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CORRECTION

Correction to: Isoliquiritigenin Provides Protection and Attenuates Oxidative Stress-Induced Injuries via the Nrf2-ARE Signaling Pathway After Traumatic Brain Injury

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Correction to: Neurochemical Research (2018) 43:2435–2445
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The original version of this article unfortunately contained a mistake. The Fluorescence Immunoassays text written in Materials and Methods section and Fig. 1i, j is incorrect. In Fig. 1j, the images corresponding to Sham and TBI + ILG are incorrect. In Fig. 1i the figure caption "TBI + EDA" are incorrect. The corrected text and Fig. 1i, j are given below.

Fluorescence Immunoassays

After deparaffinization and rehydration, brain tissue section and cells were mounted on coverslips and fixed with 4% paraformaldehyde. These samples then were blocked with 5% bovine serum albumin (BSA) in PBS for 30 min at 37 °C, and incubated overnight at 4 °C with anti-AQP4 (1:100), anti-GFAP (1:300) and anti-NFL (1:500) specific primary detection antibodies. After washing with PBS (7 min/3 times), the slides were then incubated for 2 h at room temperature with the appropriate secondary detection antibody (1:1000). After washing in PBS, the slides were re-stained with DAPI for 7 min. The slices of Negative Control group (Sham group) were incubated with primary antibody (anti-Nrf2,1:400) overnight at 4 °C. Afterward, the slides were incubated with Alexa Fluor 647 donkey antimouse and Alexa Fluor 647 donkey antirabbit secondary antibodies for 1 h at 37 °C. Then, two different fields were randomly selected in each slide, and captured for positive cells as Negative Control group (Donkey antirabbit secondary antibody) and Sham group (Donkey antimouse secondary antibody), respectively. Fluorescence was detected using a Nikon confocal laser microscope (Nikon, A1PLUS, Tokyo, Japan).

The original article can be found online at https://doi.org/10.1007/s11064-018-2671-z.

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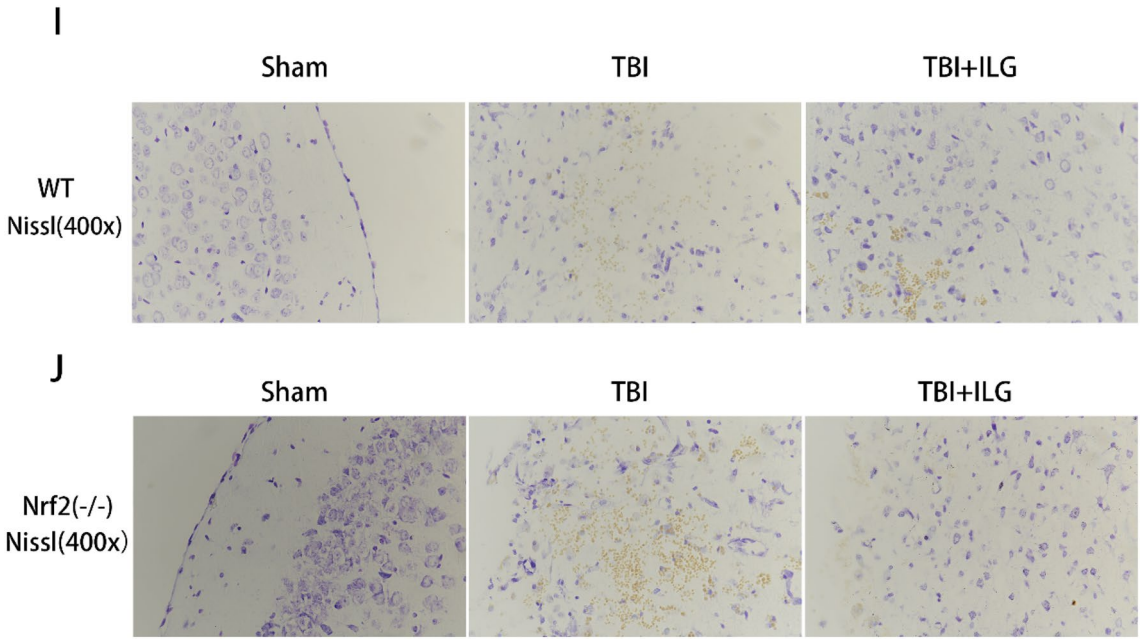

Fig. 1 ILG treatment protects against TBI-induced brain injury in mice. **a** Changes in Garcia neuroscore the different groups 24 h after TBI (n = 6 per group. ***$P < 0.001$ and *$P < 0.05$ when comparison was made in WT mice. #$P < 0.05$ when comparison was made between WT mice and Nrf2−/− mice). **b** Changes in brain water content in the different groups (n = 5 per group. **P < 0.01 and*P < 0.05 when comparison was made in WT mice. #$P < 0.05$ when comparison was made between WT mice and Nrf2−/− mice). **c, d** The protein expression of AQP4 after TBI; β-actin was used as the loading control and for band density normalization (n = 6 per group. **P < 0.05 vs sham group, *P < 0.05 vs TBI group). **e** Immunofluorescence staining for AQP4 (green) and DAPI (blue) in different groups after TBI (scale bar = 50 μm, n = 6 per group). **f–h** Evans blue (EB) leakage at 24 h after TBI (n = 6 per group. ***P < 0.001 and *P < 0.05 when comparison was made in WT mice. #$P < 0.05$ and ##$P < 0.01$, when comparison was made between WT mice and Nrf2−/− mice.). **i, j** Nissl staining results of the different groups after TBI (scale bar = 50 μm, n = 6 per group). **k–m** Expression of cleaved-caspase3 protein at 24 h after TBI (n = 6 per group. ***P < 0.001 and **P < 0.01 when comparison was made in WT mice. $^{\#}P < 0.05$ and $^{\#\#}P < 0.01$ when comparison was made between WT mice and Nrf2−/− mice). **n**, **o** Double staining for GFAP (Pink) and NFL (green) in brain from different groups 24 h after TBI (scale bar = 50 μm. n = 6 per group)