africa)

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- 114 A study of LRRK2 mutations and Parkinson's disease in Brazil
M.M.G. Pimentel, K.C.V. Moura, C.B. Abdalla, J.S. Pereira, A.L.Z. Rosso, D.H. Nicaretta, M. Campos, Jr., R.M. Almeida, J.M. Santos, I.C.G. Bastos, M.F.X. Mendes, H. Maultasch, F.H.R. Costa, A.L.S. Werneck, C.B. Santos-Rebouças (Rio de Janeiro, Brazil)
- 115 Screening for the LRRK2 G2019S and codon-1441 mutations in a pathological series of parkinsonian syndromes and frontotemporal lobar degeneration
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- 218 Investigating 5HT_{2A} receptor binding in PD patients with visual hallucinations: A [¹⁸F]setoperone PET study
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- 219 How common are "funny signs" in brain magnetic resonance imaging in newly diagnosed patients with parkinsonism?
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- 220 Iron accumulation in basal ganglia with normal ageing and Parkinson disease assessed with T2* MRI
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- 221 Progression of monoaminergic transporter decline in MSA-P and Parkinson's disease: A voxel-by-voxel analysis of [¹²³I] β-CIT SPECT
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- 222 Usefulness of [¹²³I]FP-CIT striatal asymmetry index as specific tool to differentiate Parkinson's disease from vascular parkinsonism
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- 223 Diffusion weighted imaging in presymptomatic and early Huntington's disease
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- 224 The pattern of cerebral perfusion in Parkinson's disease according to age
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225 Abnormalities in white matter of treatment naïve patients with Wilson's disease: A voxel-based diffusion tensor study
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226* Neurological effects with recombinant human erythropoietin in Friedreich's ataxia
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227* A detailed behavioural, neurochemical and histological characterization of an α -synuclein transgenic mouse line as a model of Parkinson's disease
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228* Motor and non-motor behavioral impairments associated to decreased expression of tyrosine hydroxylase after intracerebral administration of lactacystin
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229* Administration of the cannabinoid receptor agonist CP-55,940 reduced motor impairment and tyrosine hydroxylase expression loss in 6-hydroxydopamine-lesioned mice
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230* Gardenosides increase astrocyte's neuroprotective effect on dopaminergic neurons by inhibiting lipopolysaccharide-induced secretion of inflammatory factors
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231* Glucocorticoid-mediated and cytokine-mediated inflammatory responses in MPTP-treated monkeys. Implications in the progressive degeneration process
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232* Migration of type A botulinum toxin in vivo is not related to the size of the toxin complex
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233* The relationship between exposure to aripiprazole and development of parkinsonism
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234* Efficacy of NT 201 (Xeomin®) in focal dystonia
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235* Disabling alien limb phenomena improved with clonazepam and botulinum toxin injections
I.U. Haq, I. Malaty, H.H. Fernandez, M.S. Okun, R.R. Rodriguez (Gainesville, FL)

236* Case-control study of plasma uric acid in Parkinson's disease
F. Moisan, M.-A. Lorient, A. Le Floch, M. Vidailhet, I. Ceballos, J.-L. Perignon, C. Tzourio, A. Elbaz (Paris, France)

237* Striatal histone post-translational modifications in animal models of levodopa-induced dyskinesia
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238* [11C]raclopride PET imaging demonstrates correlation between correction of dopamine neurotransmission and behavioral recovery following gene therapy
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239* Innovative options for the conservative treatment of Tourette syndrome (TS): The role of tetrabenazine (TBZ) on the basis of a selected cohort of 120 patients treated at the AIST Milan
M. Porta, M. Sassi, A. Brambilla, S. Defendi, D. Servello (Milan, Italy)

240* The role of dopaminergic medication doses in impulse control disorders (ICD) in Parkinson's disease (PD)
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241* Coenzyme Q10 in Parkinson's disease (PD)
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242* Does intolerance to a diagnostic acute L-dopa challenge in parkinsonian syndromes differentiate MSA from IPD?
S. Estévez, S. Perez-Lloret, M. Merello (Cdad. Aut. Bs. As., Argentina)

243* Glutamate neurotransmission in dyskinetic Parkinson's disease: An 11C-CNS 5161 PET study
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244* A pharmacokinetic-pharmacodynamic model for duodenal levodopa infusion
J. Westin, T. Willows, T. Groth, M. Dougherty, M. Karlsson, D. Nyholm, S. Pålhagen (Borlange, Sweden)

245* Applying the UK PD Brain Bank criteria to SWEDDs (scans without evidence of dopaminergic deficit)
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246 Nicotinamide and redox (NADH/NAD+) effects in replicating and terminally differentiated human neuronal cell models exposed to neurotoxins
D.B. Ramsden, L.S. Cartwright, A.C. Williams (Birmingham, United Kingdom)

247 Low dose methylphenidate improves freezing in advanced Parkinson's disease during off-state
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248 Successful and rapid improvement of tardive dyskinesia with a combination of tetrabenazine, clonazepam and clozapine at low doses
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249 Continuous vs pulsatile administration of rotigotine in rat and monkey models of Parkinson's disease: A comparison
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250 Involvement of D1 dopamine receptor signaling in hyperphosphorylation of the microtubule associated protein tau: A pivotal role for CDK5 and GSK3
M. Le Bel, M. Cyr (Trois-Rivieres, QC, Canada)

251 The safety and efficacy of istradefylline, an adenosine A2A antagonist, as monotherapy in early Parkinson disease: Results of the KW-6002-US-051 trial
H.H. Fernandez, 6002-US-051 Study Group (Gainesville, FL)

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- 253 Organization of natural eye blinks and dopaminergic function
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- 254 Calcium homeostasis is dysregulated in parkinsonian patients with L-Dopa induced dyskinesias
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- 258* Cognitive impairment in early and untreated Parkinson's disease: Associations with cerebrospinal fluid levels of β -amyloid1-42, total tau, and phosphorylated (181p) tau protein
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- 259* Clinical factors associated with age-related variability in cognitive test performance in Parkinson's disease
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- 260* Comparison of cognitive function in early-onset versus late-onset Parkinson's disease
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- 261* Cognitive changes without dementia in PD: Evidence for neuropsychological subtypes
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- 262* Relevance of new Movement Disorders Task Force recommendations for Parkinson Disease Dementia diagnosis (PD-D).
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- 263* Impact of mild cognitive deficits on daily functioning in Parkinson's disease
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- 264* Impaired curve negotiation in drivers with Parkinson's disease
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- 265* Switching between motor representations in Parkinson's disease – an fMRI study
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- 266* Brain metabolic pattern (BMP) of cognitive decline in Parkinson's disease (PD)
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- 267* Relationship of cortical Pittsburgh compound B (PIB) binding and clinical features in Parkinson disease dementia
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- 268* Relationship between neuropsychological functioning and PIB binding in Parkinson disease
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- 269* Combined use of 3T proton spectroscopy, DTI and VBM for capturing cortical changes in Parkinson's disease with cognitive dysfunction: A preliminary study
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- 270* Implicit motor sequence learning in patients with Parkinson's disease depends on the stage of disease
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- 271* Effect of dopaminergic therapy on the fronto-striatal patterns of activity observed in patients with Parkinson's disease during the execution of a cognitive task
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- 272* Changes in cerebral glucose metabolism in patients with Parkinson's disease dementia after cholinesterase inhibitor therapy
S.W. Yong, I.S. Joo, P.H. Lee (Suwon, Republic of Korea)
- 273* Medication improves executive function in tremor-dominant subtype of idiopathic Parkinson disease
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- 274* White matter disease as a risk factor for memory decline following deep brain stimulation surgery
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- 275* A longitudinal study of cognitive dysfunction in patients affected by Parkinson's disease with and without freezing of gait
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- 276* Visual hallucinations in Parkinson's disease, preliminary fMRI results
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- 277 Cardiac 123I-metaiodobenzylguanidine scintigraphy in patients with Parkinson disease associated dementia
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- 278 Abstract Withdrawn

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- 279 Pellagra, the classic redox (NAD(H)) disorder, showed clinical signs of disordered gamma network oscillations
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- 280 Connecting laboratory executive function to everyday executive function and complex activity participation in non-demented individuals with Parkinson's disease (PD)
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- 281 Rasagiline and the improvement of cognition in Parkinson's disease population
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- 282 Exacerbated objective mental fatigue in PD
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- 283 Cognitive decline in Parkinson's disease
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- 284 Delayed cognitive changes with DBS on/off stimulator testing in a 62 year-old Parkinson's disease patient
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- 285 Performance on the mini-mental state exam in carriers and non-carriers of parkin mutations
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- 286 Prospective memory in Parkinson's disease (PD) without dementia across laboratory and everyday life measures
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- 287 A comparison of two brief cognitive measures in Parkinson's disease (PD)
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- 288 A diminished role for the Lewy body in Parkinson's disease dementia
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- 289 Increased amygdala activation during emotional processing in unmedicated Parkinson patients
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- 290 What are the typical neuropsychological deficits in patients with Parkinson's disease prior to deep brain stimulation?
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- 291 Impaired early learning of explicit motor sequences by dopaminergic medications in Parkinson's disease
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- 292 Parkinson's disease increases the risk for developing mild cognitive impairment
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- 293 Cognitive decline progression in Parkinson's disease: An anatomical and neuropsychological study
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- 294 Impaired recognition of emotions in music in Parkinson's disease
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- 295 Excessive daytime sleepiness in Parkinson's disease with and without dementia
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- 296 Differential cognitive features of Parkinson's disease dementia and Alzheimer's disease
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- 297 Frontal lobe dysfunction in parkinsonian disorders. The PRIAMO study
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- 298 Differences in naming objects and actions between Alzheimer's disease and Parkinson's disease
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- 299 Neuropsychological profile in Parkinson's disease: Cognitive impairment evolution
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- 300 Effects of continuous duodenal levodopa infusion on cognitive function and behaviour
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- 301 Subtypes of mild cognitive impairment in Parkinson's disease with REM sleep behavior disorder
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- 304 Can aerobic exercise enhance cognition and language production in Parkinson's disease? A clinical case report
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- 305 Everyday functioning in Parkinson's disease (PD) and PD dementia (PDD): Direct assessment from a neuropsychological perspective
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- 306 Parkinson's disease: Does the estimation of disease duration depend on how you asked the question?
G. Di Virgilio, F. Siclari, F. Vingerhoets (Lausanne, Switzerland)

My Parkinson's disease has advanced, but...
I don't like to take *off* episodes sitting down.

Please Visit
Booth 425
During
MDS



APOKYN® moves me.

- ◆ Only APOKYN is proven to **REVERSE off episodes***¹
- ◆ **90% of patients achieved a therapeutic response within 20 minutes** that was approximately equivalent to their usual response to levodopa (n=20)
- ◆ **Turn off time into on time** for patients who are²⁻³:
 - Experiencing *off* episodes despite optimized oral PD therapy
 - Stages II to IV of the 5-stage Hoehn and Yahr scale
- ◆ **Use prn as needed up to 5 times a day**
 - In clinical trials, the mean dose of APOKYN was 0.54 ± 0.05 mL in patients who had no prior exposure to apomorphine¹

*Indicated for the acute, intermittent treatment of hypomobility, *off* episodes (end-of-dose *wearing-off* and unpredictable *on-off* episodes) associated with advanced Parkinson's disease. APOKYN has been studied as an adjunct to other medications.

The concomitant use of apomorphine with drugs of the 5HT₃ antagonist class is contraindicated.

Apomorphine should not be administered intravenously.

At the recommended doses of apomorphine, severe nausea and vomiting can be expected. Therefore, trimethobenzamide should be started 3 days prior to the initial dose of apomorphine and continued for at least 2 months.

Caution is recommended when administering apomorphine to patients with increased risk of QT prolongation.

Apomorphine can cause hypotension, orthostatic hypotension, and syncope. Apomorphine has the potential to exacerbate coronary (and cerebral) ischemia.

There have been literature reports of patients treated with apomorphine who suddenly fell asleep while engaged in activities of daily living.

The most common adverse events seen in controlled trials were yawning, dyskinesias, nausea and/or vomiting, somnolence, dizziness, rhinorrhea, hallucinations, edema, chest pain, increased sweating, flushing, and pallor.

Please see accompanying Brief Summary of Prescribing Information.

References:

1. Dewey RB, Hutton JT, LeWitt PA, Factor SA. A randomized, double-blind, placebo-controlled trial of subcutaneously injected apomorphine for parkinsonian off-state events. *Arch Neurol*. 2001;58:1385-1392. 2. Data on file, Clinical Report APO401. Vernalis Pharmaceuticals Inc, Morristown, NJ.
3. Data on file, Clinical Report APO302. Vernalis Pharmaceuticals Inc, Morristown, NJ.

For more information about APOKYN, please visit www.APOKYN.com.



APOKYN®
apomorphine hydrochloride injection

Help Them **Get Up and Go**



10 mg/mL For Subcutaneous Use Only Not for IV Use
Rx only

BRIEF SUMMARY OF PRESCRIBING INFORMATION

CONTRAINDICATIONS: Based on profound hypotension and loss of consciousness when apomorphine was administered with ondansetron, concomitant use of apomorphine with 5HT₃ antagonists (eg, ondansetron, granisetron, dolasetron, palonosetron, and alosetron) is contraindicated. Contraindicated in patients who have demonstrated hypersensitivity to the drug or its ingredients (notably sodium metabisulfite).

WARNINGS: Avoid Intravenous Administration: Serious adverse events (intravenous crystallization of apomorphine, leading to thrombus formation and pulmonary embolism) have followed intravenous administration of apomorphine. **General:** Almost all of the significant adverse events below with subcutaneous apomorphine occurred in open-label, uncontrolled studies. Controlled trial data involved relatively few patients, and examined effects of single doses. Because the background rate of many of these events in advanced Parkinson's disease (PD) patients is unknown, it is difficult to assess the causal role of apomorphine.

Nausea and Vomiting: At recommended doses, severe nausea and vomiting can be expected. In domestic clinical studies, 98% of patients were treated with the antiemetic trimethobenzamide for 3 days prior to beginning apomorphine and were encouraged to continue trimethobenzamide for at least 6 weeks. A total of 262/522 (50%) patients discontinued trimethobenzamide while continuing apomorphine. Average time to discontinuation of trimethobenzamide was about 2 months (range: 1 day to 33 months). For the 262 patients who discontinued trimethobenzamide, 249 patients continued apomorphine without trimethobenzamide for a duration of follow-up that averaged 1 year (range: 0-3 years). Even with trimethobenzamide, 31% had nausea and 11% had vomiting. In clinical trials, 3% of the patients discontinued apomorphine due to nausea and 2% discontinued due to vomiting. In the domestic development of apomorphine, there was no experience with antiemetics other than trimethobenzamide. Some antiemetics with anti-dopaminergic actions have the potential to worsen the clinical state of PD patients and should be avoided. **Syncope:** In clinical studies, about 2% of patients experienced syncope. **QT Prolongation and Potential for Proarrhythmic Effects:** In a study in which patients received increasing single apomorphine doses from 2 to 10 mg (if tolerated) and placebo, the mean difference in QTc between apomorphine and placebo, as measured by Holter monitor, was 0 msec at 4 mg, 1 msec at 6 mg, and 7 msec at 8 mg. Too few patients received a 10 mg dose to characterize the change in QTc interval at that dose. In a controlled trial in which patients were administered placebo or a single apomorphine dose (mean 5.2 mg; range of 2-10 mg, with 30 of 35 patients receiving a 6 mg dose or less), the mean difference between apomorphine and placebo in the change in QTc was about 3 msec at 20 and 90 minutes. In the entire database, 2 patients (one at 2 and 6 mg, one at 6 mg) exhibited large QTc increments (>60 msec from pre-dose) and had QTc intervals greater than 500 msec acutely after dosing. Doses of 6 mg or less thus are associated with minimal increases in QTc. Doses greater than 6 mg do not provide additional clinical benefit and are not recommended. Some drugs that prolong the QT/QTc interval have been associated with torsades de pointes and with sudden unexplained death. The relationship of QT prolongation to torsades de pointes is clearest for larger increases (20 msec and greater), but it is possible that smaller QT/QTc prolongations may also increase risk, or increase it in susceptible individuals, such as those with hypokalemia, hypomagnesemia, bradycardia, concomitant use of other drugs that prolong the QTc interval, or genetic predisposition (eg, congenital prolongation of the QT interval). Although torsades de pointes has not been observed with apomorphine at recommended doses in premarketing studies, experience is too limited to rule out an increased risk. Palpitations and syncope may signal the occurrence of torsades de pointes. Use caution when administering apomorphine to patients with these risk factors. **Symptomatic Hypotension:** Dopamine agonists may cause orthostatic hypotension at any time, especially during dose escalation. PD patients may have an impaired capacity to respond to an orthostatic challenge. Carefully monitor PD patients being treated with dopaminergic agonists for signs and symptoms of orthostatic hypotension, especially during dose escalation, and inform them of this risk. Apomorphine causes dose-related decreases in systolic (SBP) and diastolic blood pressure (DBP). Dose-dependent mean decrements in SBP ranged from 5 mmHg after 2 mg to 16 mmHg after 10 mg. Dose-dependent mean decrements in DBP ranged from 3 mmHg after 2 mg to 8 mmHg after 10 mg. These changes were observed at 10 minutes, appeared to peak at about 20 minutes after dosing, and persisted up to at least 90 minutes post-dosing. Patients undergoing apomorphine titration showed an increased incidence (from 4% pre-dose to 18% post-dose) of systolic orthostatic hypotension (≥ 20 mmHg decrease) when evaluated at various times after in-office dosing. A small number of patients developed severe systolic orthostatic hypotension (≥ 30 mmHg decrease and systolic BP ≤ 90 mmHg) after apomorphine. In apomorphine clinical trials in patients with advanced PD, 59/550 patients (11%) had orthostatic hypotension, hypotension, and/or syncope. Events were considered serious in 4 patients (<1%) and resulted in withdrawal of apomorphine in 10 patients (2%). These events occurred with initial dosing and during long-term treatment. Whether or not hypotension contributed to other significant adverse events seen (eg, falls), is unknown. The effects of apomorphine on blood pressure may be increased by concomitant use of alcohol, antihypertensive medications, and vasodilators (especially nitrates). Avoid alcohol when using APOKYN and exercise extra caution if APOKYN must be administered with concomitant antihypertensive medications and/or vasodilators (see PRECAUTIONS: Drug Interactions and Information for Patients). **Falls:** Patients with PD are at risk of falling due to the underlying postural instability and concomitant autonomic instability seen in some patients with PD, and from syncope caused by the blood pressure lowering effects of PD drugs. Subcutaneous apomorphine might increase the risk of falling by simultaneously lowering blood pressure and altering mobility (see WARNINGS: Symptomatic Hypotension; PRECAUTIONS: Dyskinesias). In clinical trials, 30% of patients had

events that could be considered falls and about 5% of patients had serious falls. Because these data were obtained in open, uncontrolled studies, and given the unknown background rate of falls in patients with advanced PD, it is impossible to definitively assess the contribution of apomorphine to these events. **Hallucinations:** During clinical development, hallucinations were reported by 14% of patients and resulted in discontinuation of apomorphine in 1% of patients. **Falling Asleep During Activities of Daily Living (ADL):** There have been literature reports of patients treated with apomorphine subcutaneous injections who suddenly fell asleep without prior warning of sleepiness while engaged in ADL. Somnolence is commonly associated with APOKYN and clinical experts believe that falling asleep while engaged in ADL always occurs in a setting of pre-existing somnolence even if patients do not give such a history. Therefore continually reassess patients for drowsiness or sleepiness, especially since some of the events occur well after the start of treatment and be aware that patients may not acknowledge drowsiness or sleepiness until directly questioned. Before initiating APOKYN, advise patients of drowsiness and specifically ask about factors that could increase the risk, such as concomitant sedating medications and the presence of sleep disorders. If significant daytime sleepiness or episodes of falling asleep develop during activities that require active participation (eg, conversations, eating, etc), APOKYN should ordinarily be discontinued. If APOKYN is continued, advise patients not to drive and to avoid other potentially dangerous activities. It is unknown whether dose reduction will eliminate episodes of falling asleep while engaged in ADL. **Coronary Events:** During clinical development, 4% of apomorphine-treated patients had angina, myocardial infarction, cardiac arrest and/or sudden death; some angina and myocardial infarction cases occurred within 2 hours of apomorphine dosing, while cardiac arrest and sudden death cases were at times unrelated to dosing. Apomorphine reduces resting systolic and diastolic blood pressure and, as such, has the potential to exacerbate coronary (and cerebral) ischemia. Use extra caution in prescribing apomorphine for patients with known cardiovascular and cerebrovascular disease. Re-evaluate the continued use of apomorphine if patients develop signs and symptoms of coronary or cerebral ischemia. **Contains Sulfite:** APOKYN contains sodium metabisulfite, a sulfite that may cause allergic-type reactions, including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people. The prevalence of sulfite sensitivity in the general population is unknown and probably low, and is seen more frequently in asthmatic than in non-asthmatic people. **Injection Site Reactions:** Among 550 apomorphine-treated patients, 26% complained of injection site reactions, including bruising (16%), granuloma (4%), and pruritus (2%). There was a limited experience (both for overall numbers of patients and total number of injections per patient) with apomorphine injections in controlled trials; the number of injection site reactions reported by patients receiving apomorphine was similar to that reported for placebo. **Potential for Abuse:** There are rare reports of apomorphine abuse by PD patients in other countries. Psychosexual stimulation with increased libido is believed to underlie these cases that are characterized by increasingly frequent dosing leading to hallucinations, dyskinesia, and abnormal behavior. Prescribers should be vigilant for evidence that patients are abusing apomorphine, such as use out of proportion to motor signs (see DRUG ABUSE AND DEPENDENCE).

