## CORRECTION

Gankam-Kengne F, Couturier BS, Soupart A, Brion JP, Decaux G: Osmotic Stress–Induced Defective Glial Proteostasis Contributes to Brain Demyelination after Hyponatremia Treatment. *J Am Soc Nephrol* 28: 1802–1813, 2017.

This article contained errors in Western blot images in two of the figures. In Figure 3C, the blot shown for pEIF2a was a replicate of the blot for BIP shown in Figure 3A and not the intended image. In Figure 6A, an incorrect blot was shown for Bax, which did not match the $\beta$-actin ($\beta$-Act) blot shown beneath it. The corrected versions of these figures are presented here. The errors do not alter the conclusions of the study.

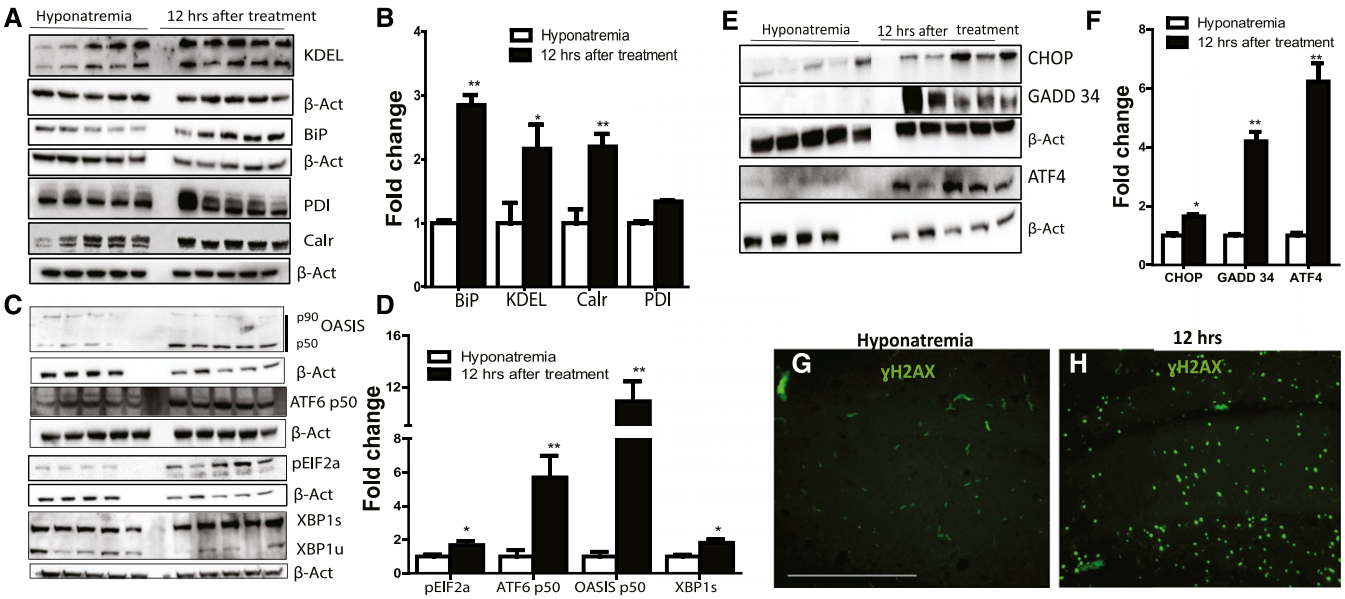


**Figure 3.** UPR and ER stress are activated during rapid correction of chronic hyponatremia. A and B are (A) representative images and (B) semiquantitative densitometric analysis of Western blot for ER stress and ERAD-related proteins from brain extract of animals in chronic hyponatremia and 12 hours after the correction of chronic hyponatremia. As shown by B, protein levels of KDEL (GRP94 and GRP78), BiP (GRP78), and calreticulin (Calr) significantly increased 12 hours after the correction of chronic hyponatremia. $P$=0.20 for PDI in B. C and D show (C) images of Western blot and (D) densitometric quantification for OASIS, ATF6, peIF2$\alpha$, XBP1 spliced (XBP1s), and unspliced (XBP1u) in the brains of chronic hyponatremic control animals compared with chronic hyponatremic animals treated with hypertonic saline 12 hours after the beginning of the treatment. E shows representative images of Western blot of brain homogenates for ER stress effectors of cell death (CHOP, GADD34, and ATF4) in uncorrected hyponatremia controls and 12 hours after rapid correction of chronic hyponatremia. Quantification of protein expression is depicted in F, showing significant increase in the expressions of CHOP, GADD34, and ATF4 on correction of chronic hyponatremia. *$P$<0.05 for CHOP; **$P$<0.01 for GADD34 and ATF4. G shows an image of the hippocampus of an uncorrected hyponatremia control animal stained with DNA damage marker $\gamma$H2AX in green; only background and intravascular staining is present, in contrast to D, which shows strong cellular staining in the hippocampus of an animal 12 hours after treatment with hypertonic saline was initiated. $\beta$-actin ($\beta$-Act) was used as loading control. GADD34, growth arrest and DNA damage protein 34; XBP1, X-box-binding protein. *$P$<0.05 by unpaired $t$ test ($n$=4–5 in each group); **$P$<0.01 by unpaired $t$ test ($n$=4–5 in each group). Scale bar, 200 $\mu$m.

