Correction: Elevated p62/SQSTM1 determines the fate of autophagy-deficient neural stem cells by increasing superoxide

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After publication, the authors discovered an error in Fig. 8 J. In the published version, arrowheads were inadvertently left out of the image. The authors apologize for this error. The corrected image is included below.

The HTML and PDF versions of this article have been corrected. The error remains only in the print version.
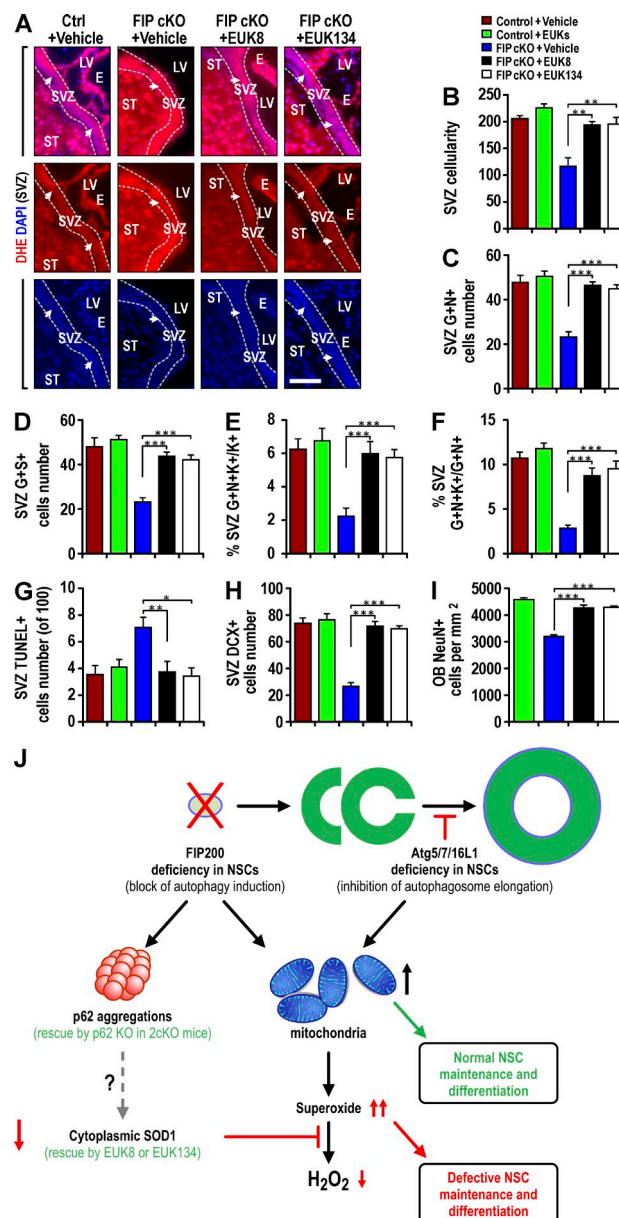
Figure 8. **SOD mimetics EUK-8 and EUK-134 restore normal $O_2^{\bullet-}$ levels and rescue defective NSCs in *Fip200*[GFAP] cKO mice.** (A–I) Ctrl and *Fip200*[GFAP] cKO mice at P28 were treated with vehicle, EUK-8, or EUK-134. (A) DHE and DAPI fluorescence in SVZ. Arrows indicate SVZ cells. Dotted lines indicate the boundaries of the SVZ (*n* = 4 mice each). (B) Mean ± SEM of SVZ cellularity per section (*n* = 3 mice each). (C and D) Mean ± SEM of GFAP+Nestin+ (C) and GFAP+SOX2+ (D) cells per section (*n* = 4 mice each). (E and F) Mean ± SEM of the percentage of GFAP+Nestin+Ki67+ to total Ki67+ cells (E) or total GFAP+Nestin+ cells (F) in SVZ (*n* = 4 mice each). (G–I) Mean ± SEM of TUNEL+ (G) or DCX+ (H) cells per SVZ section and NeuN+ cells in the OB per square millimeter (I) of mice (*n* = 4 mice each). E, ependymal layer; LV, lateral ventricle; ST, striatum. Bars, 50 µm. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$. (J) A working model of differential p62 aggregate formation and $O_2^{\bullet-}$ accumulation by deletion of *Fip200*, but not *Atg5*, *Atg16L1*, or *Atg7*, leading to defective neural stem cell maintenance and differentiation.