PRECAUTIONS: Dyskinesias: Apomorphine may cause dyskinesia or exacerbate pre-existing dyskinesia. During clinical development, dyskinesia or worsening of dyskinesia was reported in 24% of patients. Overall, 2% of patients withdrew from studies due to dyskinesias. **Events Reported with Dopaminergic Therapy:** Events enumerated below have not been reported with apomorphine, but are associated with other dopaminergic drugs. **Withdrawal-emergent Hyperpyrexia and Confusion:** Although not reported with apomorphine, 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 antiparkinsonian therapy. **Fibrotic Complications:** Cases of retroperitoneal fibrosis, pulmonary infiltrates, pleural effusion, pleural thickening, and cardiac valvulopathy have been reported in some patients treated with ergot-derived dopaminergic agents. They may resolve with drug discontinuation, however, 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. **Priapism:** Apomorphine may cause prolonged painful erections in some patients. During clinical development, painful erections were reported by 3/361 males (<1%), and one apomorphine patient withdrew because of priapism. Although no patients required surgical intervention, severe priapism may require surgical intervention.

Hepatic Impairment: Exercise caution when administering apomorphine to patients with mild and moderate hepatic impairment due to the increased C_{max} and AUC in these patients. Studies of subjects with severe hepatic impairment have not been conducted (see CLINICAL PHARMACOLOGY and DOSAGE AND ADMINISTRATION). **Renal Impairment:** Reduce the starting dose to 1 mg when administering apomorphine to patients with mild or moderate renal impairment because the C_{max} and AUC are increased in these patients. Studies in subjects with severe renal impairment have not been conducted (see CLINICAL PHARMACOLOGY and DOSAGE AND ADMINISTRATION). **Retinal Pathology in Albino Rats:** Retinal degeneration has been observed in albino rats treated with dopamine agonists for prolonged periods (generally during 2-year carcinogenicity studies) and when exposed to these agents for shorter periods under higher intensity light exposures. Similar changes have not been observed in 2-year carcinogenicity studies in albino mice or in rats or monkeys treated for 1 year. APOKYN has not been tested in carcinogenicity studies, but based on its mechanism of action it would be expected to cause similar toxicity. The 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 (eg, disk shedding) may be involved.

Information for Patients: APOKYN is intended only for subcutaneous injection and must not be given intravenously. Urge patients and caregivers to read the Patient Package Insert and Directions for Use for the dosing pen. Instruct patients to use APOKYN only as prescribed. Patients and/or

caregivers who are advised to administer APOKYN in medically unsupervised situations should receive instruction on the proper use of the product from the physician or other qualified health care professional and observed during initial dosing. **Patients and caregivers must receive detailed instruction in the use of the dosing pen: 1) Patients need to be aware that the drug is dosed in milliliters, not milligrams. Particularly caution patients that a dose of 1 mg is represented on the dosing pen as 0.1 mL, and not as 1.0 (the latter representing a dose of 10 mg). This distinction is critical to prevent potentially life-threatening overdose if a dose of 1 mg is prescribed. 2) Inform patients and caregivers that it is possible to dial in their usual dose of apomorphine even though the cartridge may contain less than that amount of drug. In this case, they will receive only a partial dose with the injection, and the amount left to inject will appear in the dosing window. To complete the correct dose, patients/caregivers will need to "re-arm" the device and dial in the correct amount of the remaining dose. If at all possible, avoid this situation, and alert patients and caregivers that there may be insufficient drug left in the cartridge to deliver a complete dose (for example, urge patients and caregivers to keep records of how many doses they have delivered for each cartridge, so that they can replace any cartridge that has an inadequate amount of drug remaining).** Instruct patients to rotate the injection site and to observe proper aseptic technique. Inform patients that hallucinations can occur. Advise patients of 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 a dose increase at any time. Caution patients against rising rapidly after sitting or lying down, especially if they have been sitting or lying for prolonged periods, and especially at the initiation of APOKYN. Alcohol, antihypertensive medications, and vasodilating medications may potentiate the hypotensive effect of apomorphine (see WARNINGS: Symptomatic Hypotension; PRECAUTIONS: Drug Interactions). Alert patients to the potential sedating effects of APOKYN, 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 know whether APOKYN affects their mental and/or motor performance adversely. Advise patients that if increased somnolence or episodes of falling asleep during ADL (eg, 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 APOKYN. Because apomorphine has not been evaluated for effects on reproduction and embryo-fetal development, advise patients to notify their physicians if they become pregnant or intend to become pregnant (see PRECAUTIONS: Pregnancy). Because apomorphine may be excreted in breast milk, advise patients to notify their physicians if they intend to breast-feed. Rare cases of abuse (use of apomorphine significantly in excess of prescribed frequency) have been reported and may be associated with inappropriate sexual behavior.

Drug Interactions: 5HT₃ Antagonists: Based on reports of profound hypotension and loss of consciousness when apomorphine was administered with ondansetron, concomitant use of apomorphine with 5HT₃ antagonists (eg, ondansetron, granisetron, dolasetron, palonosetron, and alosetron) is contraindicated (see CONTRAINDICATIONS). **Antihypertensive Medications and Vasodilators:** The following adverse events were experienced more commonly in patients receiving concomitant antihypertensive medications or vasodilators (n=94) compared to patients not receiving these concomitant drugs (n=456): hypotension 10% vs 4%, myocardial infarction 3% vs 1%, serious pneumonia 5% vs 3%, serious falls 9% vs 3%, and bone and joint injuries 6% vs 2%. The mechanism underlying many of these events is unknown, but may represent increased hypotension (see WARNINGS: Symptomatic Hypotension). **Dopamine Antagonists:** Since apomorphine is a dopamine agonist, it is possible that dopamine antagonists, such as neuroleptics (phenothiazines, butyrophenones, thioxanthenes) or metoclopramide, may diminish the effectiveness of APOKYN. Patients with major psychotic disorders, treated with neuroleptics, should be treated with dopamine agonists only if the potential benefits outweigh the risks. **Drugs Prolonging the QT/QTc Interval:** Exercise caution when prescribing apomorphine concomitantly with drugs that prolong the QT/QTc interval (see WARNINGS: QT Prolongation and Potential for Proarrhythmic Effects).

Drug/Laboratory Test Interactions: There are no known interactions between APOKYN and laboratory tests. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Carcinogenicity studies have not been conducted with APOKYN. Apomorphine was mutagenic in the *in vitro* bacterial Ames test and the *in vitro* mammalian mouse lymphoma assay. Apomorphine was also clastogenic in the *in vitro* chromosomal aberration assay in human lymphocytes and the *in vitro* mouse lymphoma assay. Apomorphine was negative in the *in vivo* micronucleus assay in mice. In a published fertility study in male rats, an adverse effect on fertility was observed at 2 mg/kg administered subcutaneously (0.6 times the MRHD in a mg/m² basis). A significant decrease in testis weight was observed in a 39-week study in cynomolgus monkey at subcutaneous doses of 1.0 and 1.5 mg/kg (0.6 and 1 times the MRHD on a mg/m² basis). **Pregnancy: Pregnancy Category C:** Reproduction studies have not been conducted with apomorphine. It is not known whether apomorphine can cause fetal harm when administered to a pregnant woman or can affect reproductive capacity. Apomorphine should be given to a pregnant woman only if clearly needed.

Nursing Mothers: It is not known whether apomorphine 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 apomorphine, a decision should be made as to whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. **Pediatric Use:** The safety and efficacy of APOKYN in pediatric patients has not been established. **Geriatric Use:** In the apomorphine clinical development program, 239 patients were less than 65 years of age and 311 were 65 years of age or older. Adverse events were about equally common in older and younger patients (90% vs 87%), but with older patients more likely to experience confusion and hallucinations. Serious adverse events (life-threatening events or events resulting in hospitalization and/or increased disability) were more common in older patients (27% vs 17%), with older patients more likely to fall (experiencing bone and joint injuries), have cardiovascular events, develop respiratory disorders, and have gastrointestinal events. Older patients were more likely to discontinue apomorphine due to adverse events (29% vs 21%). **ADVERSE EVENTS: Adverse Event Incidence in Controlled Clinical Studies:** APOKYN has been administered to 550 PD patients who were taking some form of L-Dopa along with other PD medications. A total of 86% of patients were taking a concomitant dopamine agonist. All patients had some degree of spontaneously occurring hypomobility ("off episodes") at baseline. Adverse events were recorded by the clinical investigators using their own terminology. Similar types of events were grouped into a smaller number of standardized categories using MedDRA dictionary terminology. The most common adverse events in controlled trials were yawning, dyskinesias, nausea and/or vomiting, somnolence, dizziness, rhinorrhea, hallucinations, edema, chest pain, increased sweating, flushing, and pallor. The most extensive experience with apomorphine in randomized, controlled trials comes from a multicenter, randomized, placebo-controlled, parallel-group trial conducted in apomorphine-naïve PD patients treated for up to 4 weeks (Table 1). Apomorphine doses ranged from 2-10 mg, optimized to achieve control of symptoms comparable to the response with the usual dose of L-dopa. These figures cannot be used to predict the 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 involving different treatments, uses, and investigators. 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: Summary of Adverse Events Occurring in Two or More Patients Treated With Apomorphine (n=20) or Placebo (n=9), Respectively: Any adverse reaction 85% (17/20) vs 89% (8/9); yawning 40% (8/20) vs 0%; dyskinesias 35% (7/20) vs 11% (1/9); drowsiness or somnolence 35% (7/20) vs 0%; nausea and/or vomiting 30% (6/20) vs 11% (1/9); dizziness or postural hypotension 20% (4/20) vs 0%; rhinorrhea 20% (4/20) vs 0%; chest pain/pressure/angina 15% (3/20) vs 11% (1/9); hallucinations or confusion 10% (2/20) vs 0%; edema/swelling of extremities 10% (2/20) vs 0%. **Other Adverse Events Observed During All Phase 2/3 Clinical Trials:** Among 550 patients, 89% had at least one adverse event (AE). The most common AEs in addition to those listed above (occurring in at least 5% of the patients and at least plausibly related to treatment) in descending order were injection site complaint, fall, arthralgia, insomnia, headache, depression, urinary tract infection, anxiety, congestive heart failure, limb pain, back pain, Parkinson's disease aggravated, pneumonia, confusion, sweating increased, dyspnea, fatigue, ecchymosis, constipation, diarrhea, weakness, and dehydration.

DRUG ABUSE AND DEPENDENCE: Potential for Abuse: A rarely reported motivation for apomorphine abuse (escalation of dose beyond prescribed frequency) is the use of apomorphine to avoid all symptoms of all "off" events when "off" events occur frequently. A second, rarely reported, motivation for apomorphine abuse is a psychosexual reaction related to the stimulation of penile erection and increase in libido. Adverse events that have been reported in males with overuse include frequent penile erections, atypical sexual behavior, heightened libido, dyskinesias, agitation, confusion, and depression. No studies have been conducted to evaluate the potential for dependence when apomorphine is used as acute (rescue) treatment of "off" episodes in patients with "on/off" or "wearing-off" effects associated with late stage PD.

OVERDOSAGE: Intermittent Injection: An accidental overdose of 25 mg injected subcutaneously in a 62 year old man was published in *Journal of Neurology, Neurosurgery, and Psychiatry* (1990), Vol. 53, pp. 96-102. After 3 minutes, the patient felt nauseated and lost consciousness for 20 minutes. Afterwards, he was alert with a heart rate of 40/minute and a supine blood pressure of 90/50. He recovered completely within an hour.

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- 358 Ablative stereotactic surgery for the treatment of parkinsonism linked to G2019S LRRK2 mutation
R.P. Munhoz, A.N. Francisco, H.A.G. Teive, E. Rogaeva (Curitiba, Parana, Brazil)
- 359 Hypomania induced by subthalamic nucleus stimulation in a Parkinson's disease patient – a case report
J.-S. Kim, S.-H. Paek, B.S. Jeon (Sung Nam Si, Korea)
- 360 PD SURG: A large, randomised trial to assess the impact of surgery in Parkinson's disease
A. Williams, S. Patel, N. Ives, C. Rick, J. Daniels, C. Jenkinson, S. Gill, T. Varma, K. Wheatley (Birmingham, West Midlands, United Kingdom)
- 361 The impact of surgery in Parkinson's disease on carer quality of life: Results from the PD SURG trial
S. Patel, C. Rick, N. Ives, A. Williams, J. Daniels, C. Jenkinson, S. Gill, T. Varma, K. Wheatley (Birmingham, West Midlands, United Kingdom)
- 362 Which patients with Parkinson's disease benefit most from surgery? Subgroup analysis from the PD SURG trial
S. Patel, N. Ives, C. Rick, A. Williams, J. Daniels, C. Jenkinson, S. Gill, T. Varma, K. Wheatley (Birmingham, West Midlands, United Kingdom)
- 363 The effect of different stimulation parameters on outcome following deep brain stimulation of the pedunculo-pontine nucleus
C.A. Joint, N. de Pennington, T.Z. Aziz (Oxford, Oxfordshire, United Kingdom)
- 364 Performance deterioration during repetitive finger movement in normal subjects and PD patients
J.P. Rodrigues, G.W. Thickbroom, F.L. Mastaglia (Perth, WA, Australia)

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- 365 Subthalamic nucleus stimulation affects orbitofrontal cortex in facial emotion recognition: A PET study
F. Le Jeune, J. Peron, I. Biseul, S. Vicente, P. Sauleau, S. Drapier, D. Drapier, B. Millet, C.-H. Malbert, M. Verin (Rennes, France)
- 366 Deep brain stimulation of the subthalamic nucleus as an effective treatment of akathisia in Parkinson disease
S.-k. Song, H.-W. Shin, Y.H. Sohn (Seoul, Korea)
- 367 The role of STN DBS in the treatment of compulsions in Parkinson's disease
W. Cole, M. Barad, H. Bronte-Stewart (Stanford, CA)
- 368 Progression of Parkinson's disease after long-term electrical stimulation of subthalamic nucleus
A. Kishore, D. Gupta, R. Rao, S. Sarma (Trivandrum, Kerala, India)
- 369 State-dependent effects upon dopamine metabolism of subthalamic nucleus stimulation in a rat model of Parkinson's disease
R.H. Walker, R. Koch, C. Moore, C.K. Meshul (Bronx, NY)
- 370 Change in exertional O₂ saturation with deep brain stimulation in PD
M. Giroux, S. Farris, A. Zylstra, E. Kehl (Kirkland, WA)
- 371 Targeting the subthalamic nucleus for deep brain stimulation – a comparative study between magnetic resonance images alone and fusion with computed tomographic images
S.-Y. Chen, S.-T. Tsai, S.-H. Lin, Y.-H. Pan, H.-Y. Hung (Hualien, Taiwan)
- 372 Deep brain stimulation of STN improves some handwriting movements in Parkinson's disease patients
M.V. Alvarez, A.W. Van Gemmert, V.H. Evidente, J.N. Caviness, E.D. Driver-Dunkley, M.H. Lyons, C.H. Adler (Lackland AFB, TX)
- 373 Visual hallucinations or spiritual visions? The implication for deep brain stimulation
I. Malek, M. Samuel, A. Costello, N. Hulse, R. Selway, K. Ashkan, J. Moriarty (London, United Kingdom)
- 374 Reversible acute cognitive dysfunction induced by bilateral STN stimulation
G. Yalcin Cakmakli, H. Oruçkaptan, E. Saka, B. Elibol (Ankara, Turkey)
- 375 Deep brain stimulation (DBS) as a treatment option for Parkinson's disease and essential tremor in elderly patients
N. Hulse, K. Ashkan, A. Costello, J. Moriarty, C. Clough, R. Selway, M. Samuel (London, United Kingdom)
- 376 He said, she said: Differences between self and caregiver ratings of postoperative behavioral changes in Parkinson's disease (PD) patients undergoing bilateral subthalamic nucleus deep brain stimulation (STN-DBS)
S.J. Duff-Canning, Y.Y. Poon, T. Chang, N. Mailis, A.M. Lozano, M. Hodaie, J.A. Saint-Cyr, A.E. Lang, E. Moro (Toronto, ON, Canada)
- 377 Autologous fibroblast transplantation to internal pallidum as a method for reducing parkinsonian symptomatology
J.P.M. Finberg, Z. Gluzman, M. Reshef, O. Mohsen, Y. Loboda, E. Vlodaysky, S. Marom, Y. Feld (Haifa, Israel)
- 378 Gender differences in long-term outcome after subthalamic nucleus stimulation in Parkinson's disease
L.M.A. Romito, M.F. Contarino, A.R. Bentivoglio, M. Scerrati, A. Albanese (Milano, Italy)
- 379 Spatiotemporal locomotion disorders and temporal speech disorders in Parkinson's disease: A similar deficit?
S. Cantiniaux, T. Witjas, M. Vaugoyeau, D. Robert, J.-P. Azulay (Marseille, France)
- 380 Dopasensitive axial involvement in Parkinson's disease: A long term evaluation under subthalamic deep brain stimulation
S. Cantiniaux, T. Witjas, J. Mancini, J. Regis, J.-C. Peragut, J.-P. Azulay (Marseille, France)
- 381 Functional imaging of STN DBS-related effects on visual cortex
R. Jech, D. Urgosik, F. Ruzicka, J. Vymazal, E. Ruzicka (Prague, Czech Republic)
- 382 Deep brain stimulation and cognitive functions in Parkinson disease: A 3 years controlled study
R. Zangaglia, C. Pacchetti, C. Pasotti, M. Sciarretta, D. Servello, F. Mancini, E. Martignoni, G. Nappi (Pavia, Italy)
- 383 Prognostic factors for long-term subthalamic stimulation in Parkinson's disease
S.-T. Tsai, S.-H. Lin, H.-Y. Hung, Y.-H. Pan, S.-Y. Chen (Hualien, Taiwan)
- 384 Role of cerebral MRI as predictive factor of subthalamic DBS outcome in Parkinson's disease
L.M. Raglione, M. Moretti, F. Ammannati, S. Ramat, P. Marini, V. Boddi, P. Mennonna, S. Sorbi (Florence, Italy)
- 385 Subthalamic stimulation modulates non-motor behavior in Parkinson's disease
R. Ash, V.L. Salak, V.K. Hinson, K.J. Bergmann (Charleston, SC)
- 386 Targeting by direct visualization versus atlas coordinates in subthalamic nucleus deep brain stimulation surgery
T.S. Marios, B.J. Efstathios, S. Pantelis, T. Georgios, K.T. Andreas, S.E. Damianos (Athens, Greece)
- 387 Bilateral subthalamic deep brain stimulation impairs cognitive-motor function under dual-task conditions in Parkinson's disease patient
J.L. Alberts, C. Voelcker-Rehage, K. Hallahan, M. Vitek, R. Bamzi, M. Rueda, J.L. Vitek (Cleveland, OH)
- 388 Spheramine improves health-related quality of life in patients with moderate to advanced Parkinson's disease
N.P. Stover, R.L. Watts, A. Freeman, M. DeLong, B.A.E. Roy, R. Elke (Birmingham, AL)
- 389 Lateralization of Parkinson's disease and agenesis of the corpus callosum
N. Agrawal, L. Verhagen, C. Comella, E. Zaubler, R.A.E. Bakay (Chicago, IL)
- 390 Accurate and prospective recording of DBS adverse events: Do these complications affect quality of life?
A. Burdick, H.H. Fernandez, C. Jacobson, M.S. Okun, K.D. Foote (Gainesville, FL)
- 391 Predictive value of intraoperative macrostimulation for post operative activation of the internal capsule in STN-DBS
M. Rueda-Acevedo, B. Walter, V. Hernando-Requejo, J.L. Vitek (Medellin, Colombia)
- 392 Aggravated stuttering following subthalamic deep brain stimulation in Parkinson's disease
M. Toft, E. Dietrichs (Oslo, Norway)
- 393 Early brain abscess: A rare complication of deep brain stimulation (DBS)
L.C. Shih, E. Papavassiliou, D. Tarsy (Boston, MA)

