



A Rare Sugar D-Allulose Improves Synaptic Plasticity in D-galactose-Induced Aged Rats

Yanmei Huang^{1,2}, Chanisa Thonusin^{1,2,3}, Masaaki Tokuda⁴, Nipon Chattipakorn^{1,2,3,5}, Siriporn C Chattipakorn^{1,2,6*}

¹ Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand

² Center of Excellence in Cardiac Electrophysiology, Chiang Mai University, Chiang Mai 50200, Thailand

³ Cardiac Electrophysiology Unit, Department of Physiology, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand

⁴ Department of Cellular Physiology, Faculty of Medicine, Kagawa University, Kagawa 761-0793, Japan

⁵ The Academy of Science, The Royal Society of Thailand, Bangkok, Thailand

⁶ Department of Oral Biology and Diagnostic Sciences, Faculty of Dentistry, Chiang Mai University, Chiang Mai 50200, Thailand

INTRODUCTION

Aging is one of the major risk factors for cognitive decline and neurodegeneration via aging-induced synaptic dysfunction [1]. Previous evidence suggested that D-allulose improved synaptic plasticity under obesity-associated cognitive impairment [2]. However, its effects on the aging brain remain unclear.

References

[1] Blinkouskaya et al., Mech Ageing Dev, 2021; [2] Prachayasakul et al., Nutr Neurosci, 2022.

Aim

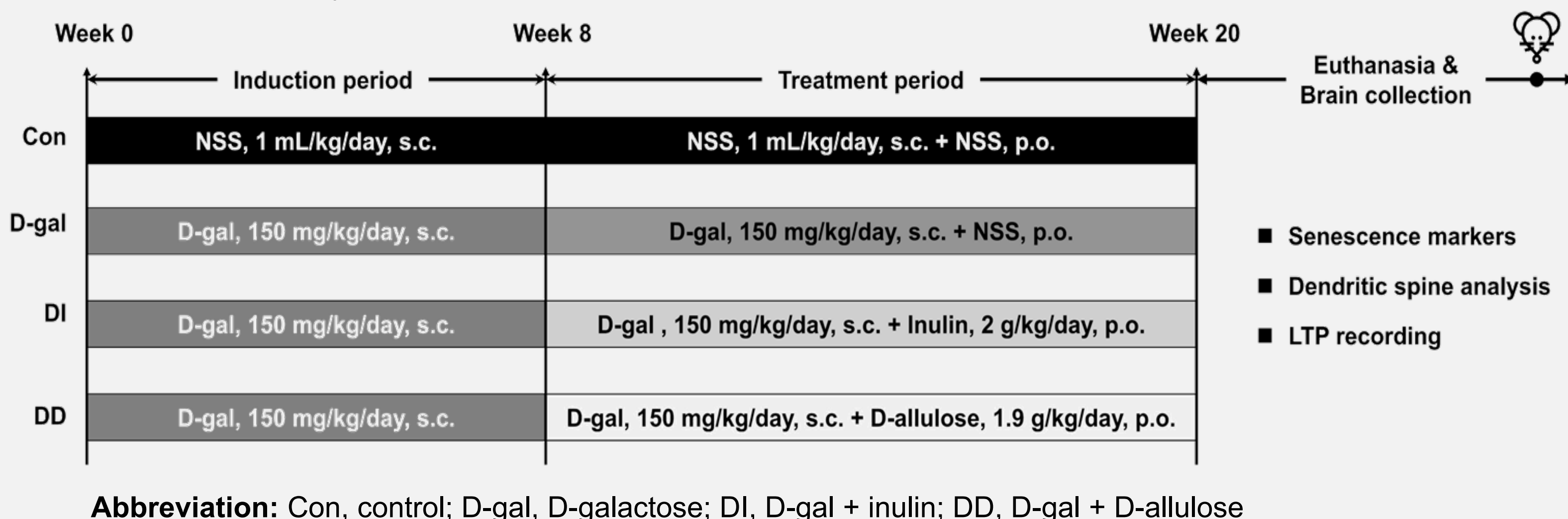
To investigate the effects of D-allulose on hippocampal senescence and synaptic plasticity in D-galactose-induced aged rats

Hypothesis

D-allulose attenuated brain senescence and improved hippocampal synaptic plasticity in D-galactose-induced aged rats.

