Cellular Physiology and Biochemistry
Cell Physiol Biochem 2022;56:312-313
DOI: 10.33594/000000537

Published by Cell Physiol Biochem Press GmbH&Co. KG, Duesseldorf
www.cellphysiolbiochem.com

Erratum

In the article "TRPML1 Participates in the Progression of Alzheimer's Disease by Regulating the PPARγ/AMPK/Mtor Signalling Pathway" [Cell Physiol Biochem 2017;43:2446–2456; DOI: 10.1159/000484449] by Zhang et al., the incorrect representative image was included in Figure 3E APP/PS1 + propranolol as a result of an error at the time of image acquisition, the naming and saving process. The authors have re-photographed the original stained slide.

The corrected Figure 3 is shown here (see next page).

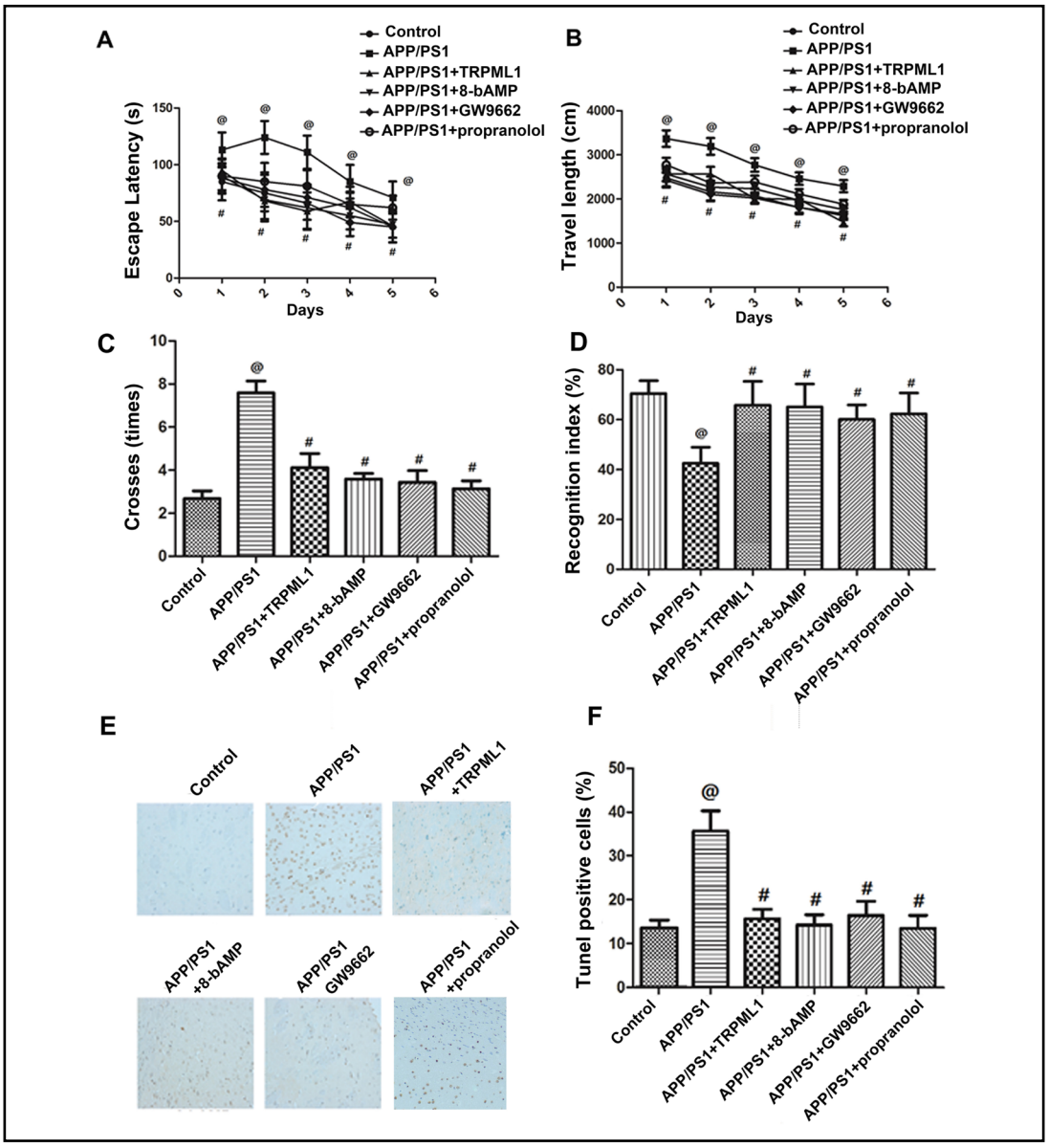

Fig. 3. TRPML1 overexpression and the inhibitors of the PPARγ/AMPK/mTOR signalling pathway rescued the memory and recognition impairments and neuronal apoptosis in mice with the APP/PS1 transgene-sTRPML1 overexpression or treatment with 8-bAMP, GW9662, or propranolol caused significant reductions in the escape latency (A), travel length (B), and time across the platform (C) and markedly improved the recognition index (D) in the APP/PS1 transgenic mice compared to mice without treatment ($p<0.05$; n=8 in each group). (E-F) The neuronal apoptotic rate in the APP/PS1 transgenic mice with TRPML1 overexpression or treatment with 8-bAMP, GW9662 or propranolol was markedly reduced compared to that in mice without treatment ($p<0.05$; n=8 in each group). @$p<0.05$ compared to controls; #$p<0.05$ compared to the APP/PS1 group.