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- 394 Subthalamic deep brain stimulation and obsessive-compulsive symptoms in Parkinson's disease
P.G. Frisina, W. Tse, B.R. Baker, H. Shapiro, M. Tagliati, E. Hollander, W. Olanow, T.D. Hälbig (Paris, France)
- 395 The effects of globus pallidus stimulation (GPS) on static and dynamic postural control in Parkinson's disease (PD)
J.P. Rodrigues, L.G. Johnson, S.E. Walters, G.W. Thickbroom, R. Stell, F.L. Mastaglia (Perth, WA, Australia)
- 396 Sensitivity and specificity of levodopa response in predicting deep brain stimulation (DBS) outcomes
E. Furr Stimming, I.J. Oh, G. Van Horn, M.C. Schiess (Houston, TX)
- 397 Long-term consequences of weight gain after deep brain stimulation of the subthalamic nucleus
A. Samier-Foubert, S. Hivert, W. Meissner, S. Maurice-Tison, P. Burbaud, V. Rigalleau, F. Tison (Pessac, France)
- 398 The pedunculo-pontine region – the atlas view
F. Alesch, T. Czech (Vienna, Austria)
- 399 Position of activated electrode contacts and their correlation to anatomical structures in deep brain stimulation of the subthalamic nucleus for treatment of advanced Parkinson's disease
W.E. Eisner, T. Fiegele, F. Sohm, F. Primavesi, E. Wolf*, J. Mueller*, W. Poewe* (Innsbruck, Austria)
- 400 Deep brain stimulation for peripherally-induced parkinsonism
M.M. Nashatizadeh, J. Jankovic (Houston, TX)
- 401 Deep brain stimulation (DBS) parameter/s and gait in Parkinson's disease (PD)
A.I. Sarwar, E.C. Lai (Houston, TX)
- 402 Safety, tolerability and efficacy of deep brain stimulation surgery in Parkinson's disease by the method of frameless stereotaxy
C.-H. Tai, S.-H. Tseng, H.-M. Liu, R.-M. Wu (Taipei, Taiwan)
- 403 Extradural motor cortex stimulation (EMCS) improves motor symptoms in advanced Parkinson's disease (PD)
N. Modugno, F. Lena, S. Ruggieri, A. Berardelli, P. Romanelli, A. Brunetti (Campobasso, Italy)
- Education in movement disorders**
Poster numbers (404-414)
- 404 Awareness of pediatric movement disorders among pediatricians
T.B. Soman, D. Lamba, W.J. Logan, A.E. Lang (Toronto, ON, Canada)
- 405 Northumbria Parkinson's Disease Service (NPDS) and the Parkinson's Disease Society (PDS) Information Prescriptions pilot project
R. Walker, S. Corbett, H. Kirrane, A. Hand, B. Wood, K. Greenwell (North Shields, Tyne and Wear, United Kingdom)
- 406 205 hours of training to become a specialist in movement disorders in the elderly? An analysis of the allocation of trainees' time
S. Bhat, R. Grue, T. Pattison, A. Abbas, S. Briggs, J. Fox, L. Wileman, P. Baker (Salford, United Kingdom)
- 407 Neurology clinical skills in internal medicine interns: The diagnosis of Parkinson's disease
A.D. Hohler (Boston, MA)
- 408 Accessing health information: Attitudes of patients with Parkinson's disease and their use of technology
A.J. Lindahl, L.J. Teare, B.A. Castleton, C.E. Clarke, D.G. MacMahon (Coventry, United Kingdom)
- 409 Consequences of perinatal hypoxic-ischemic CNS damage
M.G. Tatsiana (Minsk, Belarus)
- 410 How Parkinson's (PD) club can improve PD care in our community?
A. Nasar, P. Dyer, C. Short, L. Wheelhouse, E. Howard, L. Wright, K. Turner (Bridlington, East Riding of Yorks, United Kingdom)
- 411 Refractory Holme's tremor following midbrain and proximal brainstem infarct – challenge in neurological rehabilitation
M.H. Desai, E. Davis (Newcastle upon Tyne, Tyne and Wear, United Kingdom)
- 412 A self-management educational intervention for veterans and spouses living with Parkinson's disease: A pilot study
N.D. Nelson, S. Moore, E.C. Lai (Houston, TX)
- 413 Improving care for people with Parkinson's disease in residential facilities: Staff educational curriculum
M. Makoutonina, R. Ianssek (Melbourne, VIC, Australia)
- 414 Hand-held electronic diary for self-assessment of motor function and medication compliance in patients with Parkinson's disease
R. Bhidayasiri, H. Ling, L. Kaewilai (Bangkok, Thailand)
- History**
Poster numbers (415-420)
- 415 Pellagra and parkinsonism: Redox the common denominator?
A.C. Williams (Birmingham, United Kingdom)
- 416 The hydrogen cycle in historical perspective: Redox as maker and breaker
A.C. Williams (Birmingham, United Kingdom)
- 417 Historical underpinnings of the term "essential tremor" in the late nineteenth century
E.D. Louis, E. Broussolle, C.G. Goetz, P. Krack, P. Kaufmann, P. Mazzoni (New York, NY)
- 418 The development of stereotactic procedures in the posterior subthalamic area in the treatment of movement disorders
P. Blomstedt, U. Sandvik, A. Fytagoridis (Umea, Sweden)
- 419 The first stereotactic frame? French of course!
A.L. Benabid, S. Chabardes, E. Seigneuret, N. Torres, J.F. LeBas (Grenoble, France)
- 420 A film of patients with movement disorders made in Queen Square in the mid-1920's by Samuel Alexander Kinnier Wilson
E.H. Reynolds, A. Lees (London, United Kingdom)
- Quality of life/caregiver burden in movement disorders**
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- 421 Ultrasonographic findings of shoulder disorders in patients with Parkinson's disease
S.-B. Koh, K. Oh, J.H. Kim, B.K. Park, J.S. Yoon, S.-H. Lee (Seoul, Korea)
- 422 Quality of life impact of the long-term therapy of blepharospasm and facial hemispasm with botulinum toxin A. A retrospective assessment of ten years of treatment
H. Streitova, M. Bares (Brno, Czech Republic)

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- 423 Improving care for people with Parkinson's in residential facilities: Residents outcomes following staff education
M. Makoutonina, R. Insek (Melbourne, VIC, Australia)
- 424 Deficits in self-care reduce quality-of-life in Parkinson's disease
G. Kleiner-Fisman, M.B. Stern, D.N. Fisman (Toronto, ON, Canada)
- 425 What contributes to caregiver strain in Parkinson's disease?
L. Swearingin, D. Breslow, R. Dunlop, A. McCoy, T. Simuni, L. Marsh (Baltimore, MD)

Tics/Stereotypies

Poster numbers (426-438)

- 426 Double-blind placebo randomized study of using levetiracetam to treat tics in children and adolescents with Tourette syndrome
Y.M. Awaad (Riyadh, Saudi Arabia)
- 427 Deep brain stimulation in Tourette's syndrome
A. Koulousakis, D. Lenartz, J. Kuhn, J. Klosterkoetter, V. Sturm (Koeln, Germany)
- 428 Prolonged follow-up results of deep brain stimulation (DBS) in a series of 31 patients affected with Tourette syndrome (TS) and refractory to conservative treatments
D. Servello, M. Sassi, A. Brambilla, S. Defendi, M. Porta (Milan, Italy)
- 429 Deep brain stimulation (DBS) for Tourette syndrome (TS). Effects on psycho-behavioural comorbidities, and perception of the pulse-generator
D. Servello, M. Sassi, A. Brambilla, S. Defendi, M. Porta (Milan, Italy)
- 430 One to three year neuroleptic-induced weight gain in Tourette syndrome children
R.S. De Grauw, J. Li, D.L. Gilbert (Cincinnati, OH)
- 431 Treatment outcome correlates with knowledge of disease in hemifacial spasm
S.K.S. Ting, S. Hameed, S.F. Chong, K. Hussein, S.-Y. Lum, L.-L. Chan, E.-K. Tan (Singapore, Singapore)
- 432 Case control MR-CISS and 3-D TOF MRA imaging study of medullary compression and hypertension in hemifacial spasm
L.L. Chan, E.K. Tan (Singapore, Singapore)
- 433 Topiramate in treatment of Tourette's syndrome (TS)
S.-H. Kuo, J. Jimenez-Shahed (Houston, TX)
- 434 Impact of placebo assignment in clinical trials of tic disorders
E. Cubo, M. Gonzalez, H. Singer, M.E. Mahone, K.R. Muller-Vahl, L. Scahill, R. de la Fuente-Fernandez (Burgos, Spain)
- 435 Efficacy of donepezil for tic reduction
J. Aldred, B. Lobb, K. Chung (Portland, OR)
- 436 Streamlined video analysis in clinical trials of Tourette syndrome
B.N. Maddux, J.M. Albert, D.E. Riley, R.J. Maciunas (Cincinnati, OH)
- 437 Female gender is an independent predictor of tic severity in adult Tourette syndrome
D.G. Lichter (Buffalo, NY)
- 438 Unusual motor-phonetic tic mimicking essential palatal myoclonus (EPM)
O.R. Adam, J. Jankovic (Houston, TX)

POSTER SESSION 2

Wednesday, June 25, 2008 – 12:30 to 14:30
Northeast Exhibit Hall, Lower Level, Hilton Chicago
Poster Viewing: 9:00 to 17:00
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Ataxia

Poster numbers (439-474)

- 439 A comparative study of multimodal evoked potentials in spinocerebellar ataxia types 1, 2 and 3
P.Kr. Pal, V. Chandran (Bangalore, Karnataka, India)
- 440 Spinocerebellar ataxias type 1, 2 and 3: A study of heart rate variability
C. Pradhan, B.S. Yashavantha, P.K. Pal, T.N. Sathyaprabha (Bangalore, Karnataka, India)
- 441 Skeletal muscle energy metabolism in Machado-Joseph disease
I. Yabe, K.K. Tha, S. Terae, K. Okita, H. Sasaki (Sapporo, Japan)
- 442 Spinocerebellar ataxias in Chinese patients: Genetic analysis of familial and sporadic cases
B. Tang, J. Wang, H. Jiang, S. Zhang, L. Shen, K. Xia (Changsha, Hunan, China)
- 443 PET evidence of cerebellar hypometabolism in a patient with familial episodic ataxia-myokymia syndrome
J.-S. Kim, K.-S. Lee, H.-T. Kim (Seoul, Korea)
- 444 Gait disturbances in *Atm*^{-/-} mice, a model of ataxia-telangiectasia
A.G. Kale, I. Amende, C.J. Rothblum-Oviatt, M. Weil, T.G. Hampton (Boston, MA)
- 445 Olfactory impairment in Machado-Joseph disease (SCA3)
N.R. McFarland, L.R. Sudarsky, L. Corwin, J.H. Friedman (Charlestown, MA)
- 446 Symptomatic episodic ataxia: One case
M. Anheim, C. Boulay, C. Marcel, C. Tranchant (Strasbourg, France)
- 447 Spinocerebellar ataxia 8: Variable phenotype and unique pathogenesis
A. Gupta, J. Jankovic (Baltimore, MD)
- 448 SCA17 in Mexican-American families: Novel phenotype and association with IT15 gene expansion
J.E. Landes, C. Greco, A.H. Koeppen, V. Wheelock (Sacramento, CA)
- 449 Successful intravenous immunoglobulin treatment of a patient with cerebellar ataxia with anti-GAD antibodies
C. Garcia, O. de Fabregues, J.M. Martinez, A. Saiz, F. Graus, G. Ribera (Sabadell, Barcelona, Spain)
- 450 Falls in spinocerebellar ataxias: Results of the EuroSCA fall study
E.M.R. Fonteyn, T. Schmitz-Hubsch, C.C. Verstappen, L. Baliko, B.R. Bloem, S. Boesch, L. Bunn, P. Charles, A. Durr, A. Filla, P. Giunti, C. Globas, T. Klockgether, B. Melegh, M. Munneke, M. Pandolfo, A. de Rosa, L. Schols, D. Timmann, B.P.H. Kremer, B.P.C. van de Warrenburg (Nijmegen, Netherlands)
- 451 Alexander disease causing hereditary, late-onset ataxia in two sibs
C.C.S. Delnooz, R.J. de Graaf, G.S. Salomons, H.J. Schelhaas, B.P.C. van de Warrenburg (Nijmegen, Netherlands)

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- 452 Sporadic and hereditary spinocerebellar ataxia, a 204-patient retrospective cohort study
A. Degardin, I. Vuillaume, D. Dobbelaere, B. Sablonniere, S. Defoort-Dhellemmes, A. Destee, L. Defebvre, D. Devos (Lille, France)
- 453 Survivors from b-fluoroethyl acetate (a derivative of fluoroacetate, Compound 1080) poisoning shows selective cerebellar toxicity: A model of cerebellar degeneration
J.-M. Kim, B.S. Jeon (Seungnam, Korea)
- 454 The effect of cognitive task on postural stability in patients with spinocerebellar ataxia
F. Ozcan, M. Demirkiran, Y. Sarica (Adana, Turkey)
- 455 Dual task interference during gait in patients with ataxia
F. Ozcan, M. Demirkiran, Y. Sarica (Adana, Turkey)
- 456 Ataxia with vitamin E deficiency in Turkey
H.A. Hanagasi, B. Castellotti, C. Mariotti, C. Gellera, H. Gurvit, J. Yazici, M. Emre (Istanbul, Turkey)
- 457 Suppression of neurodegeneration in SCA3/MJD transgenic drosophila by the molecular chaperone HSP22
H. Jiang, Q.-h. Li, J.-p. Yi, Y.-f. Zhou, B.-s. Tang (Changsha, Hunan, China)
- 458 Mapping a novel locus to chromosome 16q12.1-q13 in a Chinese autosomal dominant SCA family
H. Jiang, X.-w. Song, X.-x. Cui, J.-l. Wang, B. Tang (Changsha, Hunan, China)
- 459 Spinocerebellar ataxia type 10: Frequency of epilepsy in a large sample of Brazilian patients
H.A.G. Teive, R.P. Munhoz, W.O. Arruda, L.C. Werneck, T. Ashizawa, S. Raskin (Curitiba, Parana, Brazil)
- 460 Spinocerebellar ataxia type 6 in Brazil: Case reports
H.A.G. Teive, R.P. Munhoz, S. Raskin, L.C. Werneck (Curitiba, Parana, Brazil)
- 461 The tale of spinocerebellar ataxia type 10 in Brazil: Travels of a gene
H.A.G. Teive, W.O. Arruda, S. Raskin, T. Ashizawa, L.C. Werneck (Curitiba, Parana, Brazil)
- 462 Alexanders disease presenting as cerebellar ataxia
H.A.G. Teive, R.P. Munhoz, S. Raskin, L.C. Werneck, M. van der Knaap, C. Twardowschy, O. Shelp, N. Becker (Curitiba, Parana, Brazil)
- 463 Uncertain cause of slow progressive cerebellar ataxia with prominent cervical dystonia: Is it really a new entity?
J.-H. Park, Y.-H. Yun, S.-R. Ha, S.-A. Park, T.-K. Lee, K.-B. Sung (Bucheon-si, Gyeonggi-do, Korea)
- 464 ARSACS in the Netherlands: A frequent cause of recessive cerebellar ataxia?
S. Vermeer, R.P.P. Meijer, B.J. Pijl, J. Timmermans, J.R.M. Cruysberg, M.M. Bos, H.J. Schelhaas, B.P.C. van de Warrenburg, N.V.A.M. Knoers, B.B.H.P. Kremer, H. Scheffer (Nijmegen, Gelderland, Netherlands)
- 465 Progressive cerebellar ataxia associated with dystonia, hypogonadotropic hypogonadism and chorioretinal dystrophy: A sporadic case of Boucher-Neuhauser syndrome or a new variant?
H. Ling, K. Unnwongse, R. Bhidayasiri (Bangkok, Thailand)
- 466 Symptomatic palatal tremor in a patient with mitochondrial recessive ataxia syndrome (MIRAS)
M.S. Burnett, E.P. Simpson, L.-J.C. Wong, K.A. Josephs (Rochester, MN)
- 467 Atypical presentation of Fragile X-associated tremor/ataxia syndrome: Family history of premature ovarian failure the clue to diagnosis
K.L. Poston, J. Goldman, P. Mazzoni (New York, NY)
- 468 Case report: Adult-onset familial ataxia, deafness, spasticity and leukodystrophy
S.-C. Lai, C.-S. Lu (Taoyuan, Taiwan)
- 469 Acute cerebellar ataxia: A benign presentation of paralytic shellfish poisoning
T.A. Mestre, P. Vale, R. Peralta, M. Coelho, J. Ferreira, M. de Carvalho (Lisbon, Portugal)
- 470 An atypical variant of ataxia telangiectasia presenting as idiopathic torsion dystonia
W. Meissner, D. Stoppa-Lyonnet, J. Couturier, J. Hall, P. Henry, F. Tison (Pessac, France)
- 471 Quantitative evaluation of balance in patients with spinocerebellar ataxia type 1: A case control study
G. Mohan, P.Kr. Pal, K.R. Sendhil, T. Kandavel, B.R. Usha (Bangalore, Karnataka, India)
- 472 Spinocerebellar ataxia 12 found only in Agarwals in India
A.K. Srivastava, M. Behari (New Delhi, Delhi, India)
- 473 Treatment with the GABAergic drug gabapentin in dominant cerebellar ataxias
I.J. Posada, N. Núñez-Enamorado, J. Ruiz-Jiménez, J.F. Gonzalo-Martínez (Madrid, Spain)
- 474 Prolonged cortical silent period but normal sensorimotor plasticity in spinocerebellar ataxia 6
J.T.H. Teo, S.A. Schneider, B.J. Cheeran, M. Fernandez-del-Olmo, P. Guinti, J.C. Rothwell, K.P. Bhatia (London, United Kingdom)

Dystonia

Poster numbers (475-552)

- 475* Plasticity of sensorimotor circuits in patients with SWEDDs resembles the pattern seen in dystonia and differs from Parkinson's disease
P. Schwingenschuh, D. Ruge, C. Terranova, S.A. Schneider, P. Mir, J.C. Rothwell, K.P. Bhatia, M.J. Edwards (London, United Kingdom)
- 476* Simple and complex hand movements in patients with writer's cramp: An event-related fMRI study
P. Havránková, R. Jech, N.D. Walker, G. Operto, J. Vymazal, E. Ruzicka (Prague, Czech Republic)
- 477* Premotor-motor inhibition exhibits task-specificity in patients with focal hand dystonia
S. Piro Richardson, S. Beck, B. Bliem, M. Hallett (Albuquerque, NM)
- 478* Gray matter abnormalities in spasmodic dysphonia
K. Simonyan, C.L. Ludlow (Bethesda, MD)
- 479* Indications and results for globus pallidus internus (GPI) stimulation in perinatal hypoxic injury
N. Burger, F. Vergani, L. Cif, B. Biolsi, H. El Fertit, S. Gil Robles, X. Vasques, P. Coubes (Montpellier, France)

*These posters are also part of the Guided Poster Tours. Please see pages 70-74 for more information.