METHODS

Five-month-old male Wistar rats were divided into four groups: Con, D-gal, DI, and DD (n = 5/group). Rats received subcutaneous D-galactose at 150 mg/kg/day for 20 weeks to induce aging. From week 9 to week 20, D-gal-treated rats were orally administered NSS, inulin, or D-allulose. At week 20, brain tissues were collected for subsequent analyses.



CONCLUSION

D-allulose attenuated brain senescence and improved hippocampal synaptic plasticity in a D-galactose-induced aging model. These findings suggest that D-allulose may help maintain hippocampal synaptic integrity during aging.

ACKNOWLEDGEMENT

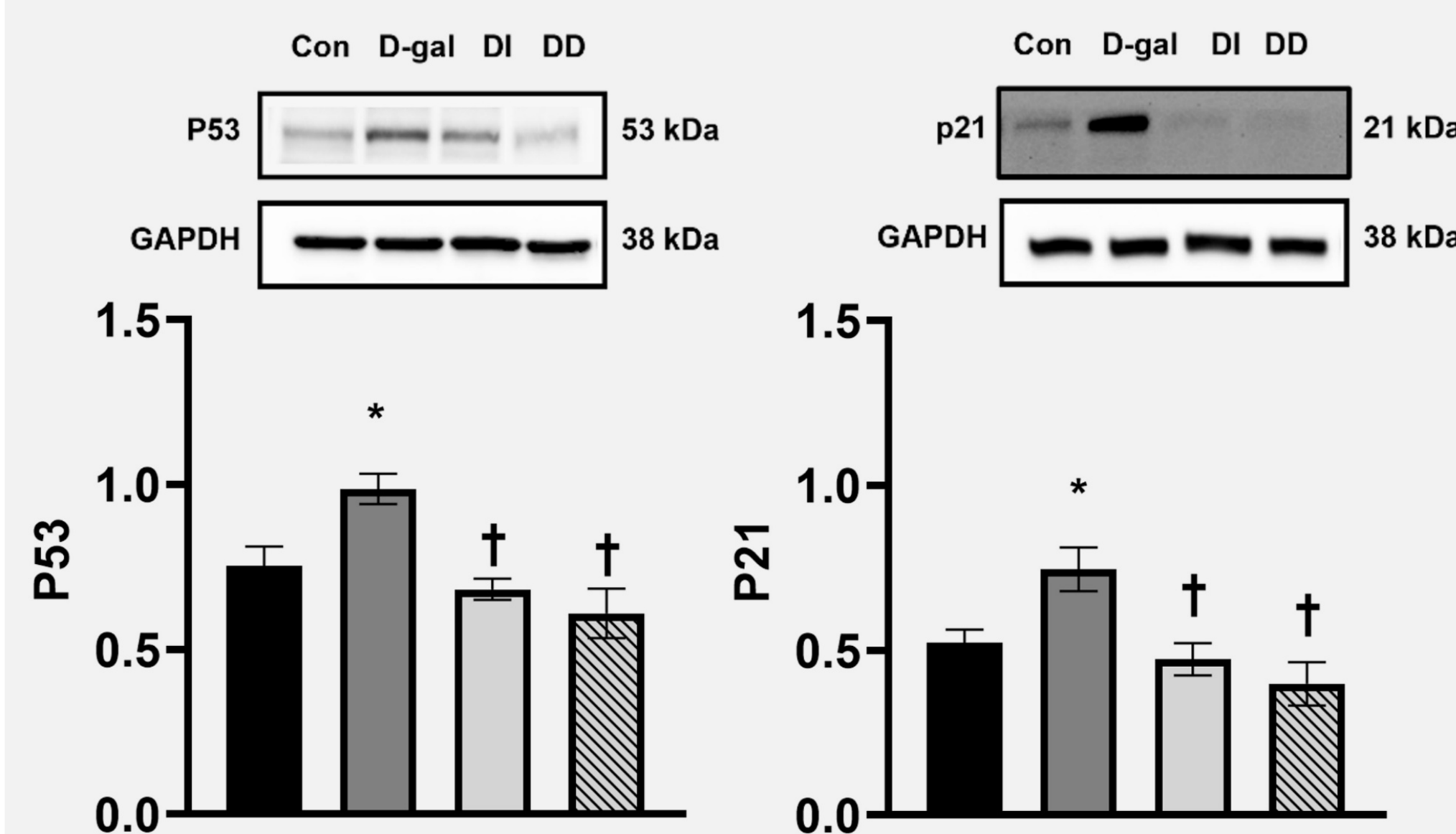
This study was supported by the Distinguished Research Professor Grant from the National Research Council of Thailand (N42A690147 to N.C.), Visiting Professor from Chiang Mai University (M.T), and a Chiang Mai University Centre of Excellence Award (N.C.).



