

## **Phytopharm enters an agreement with CHDI to evaluate the potential efficacy of Cogane™ in the treatment of Huntington's disease (HD)**

### **Huntington's disease represents a new indication for Cogane™**

GODMANCHESTER, Cambridgeshire, U.K. (23 July 2009) - Phytopharm plc (LSE: PYM) ("Phytopharm" or the "Company") today announces that it has entered into an agreement with CHDI Foundation, Inc. in the USA to evaluate the efficacy of its orally active, neurotrophic factor inducer PYM50028 (Cogane™), in a preclinical model of Huntington's disease (HD).

HD is a hereditary and degenerative condition of the central nervous system (CNS) that carries a 50% risk of being inherited by the children of an affected parent. HD affects a wide range of brain activities and symptoms, cognitive and physical impairments gradually become apparent in middle age. Changes in personality or mood may be the earliest signs of the disease, followed by problems with memory and involuntary movements. There is currently no cure for HD.

CHDI will fund the testing of Cogane™, in its network of industrial contract research organisations, employing its standardised criteria for the rigorous evaluation of novel therapeutic approaches for HD treatment. The testing will begin immediately and is expected to be completed in Q1 2010.

In preclinical models, Cogane™ increases the body's own production of a group of proteins called neurotrophic factors. One of these factors, "BDNF", is known to be decreased in the brains of HD patients. Increasing the brain level of BDNF has been postulated as a potential treatment for HD. However, since BDNF is a protein it cannot be given orally (in pill or liquid form) because it is degraded in the stomach and intestine, and also does not readily cross the blood-brain barrier. Cogane™, which can be taken orally, readily distributes into the brain and stimulates the release of endogenous BDNF in the brain. It therefore has the potential to overcome the technical difficulties associated with exogenous BDNF administration.

Mr Sandy Morrison, CEO of Phytopharm, said: "We are delighted to have entered an agreement with CHDI to explore the potential of Cogane™ in a preclinical model of Huntington's disease, a new indication for our lead compound. Huntington's disease is an area of extremely high unmet medical need and there is currently no treatment available to slow the progression or delay the onset of this devastating disease. We look forward to reporting the findings of this study in the first half of 2010. This partnership with CHDI is indicative of Phytopharm's ongoing strategy of partnering with leading charities in order to facilitate the progression of our pharmaceutical pipeline and demonstrates the potential beneficial effect of Cogane™ in other neurodegenerative diseases."

In April 2009, following approval from the Medical and Healthcare products Regulatory Agency, Phytopharm commenced a safety, tolerability and pharmacokinetic (PK) study of Cogane™ in both healthy volunteers and patients with Parkinson's disease (PD).

**-Ends-**

## Notes to Editors

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### About Phytopharm plc

Phytopharm is a pharmaceutical development and functional food company. Our products are developed from medicinal plants, thereby reducing the development risk, cost and time to market. As a virtual company, Phytopharm's model is centred on a lean cash burn with all laboratory, manufacturing and clinical work out-sourced to specialists, while core competencies such as strategy and management are maintained in-house. Close collaboration with charitable organisations provides funding for research activity, enhances our interaction with Key Opinion Leaders and accelerates our development programmes, increasing their value.

### About Huntington's disease

Huntington's disease is a hereditary and degenerative condition of the central nervous system (CNS) caused by a mutation in the *huntingtin* gene. Each child of a parent with this mutation has a 50% chance of inheriting it. As a result of carrying the mutation, an individual's brain cells undergo programmed death. This leads to cognitive and physical impairments that, over the course of the disease, significantly impair quality of life. The disease ultimately causes death. Symptoms of Huntington's disease, which generally develop in midlife and become progressively more debilitating as time passes, can also develop in infancy or old age. Once overt symptoms start, patients live for about 15 to 20 years. One person in 10,000 is believed to carry this mutation in the huntingtin gene. There is currently no way to delay the onset of symptoms or slow the progression of Huntington's disease.

### About CHDI Foundation, Inc.

CHDI is a private, US based not-for-profit research organization. We work with an international network of scientists to discover drugs that slow the progression or delay the onset of Huntington's disease (HD). CHDI seeks to accelerate scientific progress by serving as a collaborative enabler. We encourage and support cooperation and collaboration among HD researchers. Our strategy is to encourage researchers to develop practical ideas, useful research materials, and powerful technologies. CHDI's activities extend from exploratory biology to the identification and validation of therapeutic targets, and from drug discovery and development to clinical studies and trials.

### About Cogane™

Cogane™ (PYM50028) is a novel non-peptide, orally bioavailable neurotrophic factor inducer that readily crosses the blood-brain barrier. In preclinical models, Cogane™ increases the body's own production of a group of proteins called neurotrophic factors. One of these factors, "BDNF", is known to be decreased in the brains of HD patients. Increasing the brain level of BDNF has been postulated as a potential treatment for HD. However, since BDNF is a protein it cannot be given orally (in pill or liquid form) because it is degraded in the stomach and intestine, and also does not readily cross the blood-brain barrier. Cogane™, which can be taken orally, readily distributes into the brain and stimulates the release of endogenous BDNF in the brain. It therefore has the potential to overcome the technical difficulties associated with exogenous BDNF administration.