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- 480* Myeloradiculopathy secondary to cervical dystonia
D. Riicard, E. Roze, J.-F. Lepeintre, S. Thobois, M. Anheim, A. Elbaz, D. Grabli, S. Leu, J. Xie, S. Yaici, C. Tranchant, C. Mazel, P. Burbaud, P. Krack, P. Pollak, E. Broussole, M. Vidailhet (Paris, France)
- 481* Physiology of the subthalamic nucleus in patients with primary dystonia
L.E. Schrock, S. Shimamoto, J.L. Ostrem, R.S. Turner, P.A. Starr (San Francisco, CA)
- 482* A phase II, double blind randomised controlled crossover trial of dronabinol for the treatment of cervical dystonia
C. Zadikoff, P. Wadia, A. Asante, S.H. Fox (Toronto, ON, Canada)
- 483* Neurophysiological evidence for cerebellar dysfunction in primary focal dystonia
J.T.H. Teo, B.P.C. van de Warrenburg, S.A. Schneider, J.C. Rothwell, K.P. Bhatia (London, United Kingdom)
- 484* Clinical course of DYT1 dystonia patients treated with deep brain stimulation: A three to ten year-follow-up
L. Cif, X. Vasques, V. Gonzalez, S. Gavarini, B. Biolsi, G. Collod-Beroud, H. Elfertit, S. Gil Robles, P. Coubes (Montpellier, France)
- 485* Bilateral pallidal stimulation in generalised dystonia related to post-anoxic birth injury: Multicentre study
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- 487* Differentiation of botulinum toxin non-responders in idiopathic cervical dystonia utilizing diffusion tensor imaging
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- 488* Association of focal dystonia and a common SNP in the DYT1 gene
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- 489* Parkinsonism with dystonia caused by the illicit use of ephedrone
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- 490* Dystonia and progressive supranuclear palsy: A closer relation than expected
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- 493* Hyperactivation of the putamen during a discrimination task in unaffected relatives of patients with familial primary torsion dystonia
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- 494* Behavioural and neurophysiological effects of proprioceptive training in musician's dystonia
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- 505 Dependence of surround inhibition on different force levels and abnormality in dystonia
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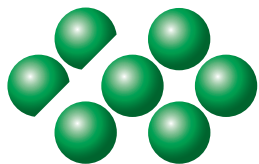
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- 510 Clinical safety of NT 201 (Xeomin®): A meta-analysis
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- 517 Retrocollis or anterocollis may predict the spreading of dystonic movements in cervical dystonia patients
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- 518 Clinical spectrum and pathological correlates of osseomuscular disability in Wilson's disease
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- 522 Sensory motor mismatch within the supplementary motor area in the dystonic monkey
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- 523 The efficacy of Dysport® (botulinum toxin type A) in the treatment of cervical dystonia: A phase III multicenter, randomized, double blind, placebo-controlled study
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- 524 Characterisation of PLA2G6 as a locus for dystonia-parkinsonism
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- 532 The temporal discrimination threshold (TDT) in patients with adult onset primary torsion dystonia (AOPTD) and their relatives – a better endophenotype?
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- 533 Localization of stimulation contacts in patients treated for segmental primary dystonia and generalized dystonia related to birth injury
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- 534 Writer's cramp, hemidystonia, and ataxia secondary to pontomedullary tumor
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- 536 Arm swing is reduced in adult onset primary cervical dystonia
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- 537 Dry eye incidence in the treated and non-treated eyes of patients with hemifacial spasm
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- 538 Changes in depression and anxiety after DBS-GPI for primary dystonia
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- 539 Somatosensory temporal discrimination in different forms of focal dystonias
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- 544 Alcohol-responsive dystonia and dystonic tremor
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- 545 A prospective survey of clinical character of patients with primary dystonia from South-West China
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- 546 Family with dopa-responsive dystonia and hypoplasia of dystonic leg
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- 547 Phenotype and genotype variations of dopa-responsive dystonia in a cohort of Taiwanese population
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- 548 Cervical dystonia and dystonic tremors following right carpal tunnel decompression
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- 550 RNA interference-mediated inhibition of wild-type torsinA expression increases apoptosis caused by oxidative stress in cultured cells
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- 551 Improvement primary focal dystonia after needle muscle puncture: Report of two cases
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- 552 Focal foot dystonia treated with botox A
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- 554 Perspectives towards predictive testing in Huntington disease
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- 555 Hyperkinetic and rigid gait in R6/2 mice, a model of Huntington's disease
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- 559 A progressive case of cerebellar ataxia without CAG repeat expansion in a family of Huntington's disease
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- 566 Neuroprotective effect of probenecid in a transgenic model of Huntington's disease
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- 569 A sensitivity comparison of clinical tests of postural instability in patients with Huntington's disease
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- 584* Effect of investigator perception of treatment efficacy on outcome measures in a clinical trial of neuroprotective agents in Parkinson disease
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- 585* The PARS study: Recruitment of a cohort at risk for Parkinson disease
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- 586* ADAGIO: A prospective, double-blind, delayed-start study to examine the potential disease-modifying effect of rasagiline in early Parkinson's disease (PD)
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- 587* PROUD: The impact of early vs. delayed treatment with pramipexole on new onset Parkinson's disease
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- 588* Pardoprinox (SLV308) in patients with early stage Parkinson's disease – a double-blind, placebo-controlled, multi-center study by the Bruegel study group
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- 589* A double-blind, randomised, placebo-controlled trial to investigate the efficacy and safety of nebicapone in levodopa-treated Parkinson's disease patients with motor fluctuations
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- 590* Safety of istradefylline as adjunctive therapy in Parkinson's disease: Pooled analysis of 5 placebo-controlled 12- to 16-week studies
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- 591* Duodenal levodopa infusion for advanced Parkinson's disease: 30-month treatment outcome
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- 592* Transdermal delivery of a levodopa produg; a pilot clinical trial
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- 594* Comparison of adjunctive ropinirole 24-hour prolonged release and ropinirole immediate release in patients with advanced Parkinson's disease: A per-protocol analysis of the PREPARED study
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- 595* Long-term safety and tolerability of transdermal rotigotine in advanced Parkinson's disease
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- 573 Botulinum toxin A in electrical injury-induced cervical myoclonus
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- 574 Efficacy of fluoxetine in the treatment of refractory spinal myoclonus
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- 575 Cortical myoclonus masquerading as spinal myoclonus
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- 578 Primary progressive myoclonus of aging
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- 579 Familial cortical myoclonic tremor with occipital lobe epilepsy in a Thai family
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- 581 Theta-burst stimulation may modulate myoclonus in cortico-basal degeneration. A case report
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- 582* Unified dyskinesia rating scale: Presentation and clinimetric profile
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- 596* The long-term antidyskinetic effect of amantadine therapy in Parkinson's disease patients
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- 597* Best medical therapy vs. deep brain stimulation for PD: Six month results from a multi-site randomized trial
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- 598* 5-year update of the safety and efficacy of unilateral intrastriatal implantation of Spheramine®
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- 599* Treating festinating speech with altered auditory feedback in Parkinson's disease – the first report of a clinical trial
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- 600* Randomised controlled trial of memantine for dementia associated with Parkinson's disease
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- 601* Design of a randomized, placebo-controlled trial of pramipexole in patients with Parkinson's disease and depressive symptoms
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- 602 Association of poor metabolizer genotypes (CYP2D6 and NAT2) with Parkinson's disease
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- 603 A pilot RCT of occupational therapy to optimise independence in Parkinson's disease (PD OT)
C.E. Clarke, A. Furmston, E. Morgan, S. Patel, C. Sackley, M. Walker, K. Wheatley (Birmingham, West Midlands, United Kingdom)
- 604 Duloxetine versus sertraline in treatment of depression in Parkinson's disease
L. Scarzella, G. Scarzella, A. Costanza, M. Di Stasi, K. Vastola (Torino, Italy)
- 605 Monoamine oxidase B inhibitors versus other dopaminergic agents in early Parkinson's disease: A systematic review of the literature
R. Caslake, A. MacLeod, N.J. Ives, R.L. Stowe, C.E. Counsell (Aberdeen, Scotland, United Kingdom)
- 606 Orodispersible priribedil (S 90049) to abort OFF episodes in apomorphine-responder patients with advanced Parkinson's disease: A single dose randomized, double-blind, placebo-controlled cross-over study
O. Rascol, O. Blin, A.-M. Bonnet, P. Cesaro, P. Damier, F. Durif, S. Pennaforte, Study Investigators (Toulouse, France)
- 607 Adding a dopamine agonist to pre-existing levodopa therapy versus levodopa therapy alone in advanced Parkinson's disease: A meta-analysis
R. Talati, K. Reinhart, A.A. Patel, W.L. Baker, C.I. Coleman (Hartford, CT)
- 608 Benefit of music therapy in patients with Parkinson disease: A randomized controlled trial
A. Shankar, N. de Bruin, S. Bonfield, L. Derwent, M. Eliasziw, B. Hu, L. Brown, O. Suchowersky (Calgary, AB, Canada)
- 609 Overnight switch from pramipexole immediate release to pramipexole extended release in patients with early Parkinson's disease: The Switch trial
C. Debieuvre, L. Salin, O. Rascol (Reims, France)
- 610 First demographic data of the Transdermal Rotigotine Surveillance Study (TRUST)
T. Muller, M. Lorrain, R. Hilker, L. Timmermann, R.M. Ehret, H.-J. Haeck, K.-W. Leffers (Berlin Weissensee, Germany)
- 611 Effect of practice on movement reaction time and learning retention in Parkinson's disease
H.R. Rostami, H. Ashayeri, Gh. Taghizadeh, M.R. Keyhani (Tehran, Islamic Republic of Iran)
- 612 Beneficial effect of levetiracetam on levodopa-induced dyskinesias in Parkinson's disease: A double-blind, placebo-controlled, crossover study (the VALID-PD study). Preliminary results
P. Stathis, S. Konitsiotis, G. Tagaris, G. Hadjigeorgiou, V. Kiriakakis, VALID-PD Study Group (Athens, Greece)
- 613 Telemedicine versus face-to-face group voice therapy for persons with Parkinson's disease
J. Searl, K. Haring, R. Pahwa, K.E. Lyons (Kansas City, KS)
- 614 Rotigotine transdermal patch in patients with fluctuating Parkinson's disease: A survey of patients treated in a specialized Parkinson's disease hospital in Wolfach/Germany
M.H. Strothjohann, P. Franz, N. Kuehnl, D. Djundja, G.A. Fuchs (Wolfach, Germany)
- 615 Treatment outcomes of expiratory muscle strength training (EMST) on swallow function in Parkinson's disease
M.S. Troche, K.M. Wheeler, J.C. Rosenbek, N. Musson, M.S. Okun, C.M. Sapienza (Gainesville, FL)
- 616 Successful treatment of Yi-Gan San in PD (Parkinson's disease) and PDD (Parkinson's disease with dementia) patients suffered from visual hallucinations and other neuropsychiatric symptoms
T. Kawanabe, A. Yoritaka, H. Oizumi, H. Shimura, S. Tanaka (Urayasu-shi, Japan)
- 617 High-frequency rTMS over the supplementary motor area for treatment of Parkinson's disease
M. Hamada, Y. Ugawa, S. Tsuji (Tokyo, Japan)
- 618 Effective occupational therapy for persons with Parkinson's disease: Adventures in translational research
E.A. Moyer (Biddeford, ME)
- 619 Compliance of dopamine-agonist-therapy with transdermal rotigotine (Neupro®) in patients with early-stage Parkinson's disease
A. Schnitzler, K.-W. Leffers, H.-J. Haeck, O. Randerath (Duesseldorf, Germany)
- 620 Wearing-off management in Parkinson's disease: The LEVOSTAR study one-year optional follow-up
J.L. Houeto, M. Vidailhet, I. Bourdeix, K. Rerat (Rueil-Malmaison, France)
- 621 Rehabilitation on gait in Parkinson's disease with deep brain stimulation
D. Volpe (Venice-MeStre, Italy)
- 622 CSF α -Synuclein as biomarker candidate in synucleinopathies
B. Mollenhauer, C. Trenkwalder, B. Otte, B. Krastins, V. Cullen, J.J. Locascio, D. Sarracino, M.G. Schlossmacher (Ottawa, ON, Canada)

*These posters are also part of the Guided Poster Tours. Please see pages 70-74 for more information.

ABSTRACTS

- 623 Leg muscle strength is independently associated with hip bone mineral density in women with Parkinson's disease: Implications for fracture prevention
M.Y.C. Pang, M.K.Y. Mak (Hong Kong, China)
- 624 Perceived balance confidence level contributes to walking capacity in people with Parkinson's disease
M.K.Y. Mak, M.Y.C. Pang (Hong Kong, China)
- 625 Long-term improvement of non-motor symptoms and health-related quality of life with levodopa/carbidopa gel
H. Honig, T. Fox, A. Gies, S. Leimbach, A. Rüssmann, P. Odin, K. Fox (Bremerhaven, Bremen, Germany)
- 626 Orodispersible priribedil (S 90049), a non-ergot dopamine agonist, decreases the time to ON and prolongs the ON duration in a dose-dependent manner in combination with levodopa
F. Durif, E. Tolosa, J. Ferreira, G. Ebersbach, L. Ducret (Clermont-Ferrand, France)
- 627 Pedunclopontine nucleus stimulation induced monocular oscillopsia
M.U. Ferraye, B. Debù, P. Gérardin, S. Chabardès, V. Fraix, E. Seigneuret, J.-F. LeBas, A.-L. Benabid, C. Tilikete, P. Pollak (Grenoble, France)
- 628 Impact of titration regimen on the safety and tolerability of pramipexole (SLV308) in patients with advanced Parkinson's disease (PD). A study by the Pramipexole study group
R.A. Hauser, J. Bronzova, C. Sampaio, A. Lang, O. Rascol, A. Theeuwes, S. van de Witte (Tampa, FL)
- 629 Meta-analysis of the efficacy and safety of adjunct treatment to levodopa therapy in Parkinson's disease patients with motor complications
N. Ives, R. Stowe, C. Clarke, K. Handley, A. Furnston, K. Wheatley, R. Gray (Edgbaston, Birmingham, United Kingdom)
- 630 Improved symptom control with fixed dose levodopa/carbidopa/entacapone versus conventional levodopa/carbidopa as first-line levodopa therapy in early Parkinson's disease patients
R.A. Hauser, M. Panisset, G. Abbruzzese, L. Mancione, N. Dronamraju, A. Kakaricka (Tampa, FL)
- 631 The role of clusterin in Parkinson's disease
H. Vranova, M. Nevrlý, J. Mares, I. Nestrasil, D. Stejskal, P. Kanovsky (Olomouc, Czech Republic)
- 632 Local research networks are an effective way of improving recruitment to clinical trials
C.E. Rick, C. Clarke, F.P. Dowling, R. Gray, N.J. Ives, S. Patel, K. Wheatley, N.P. Winkles (Birmingham, United Kingdom)
- 633 Rasagiline in daily clinical use – a post-marketing observational study in parkinsonian patients receiving monotherapy shows good efficacy and tolerability
H. Reichmann, W. Jost (Dresden, Germany)
- 634 Combination of subtle motor signs for the early diagnosis of PD in subjects with increased echogenicity of the substantia nigra
W. Ilg, I. Liepelt, C. Urban, N. Roehrich, M. Giese, D. Berg (Tubingen, Germany)
- 635 The long-term safety and tolerability of istradefylline as adjunctive therapy to levodopa in patients with Parkinson's disease (PD) and motor complications
J.M. Bertoni, 6002-INT-001 Study Group (Omaha, NE)
- 636 The addition of orally disintegrating selegiline in Parkinson's disease patients experiencing dopamine agonist related adverse events: The A to Z study
R. Pahwa, K.E. Lyons, A to Z Study Investigators (Kansas City, KS)
- 637 Home-based treadmill walking for individuals with Parkinson's disease: A pilot randomized controlled trial
C.G. Canning, N.E. Allen, V.S.C. Fung, J.G.L. Morris, C.M. Dean (Lidcombe, NSW, Australia)
- 638 The stride length-sequence effect interaction: A determinant of freezing during walking in Parkinson's disease
M. Danoudis, R. Ianseck, R. Chee, N. Georgiou-Karistianis, A. Murphy (Cheltenham, VIC, Australia)
- 639 Effects of lower-body resistance training in persons with Parkinson's disease
B.K. Schilling, M.S. LeDoux, R.F. Pfeiffer, R.E. Karlage, L.W. Weiss, M.J. Falvo (Memphis, TN)
- 640 Serum ferritin correlation study in idiopathic Parkinson disease severity and restless leg syndrome
A.Y. Eassa, G.A. Eshrawy, L.H. Sultan, O.A. Sharaki (Alexandria, Egypt)
- 641 SPECT studies in Parkinson's disease patients with dementia and hallucinations
H. Shiraishi, I. Tomita, H. Satoh, A. Satoh, M. Seto, M. Ochi, M. Tsujihata (Nagasaki, Japan)
- 642 Gait performance during simple cognitive dual task in patient with Parkinson's disease
L. Yu, M.K.Y. Mak (Hong Kong, China)
- 643 Video versus 3-dimensional movement analysis in Parkinson's disease: Preliminary results
E.L. Stack, R.M. Pickering, V.J. Pressly, T. McElwaine, J. Frankel, H.C. Roberts (Southampton, Hants, United Kingdom)
- 644 The effect of arm position on pulmonary function in subjects with Parkinson's disease and in healthy subjects
A. Genc, B. Kara, B. Donmez Colakoglu, R. Cakmur (Izmir, Turkey)
- 645 The effect of breathing exercises on pulmonary function in Parkinson's disease
A. Genc, B. Kara, B. Donmez Colakoglu, R. Cakmur (Izmir, Turkey)
- 646 Assessment of rest tremor in Parkinson's disease
A. Budzianowska, K. Honczarenko (Szczecin, Poland)
- 647 Benefits of a direct switch from levodopa/benserazide or levodopa/carbidopa to levodopa/carbidopa/entacapone on non-motor symptoms of early wearing-off in Parkinson's disease patients
H. Nissinen, K. Eggert, M. Kuoppamäki, M. Leinonen (Espoo, Finland)
- 648 Determining the benefit of levodopa/carbidopa/entacapone (Stalevo®) on the pharmacokinetic profile of levodopa: A randomized, crossover, multicenter study in patients with Parkinson's disease
R. Marttila, V. Kaasinen, P. Hartikainen, J. Lyytinen, S. Kaakkola, J. Hänninen, K. Korpela, K. Laapas, M. Kuoppamäki, J. Ellmén (Turku, Finland)
- 649 Effect of lumbosacral corset on position sense of trunk, performance and balance of Parkinson's disease patients
B. Kara, A. Genc, I. Demirbüken, B. Balci, B. Donmez Colakoglu, R. Cakmur (Izmir, Turkey)
- 650 One year follow-up study of the zonisamide (ZNS) efficacy on parkinsonism
Y. Kajimoto, I. Nakanishi, T. Kondo (Wakayama, Japan)
- 651 Safety and efficacy of mianserine on psychosis in Parkinson's disease
K. Fujimoto, T. Kawakami, K. Ikeguchi, I. Nakano (Shimotsuke, Tochigi, Japan)

