for innovative solutions in prevention, cure and care for the health and well-being of people worldwide. We combine our talents to understand and meet the needs of patients and their families to enhance the quality of life.

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Eisai Co., Ltd.

Eisai Announces Change in Regulatory Submission Strategy of E2007 for Parkinson's Disease

Eisai Co., Ltd. (Headquarters: Tokyo, President & CEO: Haruo Naito) announced today that the Company has decided to change the regulatory submission timing of E2007 (generic name: perampanel), which is under clinical investigation worldwide aiming to be the first oral AMPA receptor antagonist available for conditions including Parkinson's disease, neuropathic pain, epilepsy, migraine prophylaxis and multiple sclerosis. Eisai originally planned to file E2007 for approval in the U.S. and Europe for Parkinson's disease as the first indication within fiscal year 2007 but will do so in the fourth quarter of fiscal year 2008.

E2007, a new chemical entity developed by Eisai, has the novel mechanism of action of selectively antagonizing AMPA-type glutamate receptors. It is believed to show promise in the treatment of various neurologic diseases because AMPA receptors are widely distributed in nerves.

Eisai is conducting three Phase III studies (Studies 301, 302 and 309), primarily in the U.S. and Europe, for Parkinson's disease. The 30-week Study 301, which compared E2007 to placebo as an add-on therapy to levodopa for patients who have idiopathic Parkinson's disease, has been completed and analysis of its results is ongoing.

Concurrently, other clinical trials to investigate the efficacy and safety of E2007 for various indications, including neuropathic pain and epilepsy, are ongoing. Eisai plans to submit the E2007 application for neuropathic pain in fiscal year 2010, following the submission for Parkinson's disease.

Eisai remains committed to treating patients with cerebral and neurologic diseases and to increasing their quality of life through development of various new pharmaceuticals in addition to E2007.

Contacts:

Corporate Communications Department

Eisai Co., Ltd.
TEL: +81-3-3817-5036

Reference Data

1. AMPA Receptors and E2007

Glutamic acid is a common neurotransmitter used in central neuronal cells. It is known that excessive stimulation by glutamic acid causes excessive inflow of calcium ions into cells, resulting in central nerve cell death. AMPA receptors, one type of three ion-channel-type glutamate receptors, are believed to be engaged in almost all neuronal transmissions at excitatory synapses.

E2007 selectively antagonizes AMPA receptors and inhibits neuronal cell death. This mechanism was studied mainly by researchers at Eisai's London Research Laboratories and published in the UK's peer reviewed journal "Nature Medicine". (L. Steinman, Nature Medicine 6(1) 15-16, 2000)

2. Preclinical Studies of E2007 for Parkinson's Disease

1) Effects of E2007 on preclinical Parkinson's model

In a study designed to compare effects of E2007 and levodopa in MPTP (1-Methyl 4-phenyl 1,2,3,6-tetrahydropyridine), E2007 did not affect levodopa's maximum effect on Parkinson's disease symptoms, however, it significantly both prolonged levodopa's effective time and improved the severity of dyskinesia.

Presented at International Congress of Parkinson's Disease and Movement Disorders on November 2, 2006

2) Effects of E2007 on rotational behavior of levodopa

E2007 prolonged levodopa's duration of action in a preclinical study for 6OHDA-hemi-parkinsonian model.

Presented at International Congress of Parkinson's Disease and Movement Disorders on November 2, 2006.

3. Clinical Studies of E2007 for Parkinson's Disease

1) Study 204

Placebo control Phase II POC (proof of concept) Study

Target:	Parkinon's disease patients with motor fluctuations and dyskinesia on levodopa (maintenance dose).
Purpose:	Proof of concept
Treatment groups:	E2007 0.5mg, 1mg, 2mg or placebo
Duration:	12 weeks
Primary endpoint:	"Off" time change

Results:

- E2007 showed significant difference in reduction in "Off" time in a statistically significant dose-related manner. E2007 did not show significant difference between each dose and the placebo group in reduction of "Off" time. However, the E2007 2mg arm showed significant difference in efficacy.
- E2007 was well tolerated and no significant safety issues were observed.
- Worsening of dyskinesia was not observed.

2) Study 301

Placebo control Phase III studies

Target:	Add-on therapy in idiopathic PD patients with levodopa therapy with motor fluctuations of a “wearing off” type
Purpose:	To compare the efficacy of 2mg and 4mg of E2007 and placebo on motor function
Treatment groups:	E2007 2mg, 4 mg or Placebo
Duration:	30 weeks
Primary endpoint:	“Off” time change

3) Study 302

Placebo control Phase III studies

Target:	Add-on therapy in idiopathic PD patients with levodopa therapy with motor fluctuations of a “wearing off” type
Purpose:	To compare the efficacy of 2mg and 4mg of E2007 and placebo on motor function
Treatment groups:	E2007 2mg, 4 mg or Placebo
Duration:	20 weeks
Primary endpoint:	“Off” time change

4) Study 309

Placebo and entacapone control Phase III study

Target:	Add-on therapy in idiopathic PD patients with levodopa therapy with motor fluctuations of a “wearing off” type
Purpose:	To compare the efficacy of 4mg of E2007, placebo and entacapone on motor function
Treatment groups:	E2007 4mg, placebo or 200mg entacapone
Duration:	18 weeks
Primary endpoint:	“Off” time change