

PRESS RELEASE

Cystic Fibrosis – Axentis Pharma Takes Over Patents and Continues Development of Innovative Platform Technology

Zurich, 26 June 2008. The rights to key patents for an innovative screening tool for treatments for cystic fibrosis were transferred today to Swiss biotech company Axentis Pharma AG. The company has acquired the rights from an Austrian firm that has made a vital contribution to the development of new approaches to treating the disease. The core element of the platform technology involves human proteins which – despite offering sufficient functionality – are subjected to enzymatic digestion in untreated cells due to genetic mutations in their structure.

Axentis Pharma AG (Switzerland) today announced that, as part of its takeover of an Austrian research company, it has also acquired two patent families that incorporate a series of international patent applications. The company is taking over these rights from Austrian-based BioDevelops Pharma GmbH, which three years ago identified the therapeutic potential offered by a new approach to the treatment of cystic fibrosis and secured the relevant rights.

The technology directly targets the functional cause of cystic fibrosis – the process whereby the body destroys mutated proteins which were originally designed to regulate the concentration of salt in the epithelial cells of the lungs and other organs. In patients with cystic fibrosis, a genetic defect means that many such proteins are produced with minor structural mutations. Although the mutated proteins still offer sufficient functionality, they are eliminated by quality control functions in the endoplasmic reticulum of the cells. The therapeutic approach now under development at Axentis Pharma enables these proteins – which are still functional despite their mutated structures – to escape enzymatic digestion.

Dr. Gergely Lukacs who guided the experimental set-up in his labs at the Sick Kids Hospital in Toronto explains: "In patients with cystic fibrosis, the protein CFTR (cystic fibrosis conductance regulator) with mutated structure is marked with ubiquitin. These ubiquitin-marked proteins are degraded by multiple mechanisms prematurely. If this marker is missing, the protein evades destruction and can regulate the concentration of water and salt secretion in the lung despite its minor structural defect.

Joerg Zielasek, president of Axentis Pharma AG, comments on what the acquisition of these rights means for the company: "Just six months ago, we reached agreement with aRigen Pharmaceuticals, Japan, on terms and conditions regarding an exclusive license for developing and marketing a patented system for the liposomal encapsulation of an active ingredient designed to combat lung infections in patients with cystic fibrosis. By acquiring the rights to BioDevelops' technology platform, Axentis Pharma is both enhancing its development pipeline and offering both patients and investors extensive prospects for the treatment of this disease."

About Axentis Pharma AG (www.axentispharma.com)

Axentis Pharma is a respiratory specialty pharmaceutical company which core competence is the application of a fully patented, encapsulating drug delivery system to already established and well-characterized therapeutic agents. Currently, the company is using this technology, named Fluidosome™ technology, for the development of its lead product, a clinical stage treatment against cystic fibrosis (CF).

About Fluidosome™ technology

Axentis Pharma's Fluidosome™ technology uses biocompatible lipids endogenous to the lung that are formulated into small liposomes. This nanocapsule platform offers wide-ranging potential for unmet medical needs, including other respiratory diseases. In the case of Fluidosome™-tobramycin, the interaction between tobramycin and the microbial cell is triggered when the liposomes attach to the outer cell membrane. Tobramycin then leaches into the inner cell compartment, which leads to rapid cell death.

About cystic fibrosis

Cystic fibrosis is the most common life-threatening hereditary disease amongst Caucasian populations. The disease is caused by a mutation in the cystic fibrosis transmembrane conductance regulator (CFTR) gene found on chromosome 7. This mutation causes increased secretion deposits on mucous membranes. Lung complications represent the most serious manifestation of the disease – and the reason for the high mortality rate amongst patients. Such complications often involve infection of the bronchi by the bacteria *Pseudomonas aeruginosa*. Chronic inflammations then cause lung functions to become blocked. As well as the breakdown of lung tissue, this also leads to bronchiectasis and lung failure.

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