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- 652 Dosing of ropinirole 24-hour prolonged release in Parkinson's disease: Clinical trial data and relevance to clinical practice
F. Stocchi, D. Tompson, L. Giorgi (Rome, Italy)
- 653 Evaluation of open-label rotigotine treatment in advanced Parkinson's disease
K.E. Lyons, R. Pahwa, B. Boroojerdi (Kansas City, KS)
- 654 Significant benefits of the direct switch from conventional levodopa/benserazide or levodopa/carbidopa to levodopa/carbidopa/entacapone in Parkinson's disease patients with early wearing-off
K. Eggert, W.H. Oertel, Ö. Skogar, K. Amar, L. Luotonen, H. Nissinen (Marburg, Germany)
- 655 Comparison of adjunctive ropinirole 24-hour prolonged release and ropinirole immediate release in patients with advanced Parkinson's disease: The PREPARED study
F. Stocchi, B. Hunter, L. Giorgi, A.H.V. Schapira (Rome, Italy)
- 656 Influence of tempo-rhythmic correction of gait method on expenses for pharmacological treatment of Parkinson's disease
V.G. Abramov, D.V. Pokhobov (Krasnoyarsk, Russian Federation)
- 657 Ambulatory monitoring of motor fluctuations and freezing of gait: Objective assessment of the efficacy of pharmacological treatments in Parkinson's disease
S.T. Moore, H.G. MacDougall, W.G. Ondo (New York, NY)
- 658 Safety and tolerability of isradipine, a dihydropyridine calcium channel blocker, in patients with early Parkinson's disease
T. Simuni, A. Martel, C. Zadikoff, A. Videnovic, L. Vainio, F. Weaver, K. Williams, D.J. Surmeier (Chicago, IL)
- 659 Efficiency of a motor training with rhythmical cues to improve balance and its effects in gait and no motors aspects in Parkinson's disease
T. Capato, M. Mathias, M.E. Piemonte (Sao Paulo, Brazil)
- 660 Continuous lisuride SubQ applied via minipump compared to oral ropinirole, pramipexole, cabergoline in patients with advanced Parkinson's disease. First results from the placebo-controlled double-blind phase
G. Ebersbach, C. Gebert, F. Stocchi, Calipso Study Group (Beelitz-Heilstaetten, Germany)
- 661 Effect of rotigotine (Neupro) in the elderly with Parkinson's disease
S.H. Isaacson, M.L. Ailincal, D.L. Kreitzman (Boca Raton, FL)
- 662 Deep brain stimulation for early Parkinson's disease: Recruitment experience from a pilot clinical trial
C.E. Gill, S.G. Finder, M.J. Bliton, T.L. Davis, P.E. Konrad, D. Charles (Nashville, TN)
- 663 Effects of rivastigmine on postural instability and gait in non-demented Parkinsons disease patients: A pilot study
R.P. Munhoz, H.A.G. Teive, A.P. Gomes, C.C. Silva (Curitiba, Parana, Brazil)
- 664 Continuous lisuride infusion in advanced PD patients unresponsive to oral treatments – long term results
F. Stocchi, L. Vacca, D. Palla, C. Gebert (Berlin, Germany)
- 665 A randomized, double-blind, placebo-controlled study of atomoxetine for freezing of gait in Parkinson's disease
M.M. Nashatizadeh, J.E. Jimenez, A.L. Davidson, J. Jankovic (Houston, TX)
- 666 Adjunctive rasagiline provides significant benefits in all cardinal symptoms in patients with moderate to advanced Parkinson's disease
L.W. Elmer, Presto, LARGO Investigators (Toledo, OH)
- 667 Walking while listening to music improves gait performance in Parkinson's disease
N. de Bruin, S. Bonfield, B. Hu, O. Suchowersky, J. Doan, L. Brown (Lethbridge, AB, Canada)
- 668 Adverse events following best medical therapy or deep brain stimulation for Parkinson's disease
F.M. Weaver, VA CSP#468/NINDS Study Group (Hines, IL)
- 669 Efficiency of evidence-based physiotherapy for Parkinson's disease: the ParkinsonNet Trial
M. Munneke, S. Keus, M. Nijkrake, G. Kwakkel, H. Berendse, R. Roos, G. Borm, B. Bloem (Nijmegen, Netherlands)
- 670 Identification of a peripheral biomarker for parkinsonism syndrome (PS) using non-biased proteomics in blood
D.S. Russell, L. Ho, D. Jennings, S. Yemul, A.O. Koren, G.D. Tamagnan, K.L. Marek, G.M. Pasinetti (New Haven, CT)
- 671 A retrospective analysis of the performance of the scale for assessment of positive symptoms (SAPS) scale in patients with psychosis and Parkinson's disease (PDP)
R. Mills, A. Johnson, H. Williams, J.H. Friedman (San Diego, CA)
- 672 An open-label extension study to determine the safety of pimavanserin in patients with Parkinson's disease and psychosis
R. Mills, A. Johnson, D. Bahr, H. Williams, S. Revell (San Diego, CA)
- 673 A double-blind, placebo-controlled, dose-escalation trial of pimavanserin in Parkinson's disease and psychosis
R. Mills, S. Revell, D. Bahr, H. Williams, A. Johnson, J.H. Friedman (San Diego, CA)
- 674 Tolerability and efficacy of switching from swallowed selegiline to zydys selegiline (Zelapar) in patients with Parkinson's disease
W.G. Ondo, C. Hunter, S. Isaacson, D. Silver, J. Tetrud, M. Stuart, A. Davis (Houston, TX)
- 675 Carry-over effects of inpatient rehabilitation in Parkinson's disease
C.L. Martin, M.E. Morris, R. Iansek (Parkville, VIC, Australia)
- 676 Long-term treatment complications: Vascular parkinsonism versus idiopathic PD
M. Arnaoutoglou, G. Spanos, N. Arnaoutoglou, V. Costa, S. Baloyanis (Thessaloniki, Greece)
- 677 No case reports of cardiac valvulopathy with lisuride consistent with 5-HT2B receptor antagonism
K.P. Latté, C. Gebert, D. Palla, H. Palla (Appenzell, Switzerland)

POSTER SESSION 3

Thursday, June 26, 2008 – 12:30 to 14:30
Southwest Exhibit Hall, Lower Level, Hilton Chicago
Poster Viewing: 9:00 to 17:00
Authors Present: 12:30 to 14:30
Poster Numbers: 678-1210

Choreas (non-Huntington's disease) **Poster numbers (678-690)**

- 678 Striatal high FDG uptake and effects of pallidotomy in hemichorea-hemiballismus with hyperglycemia and striatal T1-hyperintensity
T. Hashimoto, K. Oguchi, S. Hiyayama, K. Kitazawa, T. Goto (Matsumoto, Japan)

ABSTRACTS

- 679 Lead toxicity can lead to chorea
M. Spitz, L.T. Lucato, M.S. Haddad, E.R. Barbosa (Rio de Janeiro, Brazil)
- 680 Clinical features of a new family with benign hereditary chorea carrying a novel TITF-1 mutation
G. Zorzi, F. Invernizzi, F. Zibordi, C. Costa, C. Ciano, B. Garavaglia, N. Nardocci (Milano, Italy)
- 681 An algorithm for the evaluation of chorea
R.H. Walker (Bronx, NY)
- 682 Spectrum of chorea in children: Experience from a pediatric neurology center
D. Ghosh, K. Velayudam, V. Rajasekaran, G. Erenberg (Cleveland, OH)
- 683 Autoimmune chorea: 2 cases with radiological and clinical correlations
J.S. Hui, J. Go, P.M. Girard (Los Angeles, CA)
- 684 Hypoglycemia-induced chorea
E.J. Chung, S.J. Kim (Busan, Korea)
- 685 Choreoathetosis and sub-clinical hypothyroidism
M.T. Ahmad, S. Hameed, E.-K. Tan (Singapore, Singapore)
- 686 The prevalence and severity of OCD and ADHD in children diagnosed with Sydenham's chorea: A long-term follow-up
T.D. Lipps, K.R. Ridel, D.L. Gilbert (Cincinnati, OH)
- 687 Choreoathetosis after cardiopulmonary bypass, cardiac arrest and hypothermia in an adult
S.A. Kareus, J. Steffens (Salt Lake City, UT)
- 688 Kabuki syndrome with chorea: Case report and review
M.U. Farooq, R.A. Aburashed, A.C. Bhatt, S.T. DeRoos, B.W. Betz, K.L. Chillag (East Lansing, MI)
- 689 Benign hereditary chorea: Clinical and neuroimaging data from a family with a new mutation of the thyroid transcription factor-1 gene
E. Salvatore, L. Di Maio, E. Zampella, M. Ferrara, S. Pappatà, P.E. Macchia, A. Filla, G. De Michele (Napoli, Italy)
- 690 Chorea associated with the brain stem lesions
M. Kitami, S. Nakamura, N. Izawa, H. Takubo (Koto-ku, Tokyo, Japan)
- 696 Distribution of the P3 components, P3a and P3b: A neurophysiological approach of selective attention disorders in Parkinson's disease
M.-P. Perriol, M. Delliaux, J.-L. Bourriez, P. Derambure, A. Destee, L. Defebvre, K. Dujardin (Lille, France)
- 697 Postural tremor in X-linked bulbospinal muscular atrophy (BSMA)
R. Hanajima, Y. Terao, S. Inomata-Terada, M. Hamada, A. Yugeta, H. Matsumoto, T. Yamamoto, S. Tsuji, Y. Ugawa (Tokyo, Japan)
- 698 Electrocutaneous reflex in dystonia and Parkinson's disease
M.-W. Seo, S.-Y. Jeong (Jeonju, Jeonbuk, Republic of Korea)
- 699 Reaction time (RT) as a clinical marker in Parkinson's disease
S. Papapetropoulos, A. Guevara, B. Scanlon, C. Sengun, A. Russell, B. Levin, H. Katzen, C. Singer (Miami, FL)
- 700 Different strategies for finger tapping execution in Parkinson's disease and essential tremor
E. Pelosin, L. Avanzino, C. Ogliaastro, M. Bove, C. Trompetto, G. Abbruzzese (Genova, Italy)
- 701 The novelty P3 in the subthalamic nucleus (STN). An intracerebral recording study
M. Bocková, J. Chládek, P. Jurák, J. Haláček, I. Rektor (Brno, Czech Republic)
- 702 Oculomasticatory myorhythmia and sleep disruption in Whipple disease: A longitudinal study
P. Cortelli, F. Provini, R. Vetrugno, F. Pizza, G. Calandra-Buonaura, G. Pierangeli, P. Montagna (Bologna, Italy)
- 703 Movement disorders in extremely low birth weight (ELBW) children
D.N. Kountouris, K.K. Koutsobelis, A.S. Bougioukou (Athens, Greece)
- 704 'Typing tremor' as a manifestation of psychogenic writer's cramp
R. Kuriakose, G. Castillo, R. Chen (Toronto, ON, Canada)
- 705 Dual electromyographic rhythm in a case of Holmes tremor secondary to thalamic tumor
R. Kuriakose, R. Chen (Toronto, ON, Canada)
- 706 Interactions between the premotor and motor cortices in Parkinson's disease
G. Castillo, R. Kuriakose, K. Udupa, I.-J. Yeh, B. Elahi, U. Saha, C. Gunraj, Z. Ni, R. Chen (Toronto, ON, Canada)
- 707 The role of cerebellum on rhythm generation in essential tremor: An rTMS study
L. Avanzino, A. Tacchino, C. Ogliaastro, M. Bove, C. Trompetto, G. Abbruzzese (Genoa, Italy)
- 708 Neurophysiological recordings from the thalamus of patients with Tourette syndrome
S. Marceglia, S. Mrakic-Spota, A. Stangoni, D. Servello, M. Sassi, M. Tiriticco, C. Menghetti, M. Porta, A. Priori (Milan, Italy)
- 709 Different retinal disease mechanisms behind simple visual hallucinations in dementia with Lewy bodies and in Parkinson's disease
D. Devos, M. Tir, S. Defoort-Dhelemmes, C.-A. Maurage, A. Destée, L. Defebvre (Lille, France)
- 710 Intermanual difference of surround inhibition in the human motor cortex
H.-W. Shin, Y.H. Sohn (Seoul, Korea)
- 711 Hemimasticatory spasm due to a pontine cavernoma
Z. Mari, M.K. Floeter, M. Hallett, H. Jinnah (Baltimore, MD)
- 712 The repetitive transcranial magnetic stimulation of the inferior frontal cortex modulates cognitive activities in the subthalamic nucleus
M. Balaz, H. Srovnalova, I. Rektorova, I. Rektor (Brno, Czech Republic)

Clinical Electrophysiology
Poster numbers (691-719)

- 691 Diagnostic sampling in Lyme's disease cases with movement disorders
S.G. Echebarria (Las Arenas, Spain)
- 692 Motor Lewy bodies disease and tremor – expressions
S.G. Echebarria (Las Arenas, Spain)
- 693 Spreading, overflow, overactivity and composition in associated-conjugated movements
S.G. Echebarria (Las Arenas, Spain)
- 694 Movement disorders in Whipple's disease patients samples: Diagnoses and classification work-up
S.G. Echebarria (Las Arenas, Spain)
- 695 Disruptions of the PPI of the N100/P200 component of auditory event-related potentials, prior to dementia in Huntington's disease (HD): A marker for cognitive decline
M.-P. Perriol, A. Delval, M. Delliaux, P. Krystkowiak, L. Defebvre, A. Destée, P. Derambure, K. Dujardin (Lille, France)

ABSTRACTS

- 713 Paraneoplastic limb myorhythmia: Brainstem or spinal generator?
P. van Meerbeeck, J.-P. Lefaucheur, R. Ahdab, G. Fenelon (Creteil, France)
- 714 Sensorimotor integration abnormalities in corticobasal degeneration
C. Terranova, J. Teo, P. Schwingenschuh, L. Massey, A. Lees, A. Quartarone, K. Bhatia, J. Rothwell (Messina, Italy)
- 715 Visuomotor lexical semantic reaction times are preferentially handled in the right hemisphere in patients with persistent developmental stuttering
M. Sommer, E.J. Hunter, K. Knappmeyer, A. Wolff von Gudenberg, N. Spindler, W. Paulus (Goettingen, Germany)
- 716 A case of painful hemimasticatory spasm with facial hemihypertrophy responsive to botulinum toxin
H.-I. Ma, Y.J. Kim, J.-H. Kim, M.-S. Oh, B.-C. Lee (Anyang, Gyeonggi-Do, Korea)
- 717 Electrical impedance myography (EIM) for quantification of cervical dystonia (CD)
C. Lungu, A.W. Tarulli, L.P. Garmirian, P.M. Fogerson, D. Tarsy, S.B. Rutkove (Boston, MA)
- 718 Proper facilitation technique for bilateral motor evoked potentials by transcranial magnetic stimulation
J.-Y. Lim, W.B. Park (Seongnam, Republic of Korea)
- 719 Certain tremor parameters can differentiate PD tremor from pseudoparkinsonian tremor in patients with normal DAT scans (SWEDDs)
P. Schwingenschuh, D. Ruge, C. Terranova, M.J. Edwards, S.A. Schneider, L. Silveira-Moriyama, A.J. Lees, P. Mir, J. Rothwell, K.P. Bhatia (London, United Kingdom)

