

Corrigendum

# GADD34 induces cell death through inactivation of Akt following traumatic brain injury

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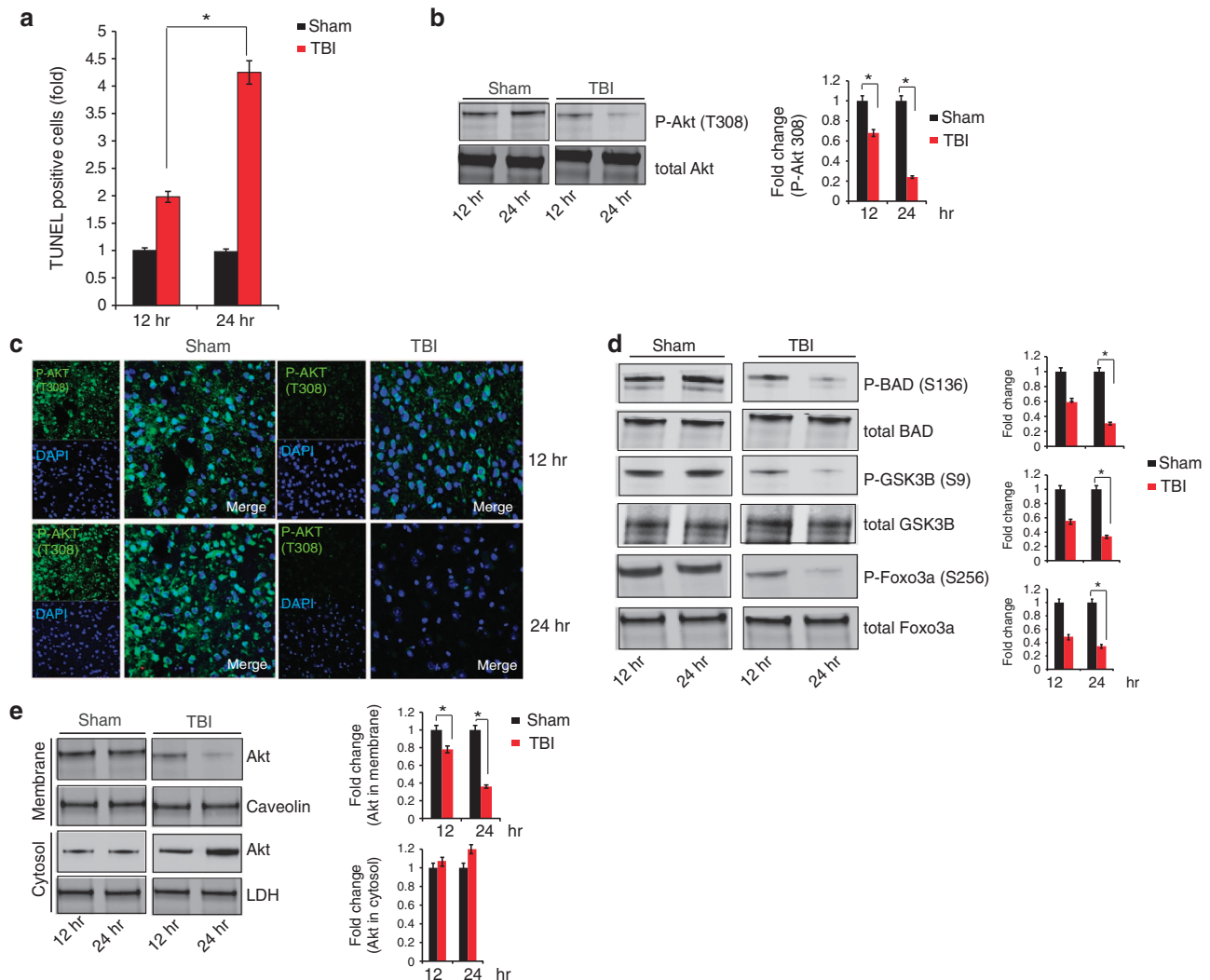
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**Correction to:** Cell Death and Disease (2013) 4, e754; doi:10.1038/cddis.2013.280; published online 1 August 2013

for TBI at 24 h was incorrect. The error has now been rectified. The corrected article appears online together with this corrigendum.

Since the publication of this paper, the authors have noticed that there was an error in Figure 1c. The confocal image

The authors would like to apologize for any inconvenience this may have caused.



**Figure 1** Inactivation of Akt is associated with cell death following TBI. (a) TUNEL staining was done to identify cell death at 12 and 24 h after TBI. Quantitative analysis shows that TUNEL staining was increased more than twofold after 24 h post TBI in the pericontusional cortex. (b and c) Phosphorylation of Akt (T308) was determined by western blot and immunofluorescent microscopy. Changes in phosphorylation status of Akt (P-Akt T308) was measured quantitatively. (d) Phosphorylation of downstream proteins of Akt, such as GSK3B, Foxo3a and BAD was determined by western blot analysis 12 and 24 h post TBI. (e) Membrane and cytosolic fraction of Akt was determined in the cortex at 12 and 24 h post Sham or TBI in mice. Level of both cytosolic and membrane Akt was determined at 12 and 24 h post TBI quantitatively. \* $P < 0.01$ ,  $n = 3$ , one-way ANOVA, mean  $\pm$  S.E.M.