RESULTS

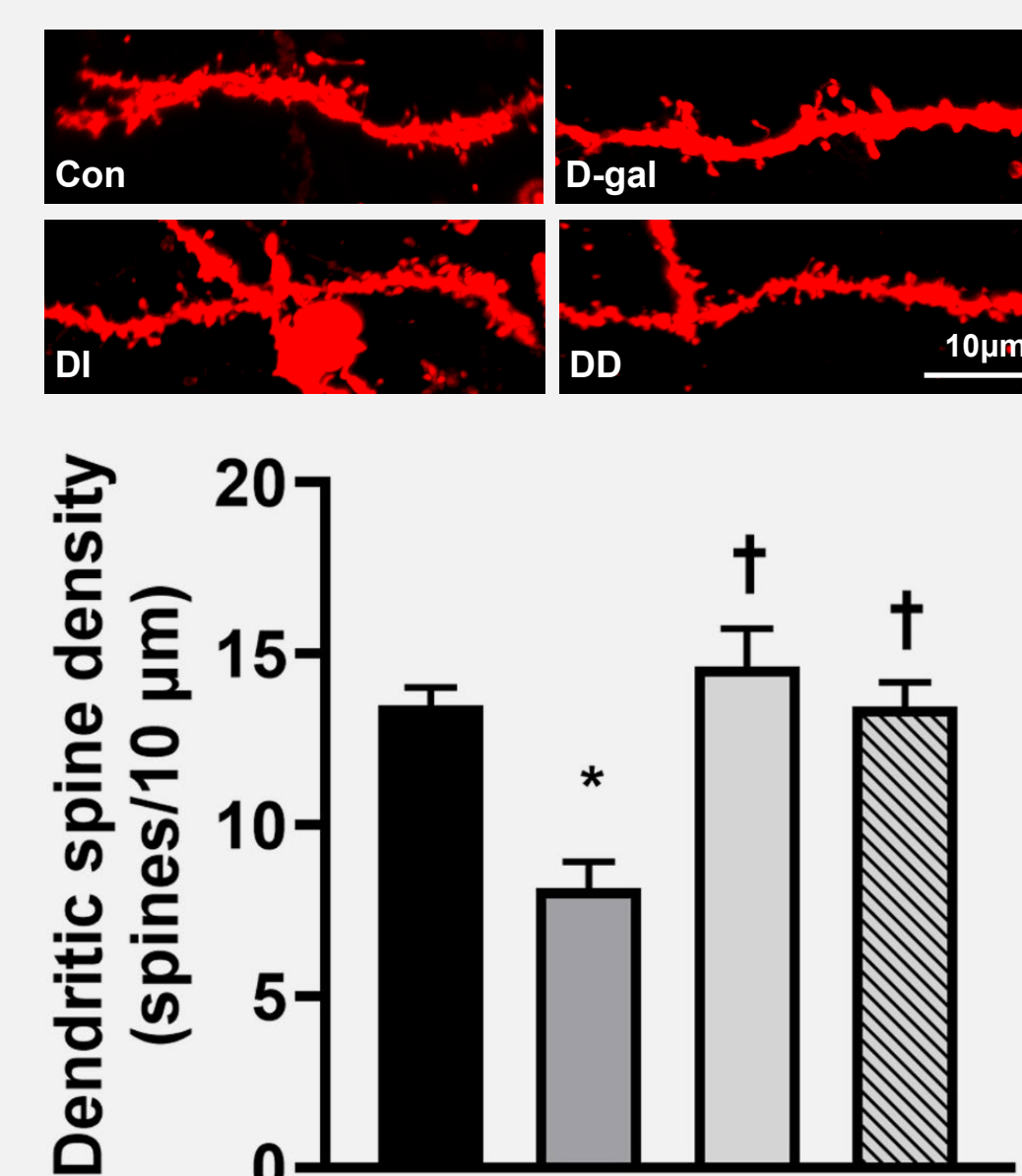
Senescence markers

D-gal-treated rats exhibited cellular senescence, as shown by increased p53 and p21 protein expression. Both D-allulose and inulin reduced these markers, suggesting attenuation of cellular senescence.