Drug-Induced Movement Disorders**Poster numbers (720-732)**

- 720 Aripiprazole induced tardive dyskinesia in a patient with Parkinson's disease
P. Agarwal, A. Griffith, D.A. Hall (Kirkland, WA)
- 721 Dyskinetic variant of neuroleptic malignant syndrome precipitated by naloxone given to reverse opioid overdose in a 57-year-old woman, a smoker, on bupropion and duloxetine
C. Armon, E. Green, E. Taylor, A. Michelucci (Springfield, MA)
- 722 Interactions between antagonists of group 5 metabotropic glutamate receptors (mGluR5) and L-DOPA in rat models of Parkinson's disease and L-DOPA-induced dyskinesia
A. Dekundy, F. Mela, M.C.J. van der Elst, M. Pietraszek, W. Danysz, M.A. Cenci (Frankfurt am Main, Germany)
- 723 Drug induced reversible parkinsonism: A report of 5 cases
G.R.K. Sarma, M. Thomas, R. Ak (Bangalore, Karnataka, India)
- 724 Tardive dystonia (TD) due to aripiprazole: Case report and literature review
C. Lungu, L. Shih, D. Tarsy (Boston, MA)
- 725 Reversible chorea associated with citalopram treatment
F. Luessi, J. Knabe, T. Vogt (Mainz, Germany)
- 726 Deep brain stimulation for tardive dystonia in patients without a history of psychosis
H.-H. Capelle, C. Blahak, C. Schrader, T. Kinfe, H. Baezner, R. Dengler, J.K. Krauss (Hannover, Germany)
- 727 Randomized, placebo-controlled, double-blinded, cross-over study of anti-dyskinetic effects of memantine in severely dyskinetic Parkinson's disease patients
H. Widner, K. Wictorin (Lund, Sweden)
- 728 Aripiprazole associated movement disorders
P.G. Aia, C.D. Esper, J.L. Juncos, S.A. Factor (Atlanta, GA)
- 729 A case of aripiprazole-induced chorea
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- 730 A case report of rasagiline induced serotonin syndrome
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- 731 Selective A2a antagonist SCH 412348 blocks haloperidol- and risperidone-induced extrapyramidal syndrome in haloperidol-sensitized Cebus apella nonhuman primates
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- 732 A proteomics and motor-function analysis of the effects of simvastatin on mitochondrial proteins in a rotenone rat model of Parkinson's disease
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- 733 Essential tremor is less prevalent than Parkinson's disease in Arabic villages of Wadi Ara
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- 734 The incidence and long-term outcome of parkinsonian disorders in North East Scotland (the PINE study): Methods and initial recruitment
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- 735 The prevalence and risk factors of Parkinson disease among Thai boxers
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- 737 Nation-wide survey for severe encephalitis of unknown etiology with prolonged clinical course in Japan
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- 738 Women with Parkinson's disease and their reproductive characteristics: A case control study
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- 740 Clinical features in early Parkinson's disease (PD) predict survival
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- 741 Prospective 1-year clinical follow-up of an incident cohort with Parkinson's disease and other parkinsonisms. The PRIAMO study
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- 742 Occupations and Parkinson's disease in Hong Kong
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- 768 Psychiatric comorbidities in a Brazilian sample of primary focal dystonia in comparison with hemifacial spasm
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- 778 Body composition in Parkinson's disease
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- 802 Corticobasal degeneration (CBD) presenting as complex regional pain syndrome (CRPS)
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- 803 Botulinum toxin treatment for pain in atypical parkinsonisms
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- 804 A case of Ogilvie's syndrome in a patient with progressive supranuclear palsy
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- 854 PRODEST – depressive symptoms in Parkinson's disease: A factor analysis of rating scales
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- 855 Case series: The effect of deep brain stimulation surgery on repetitive behavior in Parkinson patients: A report of three cases
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- 856 Prevalence of apathy in a population of Parkinson's disease patients from Argentina
A. Bottini Bonfanti, G. Persi, J.L. Etcheverry, H. Zezza, S. Starkstein, E. Gatto (Buenos Aires, Argentina)
- 857 The olfactory loss of Parkinson's disease
R.L. Doty (Philadelphia, PA)
- 858 Prevalence of hypersexual behavior in Parkinson disease patients: Not restricted to males and dopamine agonist use
C. Cooper, A. Jadidian, M. Paggi, J. Romrell, M. Okun, R. Rodriguez, H. Fernandez (Gainesville, FL)
- 859 Comorbidity of Parkinson's disease and schizophrenia spectrum disorders
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- 860 Sense of presence in Parkinson's disease: A prospective phenomenological study
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- 861 The characteristics of visuospatial impairment in Parkinson's disease without dementia: Analysis of the results of Rey Complex Figure Test
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- 862 Impulse control disorders in Parkinson disease patients followed in a community-based neurology practice
H.D. Weiss, E. Hirsch, L. Sweringen, K. Anderson, S. Goldstein, S. Grill, S. Lehman, L. John, R.L. Margolis, G. Pontone, P.V. Rabins, J.R. Williams, L. Marsh (Baltimore, MD)
- 863 Effect of electroconvulsive therapy on catatonia in Parkinson's disease: A case report
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- 864 A case of compulsion to self-harm related to dopaminergic therapy – a new dopaminergic impulse-control disorder
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- 865 Neuropsychiatric comorbidity in DBS patients: Towards a standardised postsurgical assessment
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- 866 Impaired olfaction and subsequent risk of long-term complications of Parkinson's disease
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- 867 Frequency of impulse control disorders in "de novo" patients with Parkinson's disease
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- 868 Cognitive impairment in Parkinson's disease
A. Gunduz, S. Ertan, F. Engin, C. Aksoy Poyraz, G. Kiziltan, T. Ertan, E. Eker, S. Ozekmekci (Istanbul, Turkey)
- 869 How subthalamic area stimulation may induce limbic system activation and manic behaviour?
M. Ulla, S. Thobois, P.P. Derost, J.-J. Lemaire, I. Chereau-Boudet, I. de Chazeron, A. Schmitt, P.-M. Llorca, E. Broussolle, F. Durif (Clermont-Ferrand, France)
- 870 Anxiety and Parkinson's disease: The impact on quality of life and clinical features of the disease
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- 871 Is hemispheric dominance a risk factor to develop dopamine dysregulation syndrome (DDS), in Parkinson's disease (PD) patients taking dopamine agonists (DA)?
G.D. Moguel-Cobos, N. Thakur, A. Ahmed, R.S. Burns (Phoenix, AZ)
- 872 Epidemiology of depressive symptoms in Belgian patients suffering from Parkinson's disease: PARKIDEP
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- 873 Incidence and predictors of depression in the early Parkinson's disease: Results from the DATATOP study
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- 874 Pathological gambling and hobbyism amongst internet users: Comparison of Parkinson's disease and ALS/MND
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- 875 Involuntary emotional expression disorder in Parkinson's disease
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- 876 Impulse control disorders in Parkinson's disease associated with altered neural activity to increasing risk-reward exposure
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- 877 Effects of LSVT® on four participants with PD who received deep brain stimulation
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- 878 Voice as a dynamic system: Qualitative change and application of a theoretical perspective to the Lee Silverman Voice Treatment®
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- 879 Validation of the Parkinson's disease impulsive-compulsive disorders screening questionnaire (PICS)
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- 880 Use of latent variable modeling to explore neuropsychiatric syndromes in Parkinson's disease
S. Mavandadi, S. Nazem, A. Siderowf, J. Duda, M. Stern, D. Weintraub (Philadelphia, PA)
- 881 Emotional experience and expression in Parkinson's disease (PD)
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- 882 Clinical factors associated with anxiety in Parkinson's disease
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- 883 Development of psychosis in Parkinson's disease – a 12 year follow up study
E.B. Forsaa, J.P. Larsen, D. Aarsland, G. Alves (Stavanger, Norway)
- 884 Neuropsychiatric inventory (NPI) useful instrument in dopamine dysregulation syndrome (DDS)
S. Appel, A. Evans, A.J. Lees (London, United Kingdom)
- 885 The influence of catechol-O-methyltransferase (COMT) polymorphism on excessive daytime sleepiness in patients with Parkinson's disease
G. Opala, M. Boczarska-Jedynak, B. Jasinska-Myga, M. Smilowski, G. Klodowska-Duda, M. Bialecka (Katowice, Poland)
- 886 Description of subjective and objective swallow characteristics for people with PD
L.A. Mahler, L.O. Ramig, J. Logemann, A. Halpern (Kingston, RI)
- 887 Neurotrophic support of olfactory ensheathing cells via phosphatidylinositol 3-kinase/ Akt signaling protects of neural progenitor cells on exposure to 6-hydroxydopamine
K. Seth, N. Srivastava, R.W. Ansari, A.K. Agrawal (Lucknow, Uttar Pradesh, India)
- 888 Does cognitive impairment in Parkinson's disease predispose to dopamine dysregulation syndrome?
D. Benninger, D. Waldvogel, C.L. Bassetti (Zurich, Switzerland)
- 889 Do personality traits increase the risk of apathy in Parkinson's disease?
B. Robottom, K.E. Anderson, R.J. Mullins, P.S. Fishman, S.G. Reich, W.J. Weiner, L.M. Shulman (Baltimore, MD)

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Parkinson's disease: Dysautonomia

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- 890 The prevalence, nature and associations of urinary symptoms in idiopathic Parkinson's disease
H.C. Blackett, B. Wood, R. Walker (Ashington, Northumberland, United Kingdom)
- 891 Parkinson disease patients with rhinorrhea have better olfaction than patients without rhinorrhea
K.L. Chou, N.I. Bohnen (Ann Arbor, MI)
- 892 Hemodynamic effects of clonidine in multiple system atrophy-parkinsonism and Parkinson's disease
M. Ragothaman, S. Koshy, K.D. Subbakrishna, C.J. Mathias, U.B. Muthane (Bangalore, Karnataka, India)
- 893 Skin biopsy and small-fiber pathology in Parkinson disease: A pilot study
C. Menichetti, A. Castrioto, A. Rossi, P. Giovenali, P. Sarchielli, N. Tambasco, L. Pierguidi, M. Benvenuti, W. Di Iorio, P. Calabresi (Perugia, Italy)
- 894 The effect of autonomic dysfunction on depression in Parkinson's disease
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- 895 Prevalence of helicobacter pylori infection among patients with Parkinson's disease: Impact on clinical manifestations
Y. Tsuboi, T. Yamada (Fukuoka, Japan)
- 896 Using the 16 item identification test from Sniffin Stick's (SS-16) in the diagnosis of Parkinson's disease (PD) in Sri Lanka
L. Silveira-Moriyama, D. Sirisena, P. Gamage, R. Gamage, R. de Silva, A.J. Lees (Colombo, Sri Lanka)
- 897 PD probability curves for smell tests: Interpreting smell test results in the diagnosis of Parkinson's disease (PD)
L. Silveira-Moriyama, M. de Jesus Carvalho, A. Petrie, D. Williams, A. Evans, R. Katzenschlager, A. Kingsbury, E.R. Barbosa, A.J. Lees (London, United Kingdom)

Parkinson's disease: Electrophysiology

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- 898 Optokinematic analysis of the effect of modified STN DBS frequencies on gait disorders in advanced Parkinson's disease
C. Moreau, P. Krystkowiak, S. Bleuse, J.-L. Blatt, A. Duhamel, A. Destee, L. Defebvre (Lille, France)
- 899 Analysis procedure for surface EMG and movement measurements in Parkinson's disease
S.M. Rissanen, M.P. Tarvainen, M. Kankaanpää, A. Meigal, J. Nuutinen, I.M. Tarkka, P.A. Karjalainen, O. Airaksinen (Kuopio, Finland)
- 900 Novel surface EMG/forearm acceleration parameters in the assessment of Parkinson's disease patients: Comparison with healthy controls and relationship with UPDRS motor score
M.J. Kankaanpää, S.M. Rissanen, A. Meigal, M.P. Tarvainen, J. Nuutinen, I. Tarkka, P.A. Karjalainen, O. Airaksinen (Kuopio, Finland)
- 901 Effects of deep brain stimulation on auditory evoked magnetic fields in patients with advanced Parkinson's disease
K.K. Airaksinen, J.P. Mäkelä, S. Taulu, A. Ahonen, J. Pohjola, E. Pekkonen (Helsinki, Finland)
- 902 Thalamic burst patterns during parkinsonian rest tremor
P. Zhuang, M. Hallett, J. Li, Y. Zhang, Y. Li (Beijing, China)
- 903 The disorder of LTD-like plasticity in patients with Parkinson's disease and levodopa induced dyskinesia
F. Morgante, A. Epifanio, C. Terranova, V. Rizzo, L. Morgante, P. Girlanda, A. Quartarone, R. Chen (Messina, Italy)
- 904 Blink reflex in Parkinson disease – an electrophysiological analysis
M. Umaiorubahan (Chennai, Tamilnadu, India)
- 905 Different manifestations of movement-related cortical potentials in patients with Parkinson's disease with and without rest tremor
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- 906 Peripheral nerves injury in the central nervous system degeneration (Parkinson's disease): Pilot study
Z. Chovancova, M. Nevrlý, J. Dufek, P. Otruba, P. Kanovsky (Olomouc, Czech Republic)
- 907 Central motor pathway function in idiopathic early onset Parkinson disease
A. Perretti, A. de Rosa, L. Marcantonio, A. Estraneo, A. Filla, G. De Michele (Naples, Italy)
- 908 Effects of levodopa on saccade performance in Parkinson's disease
A. Yugeta, Y. Terao, H. Fukuda, Y. Ugawa (Tokyo, Japan)
- 909 Muscle strength and power are reduced in people with Parkinson's disease
N.E. Allen, C.G. Canning, C. Sherrington, V.S.C.. Fung (Lidcombe, NSW, Australia)
- 910 Subthalamic local field potential oscillations during ongoing deep brain stimulation in Parkinson's disease
L. Rossi, S. Marceglia, G. Foffani, F. Bracchi, A. Priori (Milan, Italy)
- 911 STN DBS differentially modulates neuronal activity in the pallidal and cerebellar receiving areas of the motor thalamus
W. Xu, J. Zhang, G.S. Russo, T. Hashimoto, J.L. Vitek (Cleveland, OH)
- 912 EMG patterns: A potential neurophysiological marker of Parkinson's disease?
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- 913 Transcranial magnetic stimulation of the primary somatosensory cortex reveals abnormalities in perception of peripheral stimuli in Parkinson's disease
F.J. Palomar, F. Diaz-Corrales, F. Carrillo, M. Fernandez del Olmo, G. Koch, P. Mir (Seville, Spain)
- 914 Group II late excitation evoked in wrist muscles is enhanced in rigid PD patients and modulated by L-Dopa
A. Gerdelat-Mas, V. Marchand-Pauvert, F. Ory-Magne, F. Calvas, D. Mazevet, S. Meunier, C. Brefel-Courbon, M. Vidailhet, M. Simonetta-Moreau (Toulouse, France)
- 915 Hyperhomocysteinemia as a risk factor inducing axonal degeneration in peripheral nerves in young parkinsonian patients: A pilot study
M. Nevrlý, H. Vranova, Z. Chovancova, P. Otruba, J. Dufek, P. Kanovsky (Olomouc, Czech Republic)
- 916 Beta and low gamma frequency synchronization in basal ganglia output during rest and walk in the hemiparkinsonian rat
I. Avila, L.C. Parr-Brownlie, D.A. Bergstrom, E. Castañeda, J.R. Walters (Bethesda, MD)

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- 917 Quantitative electroencephalographic profile of Parkinson's disease with levodopa-induced dyskinesia
C.R. Erickson-Davis, J.S. Anderson, C.L. Wielinski, S.A. Parashos, S. Krohn (New York, NY)
- 918 Correlation between clinical rating of parkinsonian and essential tremor and quantitative assessments
L.G. Aguilar, J.P. Giuffrida, D.D. Heldman, J. Jankovic (Houston, TX)
- 919 The effect of high frequency repetitive transcranial magnetic stimulation on blink reflex conditioning in Parkinson disease
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- 920 Vestibulo-ocular dysfunction in patients with mild-to-moderate stage Parkinson disease: Rotatory chair test
J.-H. Park, S.-A. Park, T.-K. Lee, K.-B. Sung (Bucheon-si, Korea)
- 921 Movement related potentials (MRP) recorded from the human pedunculopontine nucleus region (PPNR) during ankle movements
E.W. Tsang, J. Purzner, C. Hamani, M. Filomena, Y.-Y. Poon, A.M. Lozano, E. Moro, R. Chen (Toronto, ON, Canada)
- 922 Preparation of anticipatory postural adjustments prior to stepping in Parkinson's disease
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- 923 Timing and frequency barriers during repetitive finger movements in patients with Parkinson's disease
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- 924 Reaching and the "gap effect" in Parkinson's disease
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- 931 An open-label pilot study of transdermal selegiline for depression in Parkinson's disease patients
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- 932 Peroxisome proliferator-activated receptor-alpha agonists fenofibrate and bezafibrate have a disease modifier effect in the acute MPTP mouse model of Parkinson's disease
A. Kreisler, A. Duhamel, A. Destée, R. Bordet (Lille, France)
- 933 Increased role of MAO-B in parkinsonian rat striatum
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- 934 Monitoring of ergot dopa agonists in Parkinsons disease: A multicenter UK audit demonstrating inadequate concordance
P.K. Shibu, D. MacMahon, L. Lanchbury (Cambridge, Cambridgeshire, United Kingdom)
- 935 Expression of LRRK2 splice variants in normal human tissues
V. Lakics, E.H. Karran, F.G. Boess (Windlesham, Surrey, United Kingdom)
- 936 The effect of pardoprunox (SLV308) treatment on L-dopa induced dyskinesia in MPTP-treated common marmosets, a chronic study
M.J. Jackson, K. Tayarani-Binazir, S. Rose, A.C. McCreary, P.G. Jenner (Weesp, Netherlands)
- 937 A study on cerebral neurotransmitters in patients with Parkinson's disease
Z.-f. Wang, Y. Han, X.-h. Cheng, H. Song, T. Cheng (Beijing, China)
- 938 Echocardiography and plasma B-type natriuretic peptide in PD treated by dopamine agonists
H. Watanabe, M. Hirayama, A. Noda, M. Ito, N. Atsuta, J. Senda, T. Kaga, G. Sobue (Nagoya, Aichi, Japan)
- 939 Helicobacter pylori infection and motor fluctuations in patients with Parkinson's disease
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- 925 Increased of oxidized/total coenzyme Q-10 ratio in cerebrospinal fluid (CSF) in patients of Parkinson's disease
C. Isobe, T. Murata, C. Ohtsuka, N. Hattori, Y. Terayama (Chitose-shi, Hokkaido, Japan)
- 926 Increased of oxidized/total coenzyme Q-10 ratio in cerebrospinal fluid in patients with Parkinson's disease
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- 927 Rotigotine (Neupro®) in the peri-operative phase, a case series
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- 928 Evaluating the safety, efficacy and tolerability of rasagiline in Parkinson's disease with an unrestricted diet and polypharmacy
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- 929 Soft-tissue-anchored transcutaneous port attached to an intestinal tube for long-term gastro-duodenal infusion of levodopa/carbidopa in Parkinson's disease. A clinical study
P. Forslund, R. Nyman, D. Lundgren, D. Nyholm (Uppsala, Sweden)
- 930 Comparison of apomorphine and levodopa infusions in Parkinson's disease
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- 940 Amelioration of levodopa-induced cognitive impairments by nicotinic therapies in a model of early Parkinson's disease
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- 941 Investigation of plasma homocysteine in patients with Parkinson's disease, treated with levodopa and in those treated with other medications
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- 942 Docosahexaenoic acid reduces 6-OHDA- and MPP+-induced translocation of Nur77 to the cytoplasm: A possible mechanism of neuroprotection
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- 943 Pramipexole and levodopa impact on the onset of dyskinesias and motor fluctuations in Parkinson's disease
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- 944 Factors predicting adverse effects (AE) to subcutaneous apomorphine (APO)
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- 945 Switching from selegiline to rasagiline treatment in patients with Parkinson's disease
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- 946 Expression profile of corpus striatum and cerebellar cortex in rats after 14 days administration of zonisamide; a new anti-parkinsonian agent
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- 947 Long-term continuous s.c. apomorphine infusion monotherapy in a case of young-onset PD
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- 948 Instituting a medication comment in an electronic ordering system to decrease the prescription of inappropriate dopamine antagonists to Parkinson's patients
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- 949 Efficacy and safety of continuous duodenal levodopa infusion in advanced Parkinson disease. 24 months follow up
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- 950 Glutamate plasticity in the subthalamic nucleus following dopamine loss
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- 951 Effects of priribedil and pramipexole in tremor predominant patients with early Parkinson's disease
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- 952 Inappropriate antidiuretic hormone syndrome possibly associated with amantadine therapy in Parkinson's disease
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- 953 Zonisamide increased metabolism of dopamine neurons in MPTP-treated C57BL/6 and common marmosets
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- 954 Neuroprotective effects of salidroside in MPTP-induced dopaminergic injury of nigrostriatal system in mice
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- 955 Indications, efficacy and safety of Duodopa® infusion in Parkinsons disease: A multicentre, retrospective study of 91 French patients
D. Devos, French DUODOPA Study Group* (Lille, France)
- 956 Helicobacter pylori eradication improves treatment response in advanced Parkinson's disease with motor fluctuations
R. Borgohain, V.C. Reddy, S.A. Jabeen, A.K. Meena (Hyderabad, India)
- 957 Content of L-dopa in *Mucuna pruriens* – the different contents in original or modified beans, and on the methods of cookings
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- 958 Severe lymphedema in a patient with Parkinson's disease using pramipexole: A case report
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- 959 Hypersexuality in a patient with Parkinsons disease after use of pramipexole: Case report
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- 960 Ambulatory treatment with continuous subcutaneous apomorphine infusion in advanced Parkinson's disease. Experience with 34 cases
A. Sesar, B. Ares, M.T. Rivas, A. Castro (Santiago de Compostela, Spain)
- 961 An open-label conversion study of pramipexole to ropinirole prolonged release in patients with Parkinson's disease
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- 962 Short term follow up of duodopa therapy in patients with advanced Parkinson's disease
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- 963 Management of complications of long-term duodenal infusion therapy
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- 964 Relevance of the additional noradrenergic neurodegeneration to 6-OHDA-lesioned rats in motor and cognitive disturbances
V. Pérez, A. Rubio, E. Aguilar, M. Barbanoj, C. Marin, J. Kulisevsky (Barcelona, Spain)
- 965 Effects of early versus late initiation of levodopa treatment in hemiparkinsonian rats
C. Marin, E. Aguilar, G. Mengod, R. Cortés, J.A. Obeso (Barcelona, Spain)
- 966 Subthalamic administration of sarizotan attenuates levodopa-induced dyskinesias in 6-OHDA-lesioned rats
C. Marin, E. Aguilar, M. Rodríguez-Oroz, J.A. Obeso (Barcelona, Spain)
- 967 Entacapone potentiates the long-duration response to levodopa but does not normalize levodopa-induced molecular changes in basal ganglia
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- 968 Nicotinic therapies fail to ameliorate levodopa-induced cognitive impairments in a model of advanced Parkinson's disease
J.S. Schneider, C. Bouquiuo, K. Clark, E. Decamp (Philadelphia, PA)
- 969 Multiple administrations of melevodopa (levodopa methyl ester) in advanced Parkinson's disease
C. Pacchetti, R. Zangaglia, M. Sciarretta, S. Cristina, M. Ossola, E. Martignoni, G. Nappi (Pavia, Italy)
- 970 Chronic antidepressant treatment is neuroprotective in a rat model of Parkinson's disease
K.L. Paumier, C.E. Sortwell, B.F. Daley, B.T. Terpstra, N.D. Levine, L.M. Madhavan, T.J. Collier (Cincinnati, OH)
- 971 Clinical effect of tolcapone on continuous subcutaneous apomorphine in Parkinson's disease (PD): An open label study six month follow up
M. Modugno, R. Alfonso, F. Lena, S. Ruggieri, A. Berardelli, A. Brunetti (Campobasso, Italy)
- 972 [³H]-ABP688 autoradiography to metabotropic glutamate receptor type 5 (mGluR5) in the brain of monkeys: Effect of MPTP lesion and levodopa treatment
B. Ouattara, L. Gregoire, M. Morissette, P. Samadi, F. Gasparini, I.T. Vranesic, G. Bilbe, K. McAllister, B. Gomez-Mancilla, T. Di Paolo (Quebec, QC, Canada)
- 973 Neurorestorative effects of PDGF-BB in models of Parkinson's disease through stimulation of cell proliferation
O. Zachrisson, A. Andersson, R. Isacson, S. Jeldes, A. Mercer, E. Nielsen, C. Patrone, H. Ronnholm, L.B. Wikstrom, D.A. Di Monte, A.L. McCormack, B.K. Ormerod, T.D. Palmer, M. Zhao, K.K. Delfani, A.M. Janson Lang, A. Haegerstrand (Stockholm, Sweden)

