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ERRATUM

Erratum to: Degradation of βII-Spectrin Protein by Calpain-2 and Caspase-3 Under Neurotoxic and Traumatic Brain Injury Conditions

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The original version of this article unfortunately contained mistakes on figs.

1) On reviewing our published paper recently, we identified that some figure images in Figs. 3, 4, 5, 6 inadvertently contain duplicate lanes due to cut and paste editing errors. We have now replaced them with corrected images from the same study. See Revised Figs. 3, 4, 5 and 6 below.

(2) We also noticed that line graph in Fig. 5b was accidentally used again in Fig. 6b. The correct line graph is now used in the revised Fig. 6b.

We want to state that these corrections do not by any means alter our overall findings, our interpretation of the data, or the conclusion we made from this study.

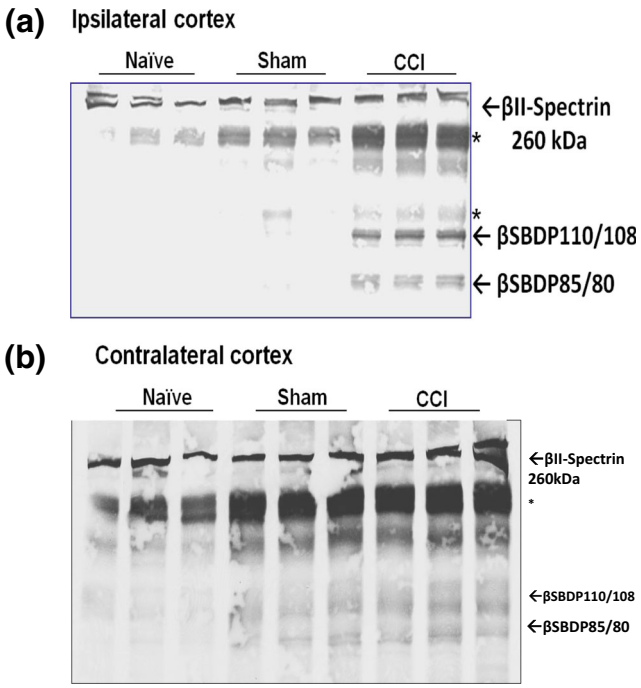

Fig. 3 βII-Spectrin BDP formation in rat cortex at 48 h after CCI. Ipsilateral cortex (**a**) and contralateral cortex (**b**) samples from naive, sham, and TBI groups were analyzed by Western blot for βsBDPs. The results showed that there was accumulation of the specific βsBDPs of either 110 and 85 kDa or 108 and 80 kDa, respectively. Minimal βsBDPs bands were observed in the ipsilateral cortex of the naive and sham groups. The contralateral hippocampal brain regions had no significant βSBDPs detected. *Asterisk* indicates nonspecific bands unrelated to βII-spectrin, which is also observed with post-CCI αII-spectrin blotting analysis previously by us (Liu et al. [86]). The results shown and BDP patterns are of high consistency and are representative of six independent experiments (n=6)

The online version of the original article can be found at https://doi.org/10.1007/s12035-014-8898-z

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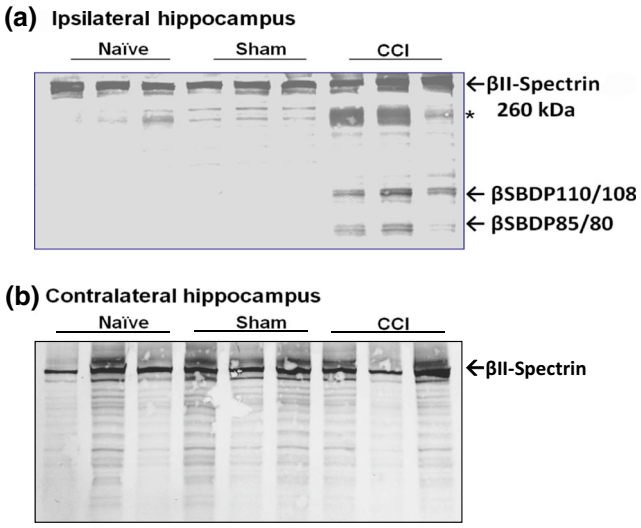

