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Corrigendum: Synthetic torpor triggers a regulated mechanism in the rat brain, favoring the reversibility of Tau protein hyperphosphorylation

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A Corrigendum on

Synthetic torpor triggers a regulated mechanism in the rat brain, favoring the reversibility of Tau protein hyperphosphorylation

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In the published article, there was an error in **Figure 4E** as published. The only **histogram bars** represented in **Panel E left side** (referred to P-Cx [i.e., Parietal cortex]) are wrong, together with the relative “y” scale. However, the representative Western blot bands depicted at the bottom of the histograms are correct, as described in the original caption that is also reported below. The corrected **Figure 4** and its correct original caption appear below.

The authors apologize for these errors and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

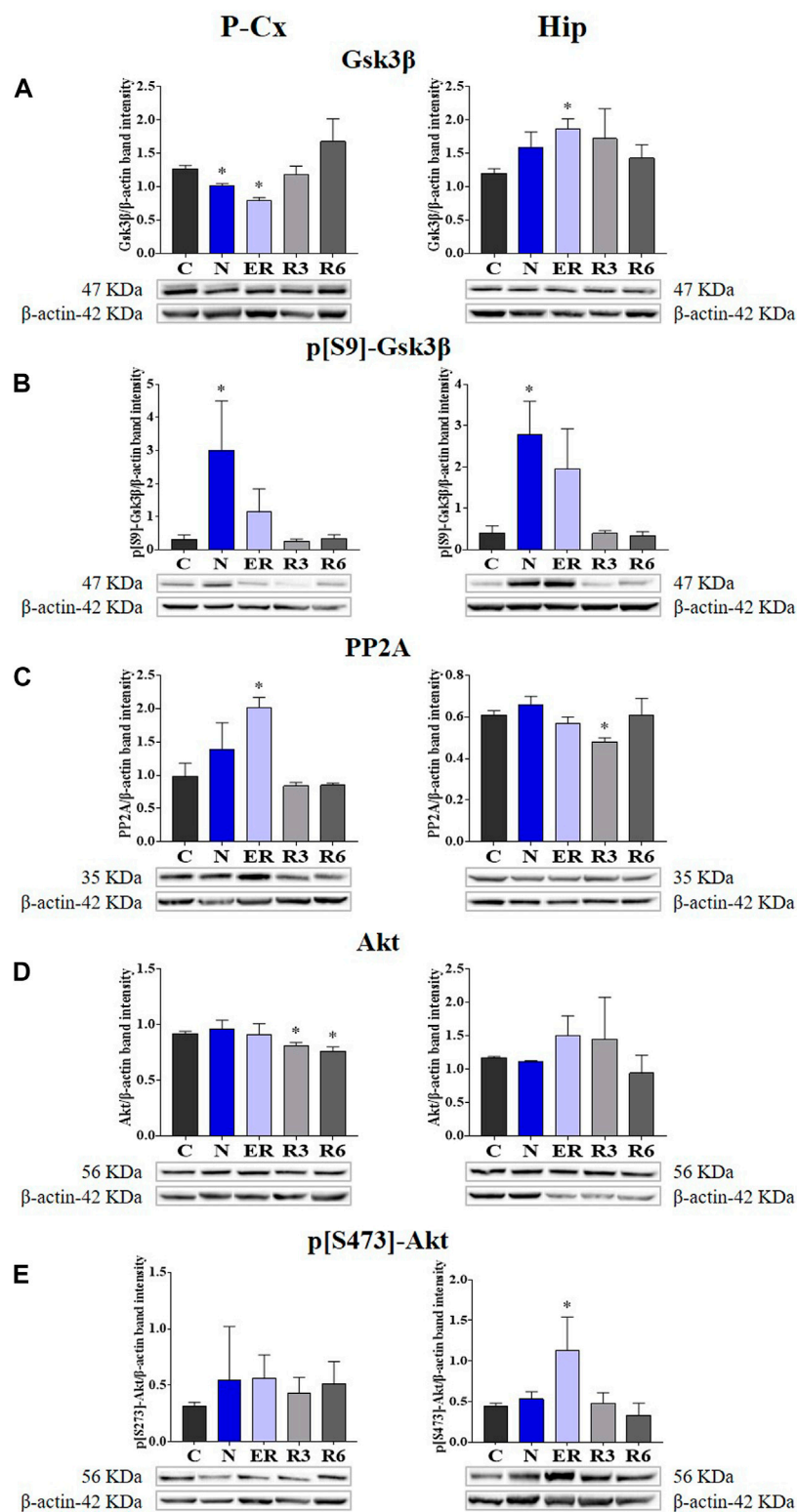


FIGURE 4

Western blot detection of the main enzymes involved in phosphorylation and dephosphorylation of Tau, determined in brain extracts of the parietal cortex (P-Cx) and hippocampus (Hip). Below each histogram, WB representative samples are shown for each experimental condition. (A) glycogen-synthase kinase-3 β (GSK3 β), the main kinase targeting Tau; (B) p[S9]-GSK3 β (inactive form of GSK3 β , phosphorylated at Ser9); (C) protein phosphatase-2A (PP2A), the main phosphatase targeting Tau; (D) different isoforms of Akt (protein kinase-B; Akt 1/2/3), kinases targeting GSK3 β at Ser9 and antiapoptotic factors; (E) p[S473] Akt, the active form of Akt 1/2/3, phosphorylated at Ser473. Data are normalized by β -actin and expressed as means \pm S.E.M., $n = 3$. *: $p < 0.05$ vs. C. Experimental groups (see Figure 1): C, control; N, samples taken at nadir of hypothermia, during ST; ER, early recovery, samples taken when Tb reached 35.5°C following ST; R3, samples taken 3 h after ER; R6, samples taken 6 h after ER.

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