ABSTRACTS

- 974 Is lactacystin activating mitochondrial-mediated apoptotic pathways?
S. Perez-Alvarez, J. Manzanares, C. Leiva, J. Jordan, M.F. Galindo (Albacete, Spain)
- 975 Correlation of renin-angiotensin-aldosterone system with quality of life in patients with Parkinson's disease and end of dose wearing off
H. Murck, A. Henriksen, K. McCague (East Hanover, NJ)
- 976 Oculo-motor impairment associated with Parkinson's disease
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- 979 Tolerability of dopamine agonists in relation to dose timing
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- 980 Parkinson's disease in pain clinic
A. Sciupokas (Kaunas, Lithuania)
- 981 Transdermal delivery of levodopa prodrugs; is inhibition of DOPA decarboxylation essential for obtaining therapeutic levodopa blood levels?
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- 982 Higher serum uric acid levels in patients with essential tremor compared to Parkinson's disease
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- 983 'Fast tapping' movements in Parkinson's disease: What parameters can be classified as bradykinesia and what's the role of dopaminergic deficit?
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- 985 Changes in diagnosis with follow-up in an incident cohort of patients with parkinsonism
R. Caslake, J.N. Moore, J.C. Gordon, C.E. Harris, C.E. Counsell (Aberdeen, Scotland, United Kingdom)
- 986 Progression of dysprosody in Parkinson's disease over time – a longitudinal study
S.K. Skodda, H. Rinsche, W. Visser, U. Schlegel (Bochum, Germany)
- 987 Vocal rhythm production and acoustical rhythm perception in Parkinson's disease
S.K. Skodda, A. Flasskamp, W. Visser, U. Schlegel (Bochum, Germany)
- 988 Cadence of gait – an objective, dopa-resistant marker of progression in Parkinson's disease
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- 989 Actigraphic evaluation of motor fluctuations in Parkinson's disease patients
S. Perez-Lloret, M. Rossi, D.P. Cardinali, M. Merello (Buenos Aires, Argentina)
- 990 Serum inflammatory biomarkers in Parkinson's disease
I. Rektor, M. Dufek, J. Lokaj, M. Hamanova, D. Goldemund, I. Rektorova, Z. Michalkova, K. Sheardova (Brno, Czech Republic)
- 991 Characteristics of nonmotor fluctuations in Parkinson's disease
T. Iwashita, K. Takahashi, N. Suzuki (Tokyo, Japan)
- 992 Freezing occurring only while eating in two patients with Parkinson's disease
P. Agarwal, A.F. Griffith, M. Borromeo Wesner (Kirkland, WA)
- 993 A case report of vocal cord paralysis in Parkinson's disease
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- 994 The prodromal phase of sporadic Parkinson's disease: Does it exist and if so how long is it?
C.H. Hawkes (Romford, Essex, United Kingdom)
- 995 Backward walking in Parkinson disease
M.E. Hackney, G.M. Earhart (St. Louis, MO)
- 996 Comparative analysis of whole body center of mass position during gait in Parkinson's disease patients compared to control subjects, and potential implications in relation to festination: A kinematic study
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- 997 Non-motor symptoms as presenting complaints in Parkinson's disease: A clinico-pathological study
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- 998 Retrospective analysis of clinical characteristics in Chinese patients with Parkinson's disease in Suzhou region
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- 999 Paradoxical kinesia at war
I. Schlesinger, I. Erikh, D. Yarnitsky (Haifa, Israel)
- 1000 Evidence of increased odds of essential tremor in Parkinson's disease
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- 1001 Cueing for a bimanual coordination task in Parkinson's disease: A comparison of patients with and without freezing of gait
A. Nieuwboer, O. Levin, S. Vercrussse, J. Spildooren, S. Swinnen (Leuven, Belgium)
- 1002 BDNF val66met influences time to onset of levodopa-induced dyskinesia in Parkinson's disease
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- 1003 Parkinson's disease (PD) and gastroesophageal reflux (GERD)
T. Kawakami, K. Fujimoto, I. Nakano (Shimotsuke City, Tochigi, Japan)
- 1004 Sex hormones and prolactin blood levels in males affected with Parkinson's disease
M. Kawulak-Krol, R. Tomasiuk, A. Friedman (Warsaw, Poland)
- 1005 Emotion, cognition, freezing of gait and dual tasking in patients with advanced Parkinson's disease: A volatile mixture
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- 1007 Causes of drooling in Parkinson's disease
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- 1009 Guidelines for occupational therapy in Parkinson's disease
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- 1010 The gait disorders: The factor of variability of step (SVF), as diagnostic criterion at Parkinson's disease (PD) and vascular parkinsonism (VP)
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- 1011 The circumstances associated with falls in Parkinson's disease
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- 1012 Functional mapping of articulatory speech in Parkinson's disease and progressive supranuclear palsy
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- 1013 Motor onset in predicting Parkinson's disease subtypes
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- 1014 The onset of levodopa-induced dyskinesias correlates with the pattern of motor symptoms onset in Parkinson's disease
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- 1015 Hyposomnia in Parkinson's disease is associated with changes in cortical metabolism
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- 1016 Course of Parkinson's disease (PD) motor subtypes – longitudinally followed autopsy confirmed cases
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- 1017 Online data-sharing community for patients with Parkinson's disease: PatientsLikeMe.com
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- 1018 Are there gender differences in motor- and non-motor symptoms in early, untreated PD?
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- 1019 Prevalence of non-motor symptoms amongst Parkinson's disease users in an online health community (PatientsLikeMe.com)
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- 1020 Gait and balance initiative (GABI): Phenomenology of falls in Parkinson's disease
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- 1021 Gait and balance initiative (GABI): Categorization of falls in Parkinson's disease
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- 1022 Gait and balance initiative (GABI): Methodology
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- 1023 Phase plane analysis of perturbed and reintegrated posture in Parkinson's disease patients
J.B. Doan, L.A. Brown, C. Dickin, O. Suchowersky (Lethbridge, AB, Canada)
- 1024 Effect of neupro on premotor and nonmotor symptoms in Parkinson's disease
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- 1025 Foot loop band as manual gait initiator in patients with freezing of gait
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- 1026 Association between laterality (right or left) of onset of motor symptoms and degree of speech impairment in veterans with Parkinson's disease (PD)
A.I. Sarwar, E.C. Lai (Houston, TX)
- 1027 Clinical features of Parkinson's disease patients with camptocormia
M. San Luciano, J. Soto-Valencia, R. Saunders-Pullman, S.B. Bressman (New York, NY)
- 1028 Effect of levodopa (LD) on pacing in Parkinson's disease (PD)
C. Cho, K. Kudo, Y. Osaki, M. Kunin, W. Olanow, B. Cohen, R. Theodore (New York, NY)
- 1029 Gait dysfunction and REM sleep behavior disorder in Parkinson's disease: Is there an association?
D. Benninger, J. Michel, D. Waldvogel, R. Poryazova, V. Dietz, C.L. Bassetti (Zurich, Switzerland)
- 1030 Lateral flexion of the trunk in Parkinson's disease (PD) – a descriptive cross-sectional analysis
A. Hotter, V. Gappmaier, E. Wolf, K. Seppi, W. Poewe (Innsbruck, Austria)

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- 1031 Is it true depression in Parkinson's disease (PD)?
A. Nasar, P. Dyer, E. Howard, C. Short, L. Wheelhouse, L. Wright, K. Turner (Bridlington, East Riding of Yorks, United Kingdom)
- 1032 Relationship between low bone mineral density and Parkinson's disease in a Korean population
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- 1033 Annual cost of Parkinson's disease in the United States
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- 1034 Service users and their carers of a typical U.K. day hospital: How much do you understand about Parkinson's disease?
M.J. Oliver, E. Morgan (Cardiff, United Kingdom)
- 1035 Improvements in daily functioning and walking ability with motivating moves for people with Parkinson's exercise program: A pilot study
J. Hamburg, A. Clair, K.E. Lyons (Lawrence, KS)
- 1036 Factors related to quality of life in patients with Parkinson's disease
B. Tserensodnom, T. Ragchaa, D. Purev (Ulaanbaatar, Mongolia, Mongolia)
- 1037 Getting it right on time? An audit of the administration of Parkinson's disease medications in hospital
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- 1038 Effect of depressive syndrome on the quality of life in patients with Parkinson's disease
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- 1039 The impact of Parkinson's disease (PD) on patients' adult siblings
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- 1040 A retrospective review of admissions of patients with Parkinson's disease: Further evidence to support multi-disciplinary care
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- 1041 Coping strategies for motor fluctuations and dyskinesias developed by patients with PD and their carers
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- 1042 Effects of cardiovascular disorders on life quality of patients with Parkinson disease
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- 1043 Determinants of disability and quality of life in mild to moderate Parkinson's disease
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- 1044 Parkinsonian patients and their spouse during medical consultation
A. Feve, J.P. Brandel (Paris, France)
- 1045 Is depression related to quality of life (QOL) and activities of daily living (ADL) in veterans with Parkinson's disease (PD)?
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- 1047 Caregiver burden and its main predictors for Parkinson's disease: An Indian perspective
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- 1048 Osteoporosis and the patient with Parkinson's disease: Are dietary patterns risk factors?
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- 1050 Constipation: An observational study of the experiences of people with Parkinson's and their carers
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- 1051 Effects of speech and voice disorders on quality of life in patients with Parkinson's disease
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- 1053 Fatigue in early Parkinson's disease. Minor inconvenience or major distress?
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- 1055 Patient care satisfaction in Parkinson disease
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- 1057 Detection of non-motor symptoms in Parkinson's disease and impact on health-related quality of life
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- 1060 Group patient visits for Parkinson disease
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- 1062 Direct costs and working capacity in Parkinson's disease: A patient survey
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- 1063 Parkinson's Wellness Camp: Effect on QOL
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- 1064 Detecting depression in Parkinson's disease: How useful is the unified Parkinson's disease rating scale (UPDRS)?
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- 1065 Foot tapping as an outcome measure for Parkinson's disease clinical trials: Results from two clinical studies
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- 1066 Addenbrooke's cognitive examination (ACE) validation in Parkinson's disease
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- 1067 Quantitative assessment of motor disability in Parkinson's disease: Upper limb motor performance index
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- 1068 Nonmotor symptoms (NMS) in Parkinson's disease in New Zealand – a pilot study in Hawke's Bay
C. Sixsmith, M.T. Grist (Southampton, United Kingdom)

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- 1069 The frequency of individual nonmotor symptoms (NMS) and symptom subgroups in Parkinson's disease in Hawke's Bay, New Zealand
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- 1070 Quantifying rigidity in MPTP-treated primates
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- 1071 Detecting early changes of functional status in Parkinson's disease
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- 1072 Analysis of tapping test results in a test battery for advanced PD patients
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- 1073 Client-centered evaluation of duodopa treatment at 6 month post therapy
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- 1074 Differences in progression rates and levodopa responsiveness of UPDRS subscales in the DATATOP cohort
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- 1075 The validity of calculating stride length from walking speed and stride time: An uncomplicated clinical complement to assessing gait in Parkinson's disease
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- 1076 Prevalence of non motor symptoms and relationship with Parkinson's disease severity
S. Raha, L. Ebenezer, S. Al-Hasani (Bridgend, United Kingdom)
- 1077 A study of depression, personality and quality of life in Parkinson's disease
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- 1078 Non-motor symptoms in parkin gene-related parkinsonism
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- 1079 Does Parkinson disease alter that natural ipsilateral influence of the dominant left hemisphere?
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- 1080 A new test to evaluate olfaction impairment in Parkinson's disease
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- 1081 Results of a new test detecting smell impairment in Parkinson's disease
S. Bannier, M. Jollerovski, J.-L. Berdague, N. Kondjoyan, F. Durif (Clermont-Ferrand, France)
- 1082 A community-based study of Parkinson's non-motor symptoms
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- 1083 Step activity in physically active persons with Parkinson's disease
M.P. Ford, L. Malone, H.C. Walker, I. Nyikos (Birmingham, AL)
- 1084 Objective measurement of levodopa-induced dyskinesia with a force plate
K.A. Chung, B. Lobb, F. Horak, J.G. Nutt (Portland, OR)
- 1085 Objective at-home testing measures as predictors of UPDRS change in early Parkinson's disease (PD)
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- 1086 Static and dynamic balance at different stages of Parkinson's disease
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- 1087 Kinematic akinesia feature extraction using Kinesia™
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- 1088 Quantitative comparisons of bradykinesia in the more-affected versus less-affected limb in patients with Parkinson's disease as a function of disease severity
S. Louie, A. Frenklach, M. Koop, H. Bronte-Stewart (Stanford, CA)
- 1089 Application of general systems performance theory to the interpretation and scoring of UPDRS I & II
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- 1090 The Parkinson's disease sleep scale (PDSS) modified – preliminary results from a validation study
C. Trenkwalder, B. Hoegl, R. Kohlen, F. Sixel-Doering, B. Frauscher, A. Schoerling, P. Martinez-Martin, W. Poewe, R. Chaudhuri (Kassel, Germany)
- 1091 Performance of the scale for assessment of positive symptoms in Parkinson disease psychosis
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- 1092 Unified Parkinson's disease rating scale (UPDRS) part I as a screening and diagnostic instrument for apathy in patients with Parkinson's disease
D.A. Gallagher, A. Lees, A. Schrag (London, United Kingdom)
- 1093 Interrater reliability of dynamic balance in persons with Parkinson's disease
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Rating scales

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- 1094 The essential tremor rating assessment scale (TETRAS)
R. Elble, C. Comella, S. Fahn, M. Hallett, J. Jankovic, J. Juncos, E. Louis, K. Lyons, W. Ondo, R. Pahwa, K. Sethi, M. Stern, C. Tanner, D. Tarsy, R. Tintner, R. Watts, Tremor Research Group (Springfield, IL)
- 1095 Validation of an instrument to measure older adults' expectations about developing parkinsonism
N. Dahodwala, C.D. Zubritsky, J.H. Karlawish, T. Houry, M.B. Stern, D.S. Mandell (Philadelphia, PA)
- 1096 The Timed Up and Go test: More than meets the eye
T. Herman, N. Inbar-Borovsky, M. Brozgol, L. Maryasin, N. Giladi, J.M. Hausdorff (Tel Aviv, Israel)
- 1097 Gait scale for Parkinson's disease (GS-PD-V2)
M. Serrano-Dueñas, B. Calero, S. Serrano (Quito, Pichincha, Ecuador)
- 1098 Spanish validation of the Lille apathy rating scale in Parkinson disease
R. Garcia-Ramos, C. Villanueva, J. Del Val, M.J. Catalan, J. Matias-Guiu, A. Reig-Ferrer (Madrid, Spain)
- 1099 Image analysis of standardized video to quantify dyskinesia in Parkinson's patients
A.S. Rao, R. Li, T.L. Davis, C. Voight, R.E. Bodenheimer, B.M. Dawant (Nashville, TN)
- 1100 Validation of the global assessment scale for Wilson's disease (GAS for WD)
A. Aggarwal, N. Aggarwal, A. Nagral, G. Jankharia, M. Bhatt (Mumbai, India)

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- 1101 Tracking Wilson's disease: Some clinical observations and evaluation of GAS for WD
A. Aggarwal, N. Aggarwal, M. Bhatt (Mumbai, Maharashtra, India)
- 1102 Use of the Nintendo Wii™ remote to quantify finger tapping in Parkinson's disease
A.J. Chambers, P.A. Harris, N.D. Snyder, T.L. Davis (Nashville, TN)
- 1103 The burden of depression and anxiety in Parkinson's disease. Metric properties of the Beck depression and Beck anxiety inventories, with K2 factorial design
M. Serrano-Dueñas, S. Sevilla, P. Lastra (Quito, Pichincha, Ecuador)
- 1104 Use of the Nintendo Wii™ remote to measure tremor in essential tremor
R.D. Connors, J.Y. Fang, P. Hedera, A.S. Rao, N.D. Snyder, T.L. Davis (Nashville, TN)
- 1105 Evaluation of a Parkinson's disease screening questionnaire for use in a community-based setting
C.B. Hunter, L.G. Aguilar, M.M. Nashatizadeh, L.F. Lay, J. Jankovic (Houston, TX)

