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Intellect Neurosciences Files New Patent Applications for Immunotherapy Methods to Target Abnormal Tau Protein in Treatment of Alzheimer's disease

NEW YORK, Feb. 7, 2012 /PRNewswire/ -- Intellect Neurosciences, Inc. (OTCBB:ILNS), a biopharmaceutical company engaged in the discovery and development of disease-modifying therapeutic agents for the treatment of Alzheimer's and other neurological diseases today announced it has filed two United States and one international patent application regarding passive and active immunotherapy approaches targeting abnormal forms of tau protein that lead to death of nerve cells in Alzheimer's disease.

(Logo: https://photos.prnewswire.com/prnh/20111214/NY22484LOGO)

Intellect's new approach specifically targets neoepitopes that are formed following cleavage of intact tau protein by "executioner" caspases. This pathological process is stimulated by an accumulation of amyloid beta in the brain of Alzheimer's patients. It is believed that the smaller cleaved tau, sometimes referred to as delta tau, is especially toxic and prone to form tangles inside nerve cells. Importantly, Intellect's approach uses antibodies to clear this more toxic form, without risk of interfering with the functions of normal intact tau protein.

"These new patent filings continue to demonstrate our leadership in translational Alzheimer's research and ongoing commitment to develop improved next-generation drug products," said Dr. Daniel G. Chain, Intellect's chairman & CEO. "We are encouraged by the results of two recent studies conducted by independent groups from Columbia University and Harvard Medical School suggesting that tau may cause Alzheimer's disease to spread through the brain. Their findings indicate that an antibody approach may halt that spread, which supports our strategy of a combined vaccine against beta amyloid and delta tau that may potentially eliminate the disease."

Intellect Neuroscience is investigating the combination of its new technology for which it has filed the recent patents with its RECALL-VAX active vaccine platform, which was originally developed to target neoepitopes in amyloid beta. The company has manufactured a combination test vaccine from its RECALL-VAX platform, RV03, to target both beta amyloid and delta tau, which are increasingly understood to act in concert with synergistic effects in the pathogenesis of Alzheimer's disease. The company is embarking on a series of preclinical studies with RV03, using brain imaging and other translational tools to test both safety and efficacy to facilitate design of future clinical trials.

About Intellect

Intellect Neurosciences, Inc. develops innovative approaches aimed at arresting or preventing Alzheimer's disease and other neurodegenerative diseases especially focused on proteinopathies. Intellect's pipeline includes therapeutic vaccines, antibodies and neuroprotective antibody drug conjugates.

The company is currently developing products based on three platform technologies: ANTISENILIN® is Intellect's Alzheimer's beta amyloid monoclonal antibody platform technology, which underlies a product in Phase 3 clinical trials licensed to major pharmaceutical companies. ANTISENILIN also underlies IN-N01, a humanized monoclonal antibody being developed by the company as an antibody dug conjugate (ADC). IN-N01-OX2 is the first candidate to emerge from the company's CONJUMAB-A platform technology, which is based on a novel application of antibody drug conjugates in which the antibody is chemically conjugated to a small molecule (OX2) that has potent neuroprotective properties both as an antioxidant and inhibitor of protein aggregation. RECALL-VAX is a therapeutic vaccine technology that underlies three preclinical drug candidates, RV01 and RV02, which target beta amyloid and delta tau protein, respectively, and RV03 which is a combination of the two.

Intellect is seeking to partner/license its platform technologies to develop antibody-drug conjugates and therapeutic vaccines. RECALL-VAX and CONJUMAB-A have potential applications for treatment of serious diseases such as Alzheimer's, Parkinson's, Huntington's, Cerebral Amyloid Angiopathy, Frontotemporal Dementia, Progressive Supranuclear Palsy, Pick's disease, Cortical Basal Degeneration, Age-Related Macular Degeneration, Glaucoma, and Peripheral Amyloidosis.

The company recently licensed OX1, a small molecule multimodal antioxidant, to ViroPharma, Inc. for Friedreich's Ataxia and other neurodegenerative diseases. For more information, please visit www.intellectns.com.

Safe Harbor Statement Regarding Forward-Looking Statements:
The statements in this release and oral statements made by representatives of Intellect relating to matters that are not historical facts (including, without limitation, those regarding future performance or financial results, the timing or potential outcomes of research collaborations or clinical trials, any market that might develop for any of Intellect's product candidates and the sufficiency of Intellect's cash and other capital resources) are forward-looking statements that involve risks and uncertainties, including, but not limited to, the likelihood that actual performance or results could materially differ, that future research will prove successful, the likelihood that any product in the research pipeline will receive regulatory approval in the United States or abroad, or Intellect's ability to fund such efforts with or without partners. Intellect undertakes no obligation to update any of these statements. Readers are cautioned not to place undue reliance on these forward-looking statements, which speak only as to the date hereof. Accordingly, any forward-looking statements should be read in conjunction with the additional risks and uncertainties detailed in Intellect's filings with the Securities and Exchange Commission, including those factors discussed under the caption "Risk Factors" in Intellect's Annual Report on Form 10-K (file no. 333-128226), filed on October 13, 2011, and in our Quarterly Report on Form 10-Q for the quarterly period ended December 31, 2011, filed on February 2, 2012.

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