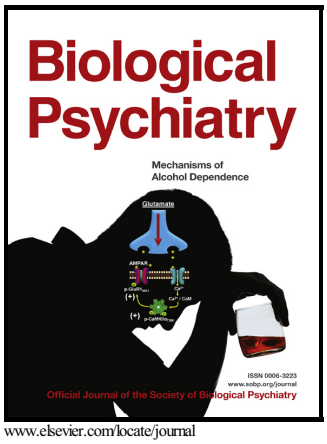
Apolipoprotein E, Receptors and Modulation of Alzheimer's Disease

Na Zhao, Chia-Chen Liu, Wenhui Qiao, Guojun Bu



www.elsevier.com/locate/journal

PII: S0006-3223(17)31358-6
DOI: http://dx.doi.org/10.1016/j.biopsych.2017.03.003
Reference: BPS13154

To appear in: *Biological Psychiatry*

Cite this article as: Na Zhao, Chia-Chen Liu, Wenhui Qiao and Guojun Bu, Apolipoprotein E, Receptors and Modulation of Alzheimer's Disease, *Biological Psychiatry,* http://dx.doi.org/10.1016/j.biopsych.2017.03.003

TITLE PAGE

Apolipoprotein E, Receptors and Modulation of Alzheimer's Disease

Na Zhao[1], Chia-Chen Liu[1], Wenhui Qiao[1], Guojun Bu[1,2*]

(Short title: ApoE, Receptors and Modulation of AD)

[1]Department of Neuroscience, Mayo Clinic, Jacksonville, FL, 32224, USA

[2]Fujian Provincial Key Laboratory of Neurodegenerative Disease and Aging Research, Institute of Neuroscience, College of Medicine, Xiamen University, Xiamen, Fujian, 361005, China

[*]Correspondence should be addressed to Guojun Bu, Department of Neuroscience, Mayo Clinic, 4500 San Pablo Road, Jacksonville, Florida 32224. E-mail: bu.guojun@mayo.edu.

Keywords: Apolipoprotein E; low-density lipoprotein receptor family; Alzheimer's disease; amyloid-β; tauopathy; synaptic plasticity.

ABSTRACT:

Apolipoprotein E (apoE) is a lipid carrier in both periphery and the central nervous system (CNS). Lipid-loaded apoE lipoprotein particles bind to several cell surface receptors to support membrane homeostasis and injury repair in the brain. Considering prevalence and relative risk magnitude, the ε4 allele of the *APOE* gene is the strongest genetic risk factor for late-onset Alzheimer's disease (AD). ApoE4 contributes to AD pathogenesis by modulating multiple pathways including but not limited to the metabolism, aggregation, and toxicity of amyloid-β (Aβ) peptide, tauopathy, synaptic plasticity, lipid transport, glucose metabolism, mitochondrial