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- 1106 Three cases of pathologic gambling in patients prescribed dopamine agonists for restless legs syndrome
B.K. Changizi, E.S. Molho (Albany, NY)
- 1107 Restless legs syndrome in Parkinson's disease: Prevalence, clinical characteristics and biochemical correlations
R.C.P. Prado, H.A. Melo, T.M. Guerreiro, D.R.C. Nishikawa (Aracaju, Sergipe, Brazil)
- 1108 Rapid reduction of nocturnal limb pain in restless legs syndrome (RLS) after pramipexole (PPX) treatment
M. Hornyak, M. Partinen, J. Koester, S. Albrecht (Freiburg, Germany)
- 1109 Patient and physician assessments of global improvement in restless legs syndrome (RLS) after pramipexole (PPX) treatment
J. Ulfberg, A. Nicolas, J. Koester, S. Albrecht (Hedemora, Sweden)
- 1110 Impaired sensori-motor integration in restless legs syndrome is restored by dopamine agonists
V. Rizzo, I. Aricò, G. Liotta, L. Ricciardi, F. Morgante, P. Girlanda, R. Silvestri, A. Quartarone (Messina, Italy)
- 1111 Cardiovascular risk factors in restless legs syndrome
I. Schlesinger, I. Erikh, O. Avizohar, E. Sprecher, D. Yarnitsky (Haifa, Israel)
- 1112 A 52-week, open-label study to assess the long-term tolerability of ropinirole CR extended release tablets in subjects with restless legs syndrome (RLS)
C. Hill-Zabala, R. Bogan, D. Lee, M. Lomax (Research Triangle Park, NC)
- 1113 XP13512/GSK1838262 1200 mg provides symptomatic relief in restless legs syndrome patients: A randomized, double-blind, placebo-controlled study
A.L. Ellenbogen, C.A. Kushida, P.M. Becker, D.M. Canafax, P. Tran (, MI)
- 1114 Restless legs syndrome associated to narcolepsy and somnambulism
H. Alonso-Navarro, F.J. Jiménez-Jiménez (Arganda del Rey, Madrie, Spain)
- 1115 Professor Karl-Axel Ekborn and restless legs syndrome
H.A.G. Teive, R.P. Munhoz, E.R. Barbosa (Curitiba, Parana, Brazil)
- 1116 Head-to-head comparison of dopamine agonists in 'restless legs syndrome'
S. Sevim, N. Ozveren, H. Kaleagasi (Mersin, Turkey)
- 1117 A case of primary restless legs syndrome exacerbated by hyperparathyroidism
A.F. Griffith, P. Agarwal (Kirkland, WA)
- 1118 No evidence of augmentation with rotigotine treatment in a 6-month, multicenter, double blind, placebo-controlled RLS trial
W.A. Hening, R. Allen, J.W. Winkelman, E. Schollmayer (Piscataway, NJ)
- 1119 The effect of food on the clinical pharmacokinetics of XP13512/GSK1838262
R. Lal, J. Sukbuntherng, W. Luo, J.F. Huff, K.C. Cundy (Santa Clara, CA)
- 1120 A 12-week, randomized, double-blind study to assess the tolerability and clinical benefits of ropinirole CR extended release tablets compared with ropinirole immediate release (IR) tablets in subjects with restless legs syndrome (RLS)
J. Tolson, R. Hodge, R. Ehsanullah, J. Gitt (Research Triangle Park, NC)
- 1121 Rotigotine 24h transdermal patch is effective in the treatment of idiopathic RLS: Results of a 6-month, multicenter, double-blind, placebo-controlled US trial
W.A. Hening, R. Allen, P.M. Becker, R.K. Bogan, J.M. Fry, J. Keffel, D.B. Kudrow, K.W. Lesh, W.G. Ondo, E. Schollmayer, P. Vrooman, A. Walters, J.W. Winkelman (Piscataway, NJ)
- 1122 Rotigotine transdermal system in the treatment of idiopathic RLS: Combined results from two 6-month, multicenter, double-blind, placebo-controlled trials in Europe and the US
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- 1123 Negative impact of restless legs syndrome on work productivity
M. Calloway, M. Bharmal, R. Allen, E. Gemmen (Durham, NC)
- 1124 Long-term safety and efficacy of rotigotine in idiopathic RLS: 3-year results from a multinational open-label trial
D. Garcia-Borreguero, W. Poewe, B. Hoegl, R. Kohonen, K. Stiasny-Kolster, J. Keffel, E. Schollmayer, B. Boroojerdi, C. Trenkwalder, W. Oertel (Madrid, Spain)
- 1125 Assessing the negative impact of sleep loss among individuals with restless legs syndrome (RLS) in the USA
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- 1126 Levodopa may benefit children with ADHD who have comorbid RLS
W.A. Hening, F. Siddiqui, V. Couvadelli, D. Picchiotti, M.L. Wagner, B. Fisher, S. England, B. Cohen, A.S. Walters, ADHD/RLS Study Group (New York, NY)
- 1127 Clinical pharmacokinetics of XP13512/GSK1838262, a novel transported prodrug of gabapentin
K.C. Cundy, S. Sastry, W. Luo, J. Zou, T.L. Moors, D.M. Canafax (Santa Clara, CA)
- 1128 Prevalence and potential under-diagnosis in patients with restless legs syndrome (RLS) in the USA
M. Calloway, R. Allen, E. Gemmen, M. Bharmal (Durham, NC)
- 1129 Restless legs syndrome and risk of Parkinson disease and erectile dysfunction
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- 1130 Prevalence of restless legs syndrome among patient with multiple sclerosis
A.P. Doneva, V.C. Daskalovska (Bitola, Macedonia, The Former Yugoslav Republic of)
- 1131 Assessment of treatment patterns and healthcare expenditures of restless leg syndrome (RLS) patients newly started on a dopamine agonist vs. other agent
B. Johnson, K. Foley, A. Patel, C. Atwood, S. Sander, H. Shah (Ridgefield, CT)
- 1132 The efficiency and safety of pramipexol in the treatment of primary idiopathic restless leg syndrome
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- 1133 Prevalence and factors associated with dopaminergic-medication induced impulse control behaviors in restless legs syndrome
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- 1134 Assessment of rotigotine in idiopathic RLS: Results of a 7-week sleep lab trial
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- 1144 Daytime somnolence in Parkinson's disease
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- 1145 Items scores in a revised Parkinson's disease sleep scale (PDSS) and sleep disorder diagnosis
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- 1146 Sleep problems in Parkinson's disease with and without REM sleep behavior disorder
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- 1147 Sleep-wake rhythm abnormalities in Parkinson's disease patients
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- 1148 The effect of repetitive transcranial magnetic stimulation on sleep in Parkinson disease – preliminary study
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- 1149 Is cognitive impairment in Parkinson's disease associated with REM-sleep behavior disorder?
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- 1150 Excessive daytime sleepiness in Parkinson's disease – parallels to narcolepsy
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- 1135 Validation of the sleep related items of the non-motor symptoms questionnaire for Parkinson's disease (NMSQuest)
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- 1136 Validation of the Parkinson's disease sleep scale by day-to-day sleep evaluation using a sleep log
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- 1137 Are there any clinical differences in Parkinson's disease with and without REM sleep behavior disorder?
A. Yoritaka, H. Ohizumi, S. Tanaka, N. Hattori (Urayasu-shi, Chiba, Japan)
- 1138 Effect of STN micro-lesioning on nocturnal disabilities of Parkinson's disease
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- 1139 Is REM sleep behavior disorder associated with visuo-perceptive dysfunction in Parkinson's disease?
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- 1140 REM sleep suppression in the presymptomatic MPTP model of Parkinson's disease
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- 1141 Case-control study of restless legs syndrome and quality of sleep in Parkinson's disease
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- 1142 Comparison of polysomnographic abnormalities in different parkinsonian syndromes. A retrospective study in 87 patients
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- 1143 Risk of neurodegenerative disease in patients with idiopathic REM sleep behavior disorder
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- 1151 Use of botulinum toxin type B in adult patients with spasticity: A multi-center retrospective chart review
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- 1152 Magnetic brainstem stimulation using double stimuli in healthy volunteers and patients with pyramidal tract lesions
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- 1153 Novel mutations of the SPG11 gene in hereditary spastic paraplegia with thin corpus callosum
L. Shen, S. Liao, J. Du, G. Zhao, Z. Xiao, Y. Yuan, B. Tang (Changsha, Hunan, China)
- 1154 Dose response in muscles treated with botulinum neurotoxin type A (BOTOX®) for upper limb chronic post-stroke spasticity: A pooled-data analysis
S.A. Yablon, A.M. VanDenBurgh, F.C. Beddingfield III, J. Wagg, T. Khariton, S. Hua, S. Abu-Shakra (Jackson, MS)
- 1155 Baseline clinical features of chronic post-stroke patients enrolled in botulinum neurotoxin type A (BOTOX®) upper limb spasticity trials
A. Brashear, A.M. VanDenBurgh, S. Abu-Shakra, S.A. Yablon, J. Wagg, T. Khariton, S. Hua, F.C. Beddingfield (Winston-Salem, NC)
- 1156 Efficacy and safety of NT 201 (Xeomin®) in the upper limb post-stroke spasticity in a double-blind, placebo-controlled, randomized, multi-center trial
P. Kanovsky, I. Sassin, G. Comes, S. Grafe, NT 201 Study Group (Olomouc, Czech Republic)

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- 1157 Ashworth scale score changes from baseline in patients post-stroke upper limb spasticity treated with botulinum neurotoxin type A (BOTOX®): Screening for covariate effects
A. Brashear, A.M. VanDenBurgh, J. Wagg, T. Khariton, S. Hua, S. Abu-Shakra, F.C. Beddingfield (Winston-Salem, NC)
- 1158 A clinicogenetic review of 138 Dutch SPAST mutation carriers
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- 1159 Botulinum toxin A and serial casting reduce disability following acquired brain injury
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- 1160 Intensive voice treatment (LSVT® LOUD) for children with spastic cerebral palsy
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- 1161 Clinical spectrum of sporadic and familial spastic paraplegia (HSP)
R. Schüle, S. Otto, S. Klimpe, K. Karle, S. Klebe, L. Schöls (Tubingen, Germany)
- 1162 Prevalence of spasticity in adults with intellectual disability living in the community: A feasibility survey
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- 1163 Effective thalamic deep brain stimulation for neuropathic tremor in a patient with severe demyelinating neuropathy: A case report
T. Wächter, S. Breit, L. Schöls, T. Gasser, T. Nägele, D. Freudenstein, R. Krüger (Tuebingen, Germany)
- 1164 Lateralized effects of unilateral thalamotomy and thalamic stimulation in patients with essential tremor
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- 1165 Deep brain stimulation (DBS) of the subthalamic nucleus (STN) in patients with segmental and focal cervical dystonia
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- 1166 Pedunculopontine nucleus deep brain stimulation in primary progressive freezing of gait: A case study
J.L. Ostrem, C.W. Christine, M.M. Volz, G.A. Glass, P.S. Larson, L.E. Schrock, P.A. Starr (San Francisco, CA)
- 1167 A computational approach to the investigation of motorcortical stimulation of the treatment of pain
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- 1168 Pallidal stimulation in primary dystonia: Results of a multicentric Spanish study
C.L. Moreno-Lopez, F. Valldeoriola, GESPALDIS (Barcelona, Catalonia, Spain)
- 1169 Deep brain stimulation in chorea-acanthocytosis
P.J. Garcia Ruiz, J. Ayerbe, F. Alonso Frech (Madrid, Spain)
- 1170 Clinical benchmarking of a paediatric deep brain stimulation (DBS) service improves delivery efficiency and quality of assessments
H. Gimeno, R. Mahoney, K. Tustin, S. Jupp, M. Kaminska, R. Selway, J.-P. Lin (London, United Kingdom)
- 1171 Video capture of rapid recovery from status dystonicus in PANK2 disease when Kinetra® DBS stimulator is switched on again 2.5 years post implant
J.-P. Lin, S. Jupp, M. Kaminska, D. Lumsden, H. Gimeno, R. Mahoney, K. Tustin, R. Selway (London, United Kingdom)
- 1172 A study on outcome of surgical treatment for focal dystonia
Y. Zhang, J. Li, Y. Li (Beijing, China)
- 1173 Comparison of micro-electrode recordings from globus pallidus internus (GPI) in children with severe dystonia from NBIAT and other syndromes: Recordings under general anaesthesia
A. Valentin, R. Selway, J.-P. Lin, M. Samuel, A. Keyoumars, G. Alarcon (London, United Kingdom)
- 1174 Entopeduncular deep brain stimulation modifies firing activity in entopeduncular neurons in the dystonic hamster
A. Leblois, R. Reese, D. Labarre, T. Boraud, M. Hamann, A. Richter, W. Meissner (Pessac, France)
- 1175 Deep brain stimulation of the entopeduncular nucleus increases striatal c-fos expression in the dystonic hamster
R. Reese, A. Nadjar, G. Charron, C. Aubert, M. Hamann, A. Richter, E. Bezaud, W. Meissner (Pessac, France)
- 1176 Chronic deep brain stimulation for segmental dystonia: Reduction of stimulation intensity after implantable pulse generator replacement for battery depletion
C. Blahak, H.-H. Capelle, H. Baezner, T. Kinfe, J.K. Krauss (Mannheim, Germany)
- 1177 Significant improvement of head and neck mobility by chronic deep brain stimulation in segmental dystonia – a study using computerized motion analysis
C. Blahak, H. Baezner, H.-H. Capelle, J.C. Woehrle, M.G. Hennerici, J.K. Krauss (Mannheim, Germany)
- 1178 Deep brain stimulation in the Zona incerta in the treatment of essential tremor
U. Sandvik, S. Tisch, P. Blomstedt (Umea, Sweden)
- 1179 Long term follow-up in GPI deep brain stimulation in 11 consecutive subject with primary segmental dystonia
M. Sensi, R. Eleopra, M. Cavallo, S. Sarubbo, V. Tugnoli, J.G. Capone, E. Gastaldo, M.R. Tola, R. Quatrone (Ferrara, Italy)
- 1180 Malfunction of implantable pulse generator
D. Apetauerova, J. Zani, J. Arle, J. Shils (Burlington, MA)
- 1181 DBS frequency screening for programming optimization in a patient with chorea-acanthocytosis
F. Gupta, N. Chan, R. Alterman, R. Walker, M. Tagliati (New York, NY)
- 1182 Bilateral stimulation of the caudal zona incerta nucleus for control of essential tremor
L.K. Mooney, S. Khan, P. Plaha, K.R. O'Sullivan, S.S. Gill (Bristol, England, United Kingdom)
- 1183 Transient anti-dystonic benefit after pallidal deep brain stimulation in a six year old girl with pantothenate kinase-associated neurodegeneration
A.P. Duker, D.L. Gilbert, G.T. Mandybur, A.J. Espay, M. Gartner, F.J. Revilla (Cincinnati, OH)
- 1184 Defibrillation in a patient with deep brain stimulation (DBS)
J.T. Al-Hinti (Little Rock, AR)
- 1185 Bilateral deep brain stimulation for treatment of medically refractory paroxysmal nonkinesigenic dyskinesia: A case report
C.B. Kaufman, J. Schwalb, J. Mink (Rochester, NY)

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- 1186 Brain regions for smile and panic phenomena induced during intraoperative test stimulation in obsessive compulsive disorder (OCD)
I.U. Haq, K.D. Foote, S. Wu, W.G. Goodman, A. Sudhyadhom, D. Bowers, H.H. Fernandez, C.C. Jacobson, M.S. Okun (Gainesville, FL)
- 1187 Response to deep brain stimulation (DBS) in primary & secondary childhood dystonias improves with the number of activated contacts within the globus pallidus internus (GPI)
D. Lumsden, R. Selway, T. Kerr, J.-P. Lin (London, United Kingdom)
- 1188 In-vitro thermocoagulation using deep brain stimulation electrodes
F. Alesch, T. Hauska (Vienna, Austria)
- 1189 Bilateral GPi DBS for craniofacial dystonia
F.T. Phibbs, P.E. Konrad, J.S. Neimat, T.L. Davis (Nashville, TN)
- 1190 Vim DBS improves working memory performance in patients with essential tremor
S.E. Zauber, D. De Alwis, M.C. Campbell, P.M. Weaver, S. Tabbal, J.S. Perlmutter, T. Hershey (Chicago, IL)
- 1200 [123I]-FP-CIT SPECT and olfaction test in patients with combined postural and rest tremor
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- 1201 Bifocal Vim DBS for kinetic tremor after microvascular decompression for trigeminal neuralgia
T.M. Kinfe, H.H. Capelle, C. Schrader, C. Blahak, H. Baezner, J.K. Krauss (Hannover, Niedersachsen, Germany)
- 1202 Effects of transcranial direct current stimulation in patients with essential tremor
H. Hellriegel, M. Poetter, M. Nitsche, W. Paulus, G. Deuschl, J. Raethjen (Kiel, Germany)
- 1203 Holmes tremor: A new videotaped case with complete ipsilateral striatal dopaminergic denervation and striking response to sub-cutaneous apomorphine
F. Viallet, D. Gayraud, E. Guedj (Aix en provence, France)
- 1204 Validation of a sensitive, standardized tool for evaluating novel therapies in essential tremor (ET)
R. Iannone, R. Zoethout, J. van Gerven, R. Schoemaker, B. Bloem, R. van Lummel, J. Palcza, G. Murphy, J. Chodakewitz, A. Buntinx, K. Gottesdiener, S. Marsilio, L. Rosen, K. VanDyck, N. Sato, S. Tokita, E.D. Louis, J. Renger (North Wales, PA)

Tremor

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- 1191 Purkinje cell (PC) count and glutamic acid decarboxylase (GAD) in essential tremor (ET) cerebellum
A.H. Rajput, C. Luo, A.H. Rajput, M.L. Rajput, C.A. Robinson (Saskatoon, SK, Canada)
- 1192 Older onset essential tremor is associated with more rapid progression and more degenerative pathology
E.D. Louis, P.L. Faust, J.-P.G. Vonsattel, L.S. Honig, C. Henchcliffe, R. Pahwa, K.E. Lyons (New York, NY)
- 1193 Familial cortical myoclonic tremor with epilepsy – importance of eye movement abnormality
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- 1194 Various movement disorders in a patient with sensory neuropathy caused by Sjögren syndrome
W.-Y. Lin, H.-S. Chang, C.-C. Huang, Y.-R. Wu (Taipei, Taiwan)
- 1195 Tremor in XYY syndrome
D.A. Hall, M. Borodyankysya, N. Tartaglia (Aurora, CO)
- 1196 Essential tremor appearing ipsilateral to cerebellar hemispherectomy: Support for the thalamus as the central oscillator
G. Debrata, L. Chahine (Cleveland, OH)
- 1197 Essential palatal tremor: Remarkable benefit from sodium valproate
N. Subutay-Oztekin, M.F. Oztekin, R. Sari-Polat (Ankara, Turkey)
- 1198 Correlation between tremor amplitude and postural instability in orthostatic tremor
H. Hellriegel, J. Volkmann, G. Deuschl, J. Raethjen (Kiel, Germany)
- 1199 Tremor in X-linked recessive spinobulbar muscular atrophy (Kennedy's disease)
H.A.G. Teive, S. Raskin, R.P. Munhoz, L.C. Werneck (Curitiba, Parana, Brazil)
- 1205 Saccades abnormalities in essentials tremor
M. Wojcik, M. Ruszinska, M. Dec, J. Dylak, J. Ober, A. Szczudlik (Krakow, Poland)
- 1206 Lower extremity tremor in essential tremor: Prevalence, clinical correlates, and comparison with age-matched controls
K.L. Poston, E.D. Louis (New York, NY)
- 1207 The postural tremor of Parkinson's disease: A video analysis of a case series
V.K. Gontu, J. Birchall, N. Bajaj (Derby, United Kingdom)
- 1208 Psychogenic movement disorders in children: A report of 15 cases and a review of the literature
P. Schwingenschuh, C. Pont-Sunyer, R. Surtees, K.P. Bhatia (London, United Kingdom)
- 1209 A controlled-study of the quantitative analysis of arm swing in essential tremor
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- 1210 Applying the criteria for adult onset dystonic tremor to SWEDDs patients
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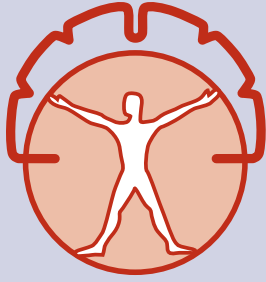
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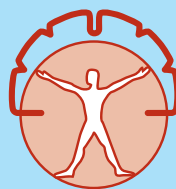
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