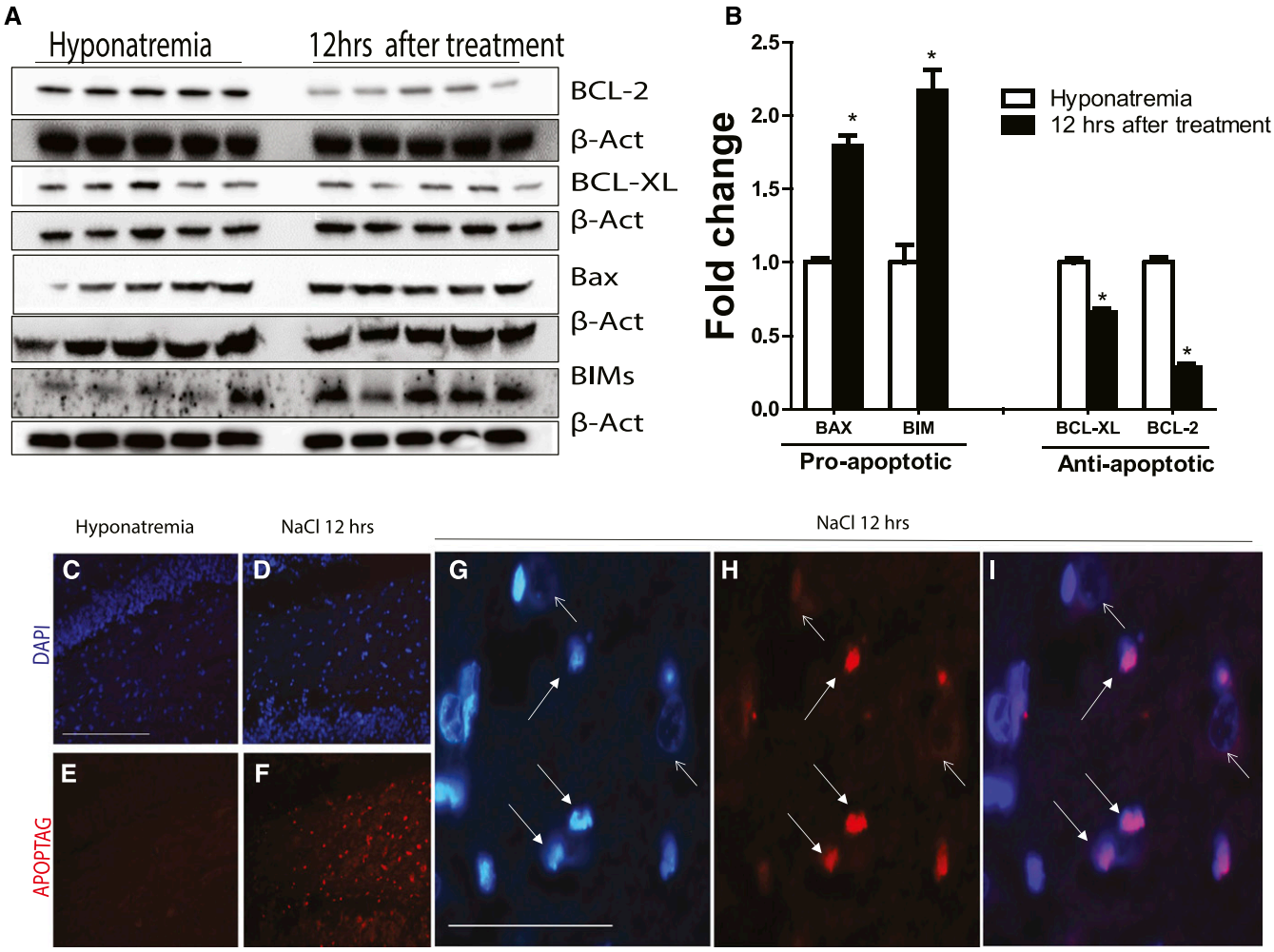


**Figure 6.** Rapid correction of chronic hyponatremia induces apoptosis. In A, Western blot images of pro- and antiapoptotic proteins before and 12 hours after the correction of chronic hyponatremia show that there is a significant increase in proapoptotic proteins (BAX and BIM) in treated animals along with a decrease in antiapoptotic proteins BCL-XL and BCL-2 (quantification shown in B; $n=4–5$ in each group of animals). *$P<0.05$ by unpaired *t* test. In C and F, *in situ* oligonucleotide ligation assay (Apoptag) staining (which marks apoptotic nuclei; red) and nuclear counterstaining (blue) were performed in the hippocampus of control animals, showing no nuclear condensation and no apoptotic nuclei (no positive staining for Apoptag). In contrast, as shown in E and G, positive Apoptag staining showing cells undergoing apoptosis is seen 12 hours after correction of chronic hyponatremia. In H, higher-magnification images show typical nuclear condensation (small white arrow) seen in apoptotic cells (nonapoptotic nuclei show dispersed chromatin [larger white arrow]). (I) Apoptag staining and (J) merged images confirm that cells with nuclear condensation also stain positive for the Apoptag marker. Scale bar, 200 $\mu$m in C–G; 50 $\mu$m in H–J.