PULSUS
WWW.PULSUSCONFERENCE.COM

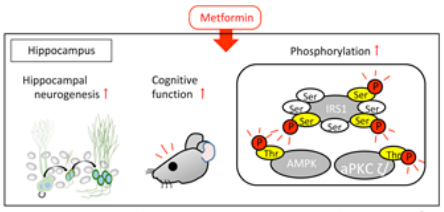
Akiko Taguchi, J Immunol Tech Infect Dis 2018, Volume 7
DOI: 10.4172/2329-9541-C2-010

8th Global Summit on
Neuroscience and Neuroimmunology
August 27-28, 2018 Madrid, Spain

Akiko Taguchi
National Center for Geriatrics and Gerontology, Japan

Metformin enhances neurogenesis and memory function via hippocampal insulin signaling independent of the hypoglycemic effect

The aging systemic milieu leads to a decline in hippocampal neurogenesis and cognitive functions, which also occurs in diabetes. Despite growing concern regarding the potential role of diabetic drugs in neural abnormalities, their effects on progressive deterioration of neurogenesis and cognitive functions remain unknown. Metformin, a biguanide anti-diabetic medication is the first-line drug for type-2 diabetes and lowers blood glucose levels by decreasing basal hepatic glucose output and increasing glucose uptake by skeletal muscle through activation of the AMP-Activated Protein Kinase (AMPK). Here we show that prolonged treatment with metformin enhances hippocampal neurogenesis while countering the microglial activation in the context of the combination of aging and diabetes in mice. Although chronic therapy with metformin fails to achieve recovery from hyperglycemia, a key feature of diabetes, it improves hippocampal-dependent spatial memory functions accompanied by increased serine/threonine phosphorylation of AMPK, Atypical Protein Kinase C ζ (aPKC ζ) and Insulin Receptor Substrate 1 (IRS1), a major mediator of the insulin/IGF1R signaling, in the hippocampus. Our findings suggest that signaling networks acting through long-term metformin-stimulated phosphorylation of AMPK, aPKC ζ/λ and IRS1 serine sites contribute to neuro-protective effects on hippocampal neurogenesis and cognitive function independent of a hypoglycemic effect.



Summary: Chronic metformin-stimulated phosphorylation of AMPK, aPKC ζ/λ, and IRS1 serine sites is involved in signaling networks that maintain hippocampal neurogenesis and cognitive function.

Biography

Akiko Taguchi has obtained her BSc from Tokyo University of Agriculture in Tokyo, Japan and her PhD from Osaka University Graduate School of Medicine, in Osaka, Japan. She worked as a postdoctoral research fellow and a research associate in Dr. Morris White's lab at Harvard Medical School, Boston, USA. She is interested in the interaction between lifestyle-related diseases such as diabetes and neurological disorder such as dementia. She is particularly interested in the roles of neural insulin/IGF1R signaling in cognitive functions and the effects of existing medications on brain functions (drug repositioning). She has received several grants from Grant-in-Aid for Scientific Research on Innovative Areas, Grant-in-Aid for Scientific Research, Grant-in-Aid for Challenging Exploratory Research and Private Foundations in Japan.

taguchia@ncgg.go.jp