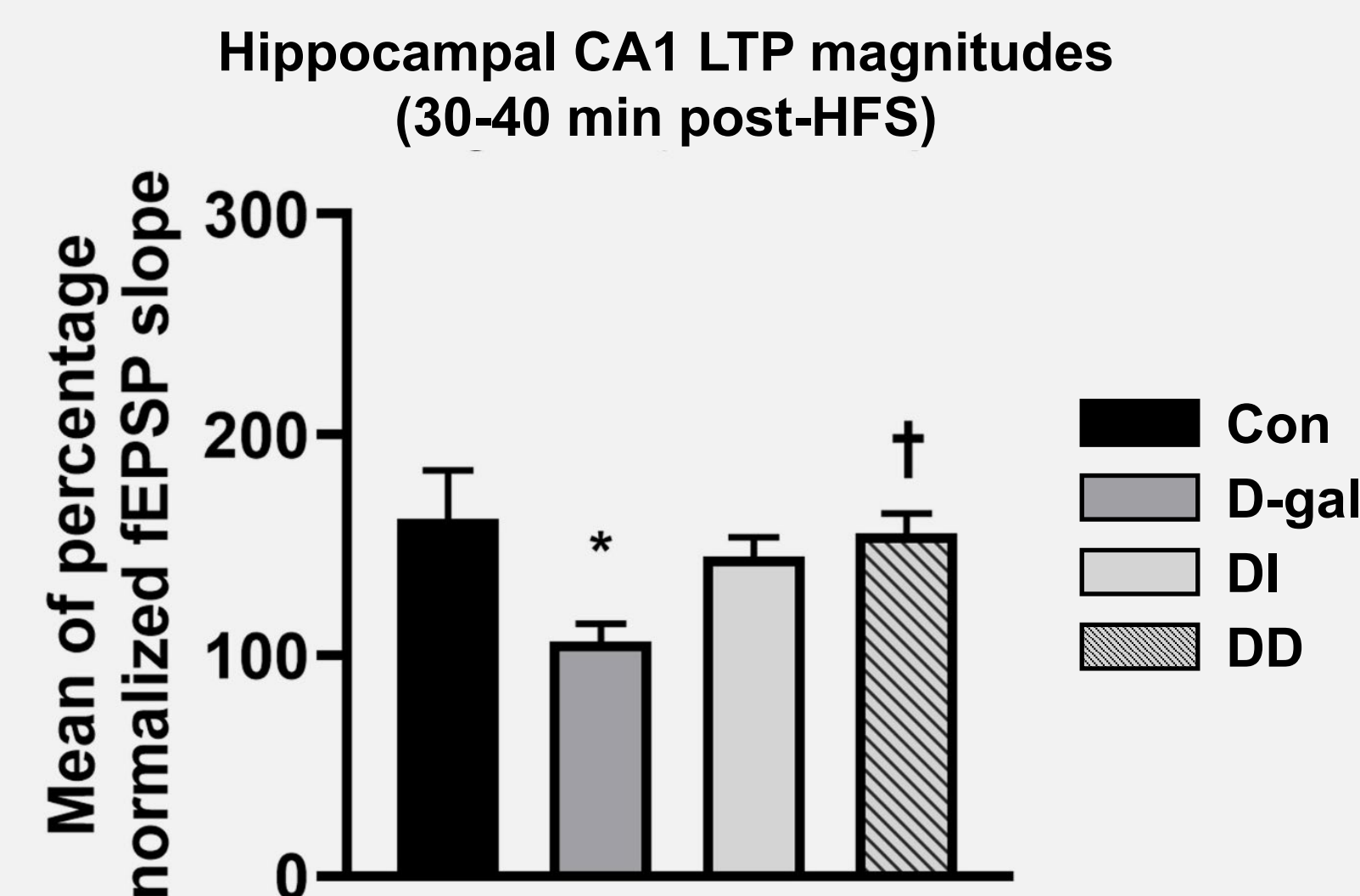
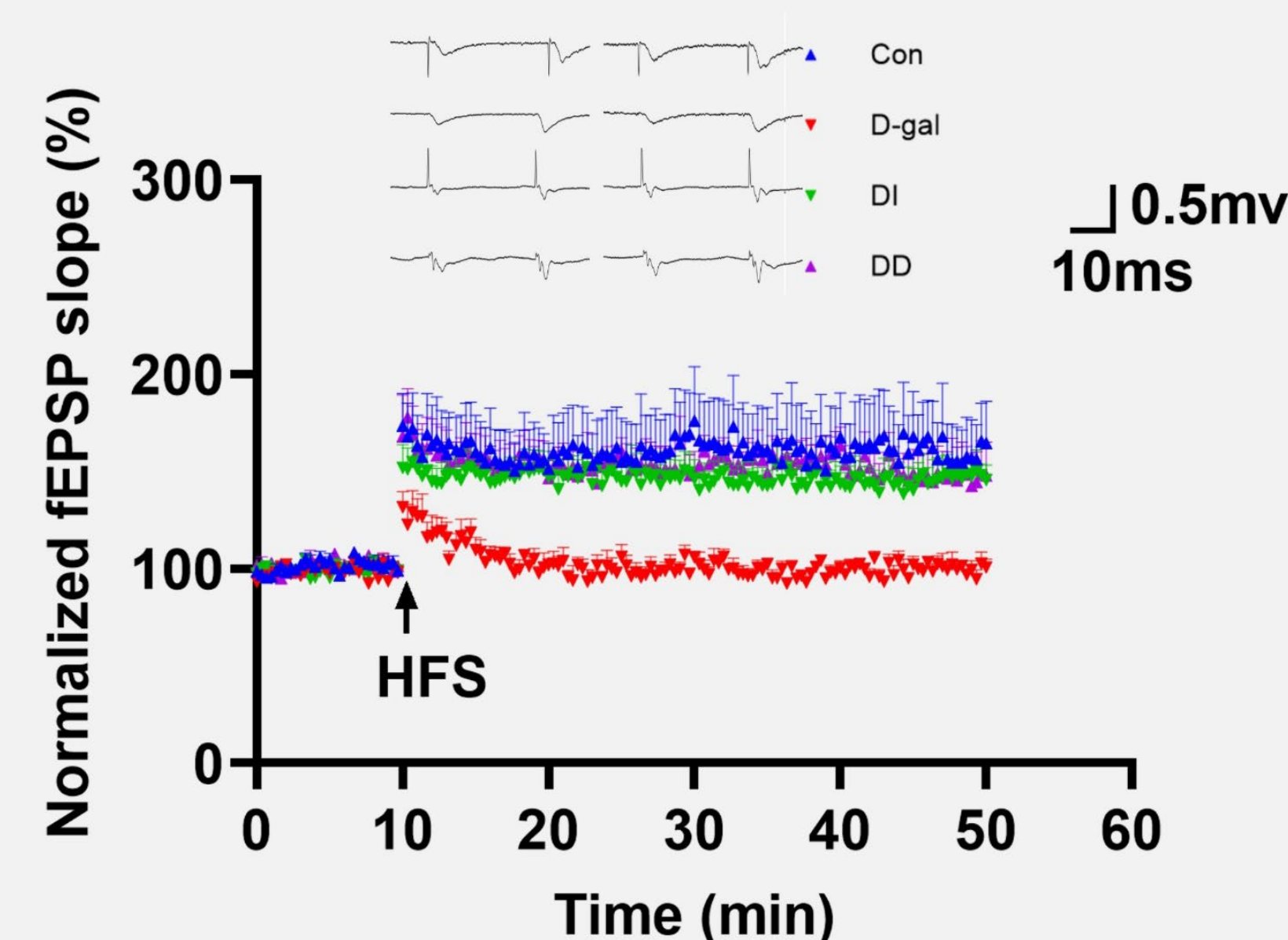
Dendritic spine analysis

D-gal-treated rats showed reduced dendritic spine density. In contrast, both D-allulose and inulin restored this level, suggesting improved hippocampal synaptic integrity.



Ex vivo LTP recording

D-gal-treated rats showed reduced hippocampal CA1 LTP magnitude, indicating impaired functional synaptic plasticity. However, D-allulose treatment restored the normalized fEPSP slope after HFS, suggesting improved hippocampal synaptic function.



Data are presented as mean ± SEM. *p < 0.05 vs. Con; †p < 0.05 vs. D-gal.

Abbreviation: Con, control; D-gal, D-galactose; DI, D-gal + inulin; DD, D-gal + D-allulose; fEPSP, field excitatory postsynaptic potential; LTP, long-term potentiation; HFS, high-frequency stimulation



: Yanmei Huang, MD, PhD Candidate
: Neurophysiology Unit, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, Chiang Mai University, Thailand
: yanmei_huang@cmu.ac.th; huangyanmei_9822@163.com

