Central Asian Journal of Global Health

Immunocytochemical Characterization of Alzheimer's Disease Hallmarks in APP/PS1 Transgenic Mice Treated with a New Anti-Amyloid-β Vaccine

Ivan Carrera[1], Ignacio Etcheverria[1], Yi Li[2], Lucia Fernandez-Novoa[1], Valter Lombardi[1], Carmen Vigo[3], Hector H. Palacios[4], Valery V. Benberin[5], Ramon Cacabelos[6], Gjumrakch Aliev[7,8]

[1]Department of Neurosciences, EuroEspes Biotechnology, La Coruna, Spain; [2]Yale University School of Medicine, New Haven, CT; [3]Atlas Pharmaceuticals, Sunnyvale, CA; [4]National Institute on Aging, National Institutes of Health, Baltimore, MD; [5]Medical Center of the Administration of the President of the Republic of Kazakhstan, Astana, Kazakhstan; [6]EuroEspes Biomedical Research Center, Institute for CNS Disorders and Genomic Medicine, La Coruna, Spain; [7]GALLY International Biomedical Research Consulting LLC, San Antonio, TX; [8]Department of Health Science and Healthcare Administration, University of Atlanta, Atlanta, GA

Vol. 2, Suppl. (2013) | ISSN 2166-7403 (online)
DOI 10.5195/cajgh.2013.119 | http://cajgh.pitt.edu

Abstract

Introduction: APP/PS1 double-transgenic mouse models of Alzheimer's disease (AD), which overexpress mutated forms of the gene for the human amyloid precursor protein (APP) and presenilin 1 (PS1), have provided robust neuropathological hallmarks of an AD-like pattern at early ages. This study aimed to characterize immunocytochemical patterns of the AD mouse brain, which is treated with the EB101 vaccine, as a model for human AD.

Material and methods: In this novel vaccine, a new approach has been taken to circumvent past failures with Aβ vaccines by judiciously selecting an adjuvant consisting of a physiological matrix embedded in liposomes, composed of naturally occurring phospholipids (phosphatidylcholine, phosphatidylglycerol, and cholesterol).

Results: Our findings showed that the administration of amyloid-β1−42 (Aβ) and sphingosine-1-phosphate emulsified in liposome complex (EB101) to APP/PS1 mice before the onset of Aβ brain deposition (at 7 weeks of age) and/or at an older age (35 weeks of age) can be effective in both halting the progression and clearing the AD-like neuropathological hallmarks. In addition, passive immunization with EB101 did not activate inflammatory responses from the immune system and astrocytes. Consistent with a decreased inflammatory background, the basal immunological interaction between the T cells and the affected areas (hippocampus) in the brain of treated mice was notably reduced.

Conclusion: These results provide strong evidence that immunization with the EB101 vaccine prevents and attenuates AD neuropathology in this type of double-transgenic mice.

Keywords: *Alzheimer's disease, anti-amyloid-β vaccine*