Fig. 4 βII-Spectrin BDP formation in rat hippocampus at 48 h after CCI. Ipsilateral hippocampus (**a**) and contralateral hippocampus (**b**) samples from naive, sham, and TBI groups were analyzed by Western blot for βII-spectrin BDPs. The results showed that there was accumulation of the specific βsBDPs of 110 and 85 kDa and of 108 and 80 kDa, respectively. Minimal βsBDPs bands were observed in the ipsilateral cortex of the naive and sham groups. The contralateral hippocampal brain regions had no significant βsBDPs detected. *Asterisk* indicates nonspecific bands unrelated to βII-spectrin, which is also observed with post-CCI αII-spectrin blotting analysis previously by us (Liu et al. [86]). The results shown and BDP patterns are of high consistency and are representative of six independent experiments (n=6)

The authors sincerely apologize for these errors.

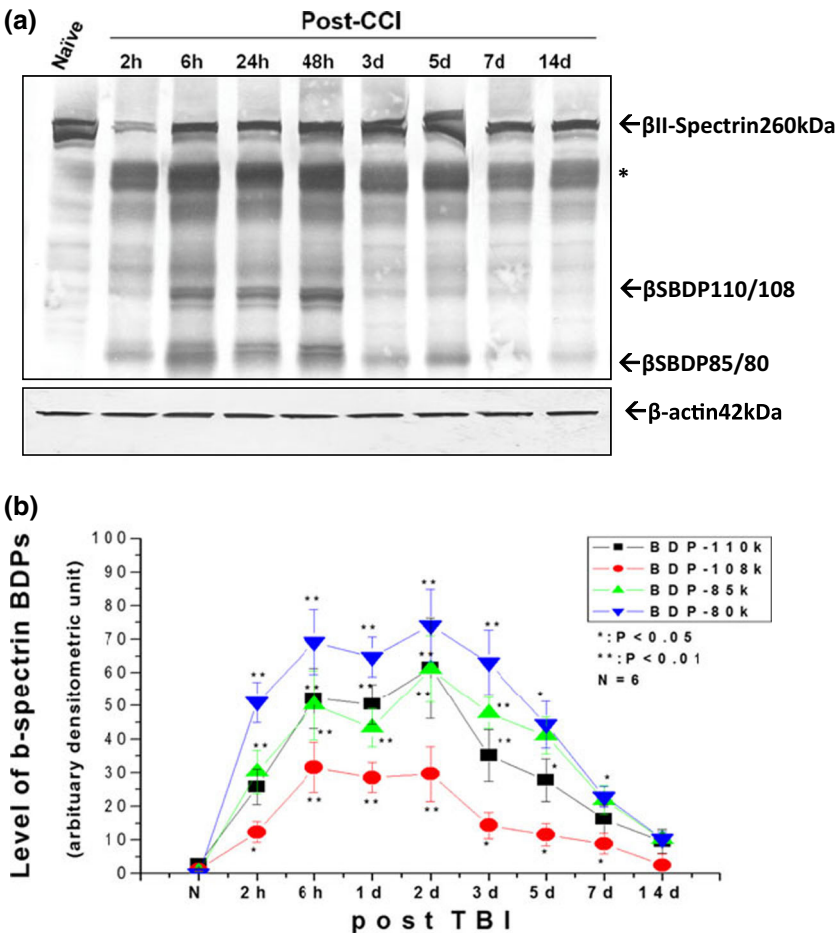

Fig. 5 Time course of TBI-induced βII-spectrin degradation and βSBDP quantification in rat cortex. **a** Western blot analysis of the temporal profile of TBI-induced βII-spectrin protein fragmentation (110, 108, 85, and 80 kDa) in the rat injured ipsilateral cortex of the TBI group. The fragments of 110, 108, 85, and 80 kDa were accumulated in rat ipsilateral cortex observed as early as 2 h after TBI and lasting up to 5 days and then gradually decrease followed by their resolution after 7–14 days. The 110/108 kDa βsBDPs were detected until day 5 compared to the 80/85 kDa βsBDPs which lasted until day 7. β-Actin Western blot as a loading control was performed to check for equal sample loading. **b** Densitometric representation of the temporal profile of the βsBDPs in the TBI group which was compared to those in naive. Statistical significance compared to naive levels were indicated (*p<0.05; **p<0.01)

