Correction

Correction for: Hypoxia-preconditioned olfactory mucosa mesenchymal stem cells abolish cerebral ischemia/reperfusion-induced pyroptosis and apoptotic death of microglial cells by activating HIF-1α

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This article has been corrected: The authors requested replacement of **Figure 6,** in which the images in **panel D** – production of ROS in BV2 microglial cells in the Normoxia group (the upper right subpanel) and Hypoxia+FG4592 group (the bottom left subpanel) – were incorrectly placed during assembly of the figures. This resulted in duplication of the image of ROS production in the Hypoxia+FG4592 group (P2=1.41%). The authors corrected **Figure 6D** by using the correct flow cytometry data/image from the original sets of experiments for the Normoxia group (P2=34.68%). This correction does not affect the article's conclusions. The authors would like to apologize for any inconvenience caused.

New **Figures 6** is presented below.

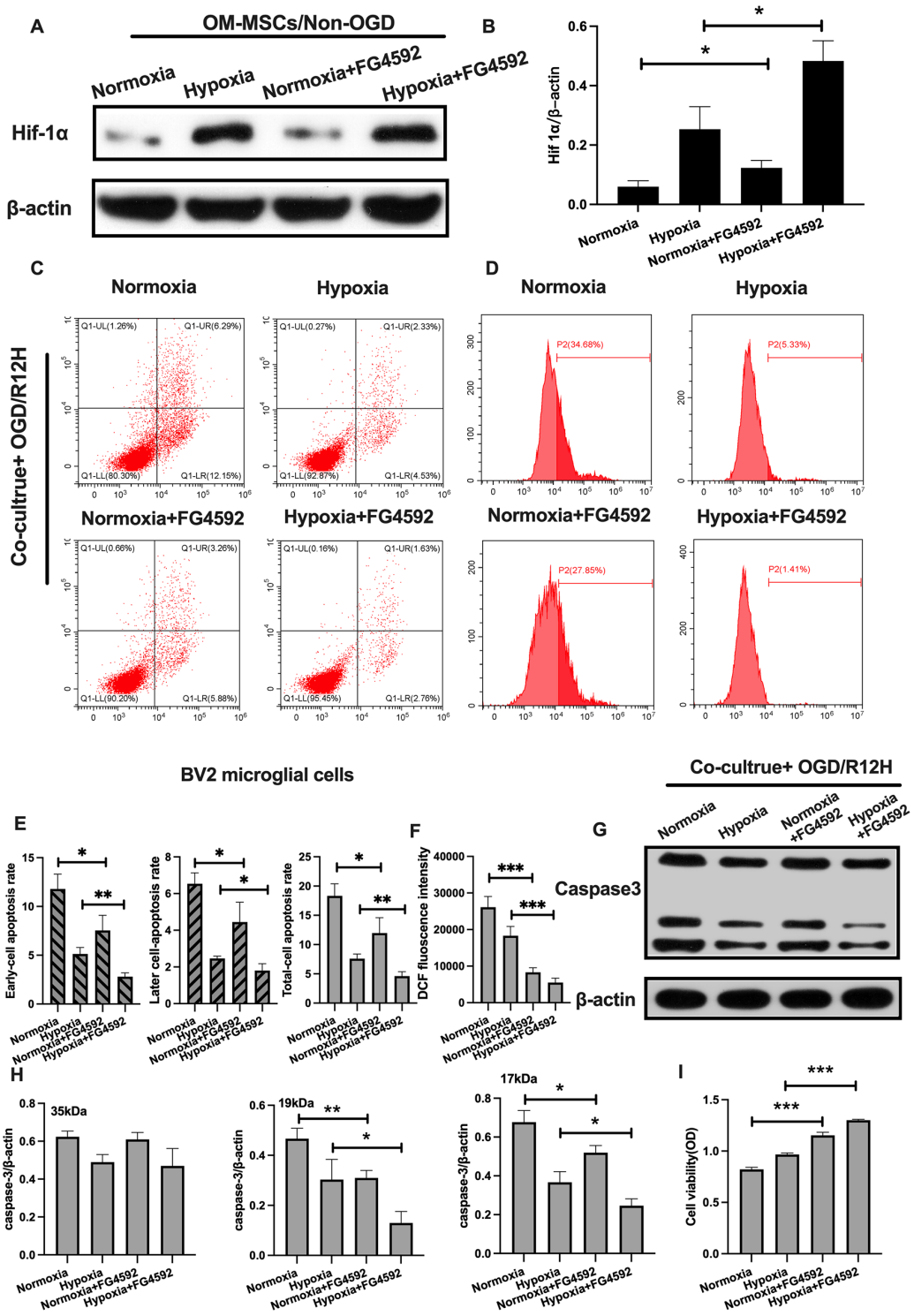

Figure 6. Induction of HIF-1α in OM-MSCs with FG-4592 inhibited cerebral OGD/R-induced apoptosis in BV2 microglial cells. (**A**, **B**) The successful overexpression of HIF-1α in OM-MSCs was confirmed by Western blotting. (**C**, **E**) The apoptosis rate among BV2 microglial cells was determined with flow cytometry and Annexin V/PI staining in each group. (**D**, **F**) Production of ROS levels in BV2 microglial cells was measured with flow cytometry. (**G**, **H**) Protein expression of caspase3 in BV2 microglial cells was quantified by Western blotting. (**I**) The viability of BV2 microglial cells was evaluated with MTT assays. All data are presented as the mean ± SD. *p<0.05; **p<0.01, ***p<0.001, compared to the normoxia or hypoxia group.