New Peer Reviewed Article Describes Role of TauC3 in AD Transgenic Mice

TauC3 Mice Showed Drastic Learning and Spatial Memory Deficits and Reduced Synaptic Density at a Young Age (2-3 months); First Published Study Describing Role of TauC3 in Vivo; Study Independently Conducted by Scientists at School of Biological Sciences, Seoul National University, Seoul, Republic of Korea

INGLEWOOD CLIFFS, NJ -- (Marketwired) -- 01/07/16 -- Intellect Neurosciences, Inc. (OTCQB: ILNS) ("Company" or "Intellect"), a biopharmaceutical company engaged in the discovery and development of disease-modifying therapeutic agents for the treatment and prevention of rare neurodegenerative conditions, today announced the publication of a scientific study of the role of TauC3 in transgenic mice in the prominent, peer-reviewed journal, Neurobiology of Disease [2015, 87:19-28]. The paper, titled "Caspase-cleaved tau exhibits rapid memory impairment associated with tau oligomers in a transgenic mouse model," describes the findings of a recent preclinical study of caspase-cleaved tau in a transgenic mouse model. The study marks the first published study indicating that the expression of caspase-cleaved tau (TauC3) in mice induces early memory deficit and reduced synaptic density. In addition, the study demonstrated that hyperphosphorylated tau oligomers and aggregates appear in TauC3 mice showing memory deficit and that aggregation blocker or rapamycin rescues memory impairment and reduces tau oligomers in TauC3 mice. The TauC3 mouse in the study is an Alzheimer's disease (AD) model used elucidate tau oligomer-associated pathogenesis and to evaluate therapeutic drug options. The paper was co-authored by scientists at the Global Research Laboratory, School of Biological Sciences, Seoul National University, Seoul, and the Department of Neurology and Neuroscience & Cell Biology, University of Texas, Galveston, Texas.

As described in the Abstract of the article, in neurodegenerative diseases like AD, tau forms neurofibrillary tangles, composed of tau protein. In the AD brain, activated caspases cleave tau at the 421th Asp, generating a caspase-cleaved form of tau, TauC3. Although TauC3 is known to assemble rapidly into filaments in vitro, the role of TauC3 in vivo remains unclear. In the study, scientists generated a transgenic mouse expressing human TauC3 using a neuron-specific promoter. The researchers found that human TauC3 was expressed in the hippocampus and cortex. Interestingly, the TauC3 mice showed drastic learning and spatial memory deficits and reduced synaptic density at a young age (2-3months). Notably, tau oligomers as well as tau aggregates were found in TauC3 mice showing memory deficits. These results suggest that TauC3 facilitates early memory impairment in transgenic mice accompanied with tau oligomer formation, providing insight into the role of TauC3 in the AD pathogenesis associated with tau oligomers and a useful AD model to test drug candidates.
Dr. Troy Rohn, Professor, Department of Biological Sciences, Boise State University, Boise, Idaho, Intellect's lead scientific advisor, commented, "This study is exciting because it provides proof-of-concept data regarding the role of TauC3 in various tauopathies. It is well known that the generation of the TauC3 fragment by caspases occurs in numerous neurodegenerative diseases and may facilitate the pathology associated with these disorders. However, to date, whether the TauC3 fragment itself can lead to behavioral deficits has been lacking. This in vivo study confirms that hypothesis and strongly supports targeting the TauC3 fragment as a therapeutic strategy in various diseases in which the fragment is produced."

In 2012, Intellect licensed a unique TauC3 antibody from Northwestern University, acquiring exclusive global rights to develop and commercialize the antibody. In January 2014, the Company announced top line data showing initial proof of concept in a preclinical Alzheimer's model, indicating the antibody's potential to be disease modifying. The study was conducted in collaboration with Dr. Frank LaFerla, University of California, Irvine, Chancellor's Professor and Chair, Neurobiology and Behavior School of Biological Sciences, Director, Institute for Memory Impairments and Neurological Disorders. The data showed that the TauC3 monoclonal antibody effectively engaged the target and reduced certain phosphorylated pathological forms of tau, indicating that the treatment with the peripherally administered antibody had an effect in the brain and potentially is disease modifying. In 2015, Intellect sponsored a research collaboration with Professor Bradley T. Hyman MD, PhD, Director, Massachusetts Alzheimer's Disease Research Center & Co-Director, Massachusetts General Hospital Memory Disorders Unit and John B. Penney Jr. Professor of Neurology, Harvard Medical School. The research project was designed to examine the detailed molecular mechanism affecting propagation of tau pathology and was aimed at developing a novel treatment for Alzheimer's disease and other tauopathies. The research yielded important data regarding target engagement, which the Company plans to use to strengthen its proprietary position in the TauC3 antibody. Intellect is planning to test its TauC3 monoclonal antibody in several orphan disease preclinical models in early 2016.

About Intellect Neurosciences, Inc.

Intellect Neurosciences, Inc. is a biopharmaceutical company engaged in the discovery and development of disease-modifying therapeutic agents for the treatment and prevention of rare neurodegenerative conditions, collectively known as proteinopathies. Compounds under development include OX1, a high affinity, copper binding molecule that protects the body against free radicals. OX1 is licensed to Shire plc, which recently completed a single ascending dose Phase 1 study to evaluate the safety, tolerability and PK/PD of OX1 (renamed by Shire as "SHP622") in subjects with Friedreich's Ataxia. Details of the study, entitled, "A Phase 1, Randomized, Double-blind, Placebo-controlled, Multicenter, Single and Multiple Ascending Dose Study to Evaluate the Safety, Tolerability, Pharmacokinetics, and Pharmacodynamics of Oral VP 20629 in Adult Subjects with Friedreich's Ataxia", may be found at: www.clinicaltrials.gov, identifier NCT01898884. For more information, please visit www.intellectns.com.

Safe Harbor Statements Regarding Forward Looking Statements
The statements in this letter made by representatives of Intellect Neurosciences 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, the continued development by Shire of SHP622 or its determination to seek Orphan Drug designation for the pharmaceutical product of SHP622 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 discussed in Intellect's Annual Report on Form 10-K (file no. 333-128226) filed on October 13, 2015 and Quarterly Report for the period ended September 30, 2015 filed on November 12, 2015.

CONTACT:
Intellect Neurosciences, Inc.
ir@intellectns.com
201-608-5101

Source: Intellect Neurosciences, Inc.