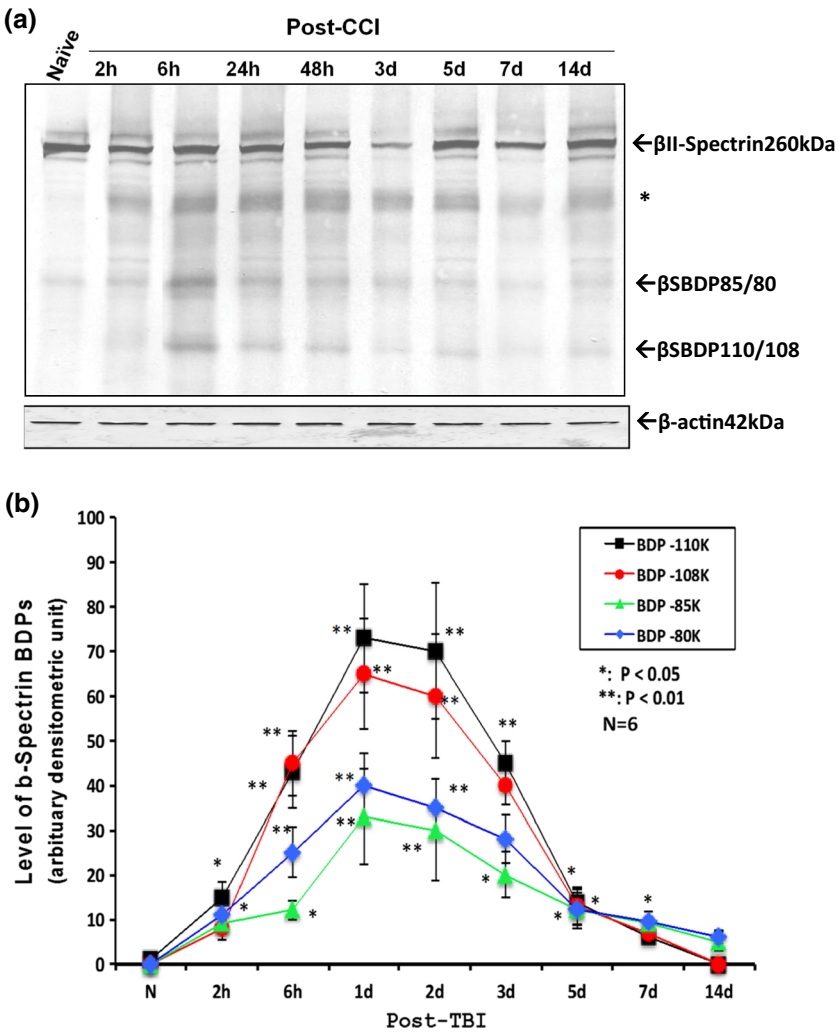

Fig. 6 Time course of TBI-induced βII-spectrin degradation and βSBDPs quantification in rat hippocampus. Western blot analysis of the temporal profile of TBI-induced βII-spectrin protein fragmentation (110, 108, 85, and 80 kDa) in the rat ipsilateral hippocampus of the TBI group (**a**). Similar to the cortical ipsilateral brain region, the hippocampus showed the fragments of 110, 108, 85, and 80 kDa which were detected at 2 h after TBI, lasting up to 3–5 days and then gradually decrease, followed by their resolution after7–14 days. The 110/108 kDa βsBDPs were detected until day 3 compared to the 80/85 kDa βsBDPs which lasted until day 5. β-Actin Western blot, as a loading control, was performed to check for equal sample loading. Densitometric representation ofthe temporal profile of the βsBDPs in the TBI group (**b**). This was compared to those innaive. Statistical significance compared to naive levels were indicated (*p<0.05; **p<0.01)