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Boehringer Ingelheim and Vitae Pharmaceuticals announce a collaboration to fight Alzheimer`s disease

Boehringer Ingelheim and Vitae Pharmaceuticals, Inc., announced today that they have entered into a significant worldwide collaboration to research and develop beta-secretase (BACE) inhibitors for the treatment of Alzheimer's disease. Current therapies for Alzheimer's disease can improve symptoms, but do not affect the progression of the disease. The inhibition of BACE - an enzyme involved in the formation of amyloid-beta plaques which accumulate in the brains of patients with Alzheimer's disease - offers the potential to slow or even halt disease progression.

Under the terms of the collaboration agreement, Vitae will receive $42 million in upfront and near-term payments from Boehringer Ingelheim, consisting of upfront cash, an equity investment in Vitae, and research funding to support further discovery efforts. In addition, Vitae will be eligible to receive $200 million in pre-commercial milestone payments based on the achievement of clinical and regulatory goals, as well as further milestone payments based on potential additional compounds and / or other approved indications. Vitae will receive commercial performance payments and royalties from Boehringer Ingelheim on all potential future product sales. Further financial details were not disclosed.

The companies will work jointly to identify and advance candidates for clinical development. Thereafter, Boehringer Ingelheim will lead development and commercialization of all products for Alzheimer's disease to capitalize on its global marketing and sales expertise. Vitae will have the right to develop products independently for certain other indications.

"I am very pleased to be working with Boehringer Ingelheim's exceptional neuroscience group on this program", said Jeffrey Hatfield, CEO of Vitae. "This collaboration accomplishes three important objectives for our company. It adds substantial neuroscience expertise and specialized resources to the BACE program, which has advanced remarkably during its 16-month life. It also extends what is already a very successful partnership model with Boehringer Ingelheim, stemming from our existing diabetes and metabolic syndrome collaboration. Boehringer Ingelheim's team-oriented culture presents an ideal environment for partnership success. And finally, it continues Vitae's business model of financing the company's growth through rapid value creation and partnering versus relying on the capital markets – which is favorable for our shareholders."

Dr Manfred Haehl, Corporate Senior Vice President R&D and Medicine of Boehringer Ingelheim worldwide, added that "Based on our research experience in Alzheimer`s disease and our excellent experience in collaborating with Vitae for the diabetes program, we will be using our overall expertise in CNS disease research, drug development and commercialization to strengthen our current neuroscience portfolio. Ultimately, we all aim to create new treatments for patients suffering from this serious debilitating disease. We therefore will see the companies` joint efforts expanding to create medicines that are urgently needed in two widespread disease areas, namely diabetes and Alzheimer's disease. It is part of our core development strategy to establish long-term alliances with innovative companies that broaden the scope of our own exciting pipeline successes."

Boehringer Ingelheim 14.06.2009

About BACE Inhibition
Alzheimer's disease is a chronic neurodegenerative disorder characterized by progressive memory loss associated with the deposition of neuritic plaques in the brains of patients. The amyloid-beta peptide is the major component of such plaques and is considered to be the major culprit in the pathogenesis of Alzheimer's disease. Amyloid-beta is generated from amyloid precursor protein (APP) by proteolytic processing by beta and gamma secretases. Since beta-secretase (beta-site APP cleaving enzyme 1, or BACE1) cleavage is rate limiting for the production of amyloid-beta, inhibition of this enzyme represents an attractive strategy to ameliorate amyloid-beta plaque deposition in Alzheimer's disease. Supporting this notion are studies demonstrating that deletion of the BACE1 gene in mice prevents the formation of amyloid-beta in cultured neurons and the brain. In addition, it has been shown that amyloid-beta-associated memory deficits, which occur in mutant mice overexpressing APP, are prevented when the BACE1 gene is deleted. Thus in patients, it is anticipated that

inhibitors blocking BACE1 could prevent the build up of amyloid-beta plaques and help slow or stop the progression of disease.

About Alzheimer's Disease
Alzheimer`s disease (AD) is the most common form of dementia in adults. It is estimated to affect 4.5 million American's and over 30 million people worldwide with an average course of 8 -12 years. It is projected that the prevalence of AD will double over the next 20 years. Marketed treatments address some symptoms, however there are no treatments available that delay or halt the progression of the disease. Global sales of Alzheimer's drugs were approximately $5 billion in 2008 and are expected to exceed $14 billion by 2015.

Press release

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