AUTOPHAGY
2023, VOL. 19, NO. 10, 2832–2833
https://doi.org/10.1080/15548627.2023.2198815

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Correction

Article title: USP14 regulates DNA damage repair by targeting RNF168-dependent ubiquitination
Authors: Sharma A., Alswillah T., Singh K., Chatterjee P., Willard B., Venere M., Summers M K., Almasan A.
Journal: *Autophagy*
Bibliometrics: Volume 14, Issue 11, Pages 1976-1990
DOI: 10.1080/15548627.2018.1496877

In the original version of this article, in Fig 2g one inadvertent duplication was generated during the assembly of our manuscript for the Western blot analysis for USP14 in sh*Ctrl*-expressing vs sh*ATG7*-expressing C4-2 cells following IR treatment for the indicated time for ACTB/β-actin that was used as a loading control, that was a duplicate of the actin blot in Fig 6f.

This error has now been corrected as shown below, and this correction has not changed the description, interpretation or the original conclusions of the manuscript. The authors apologize for any inconvenience caused.

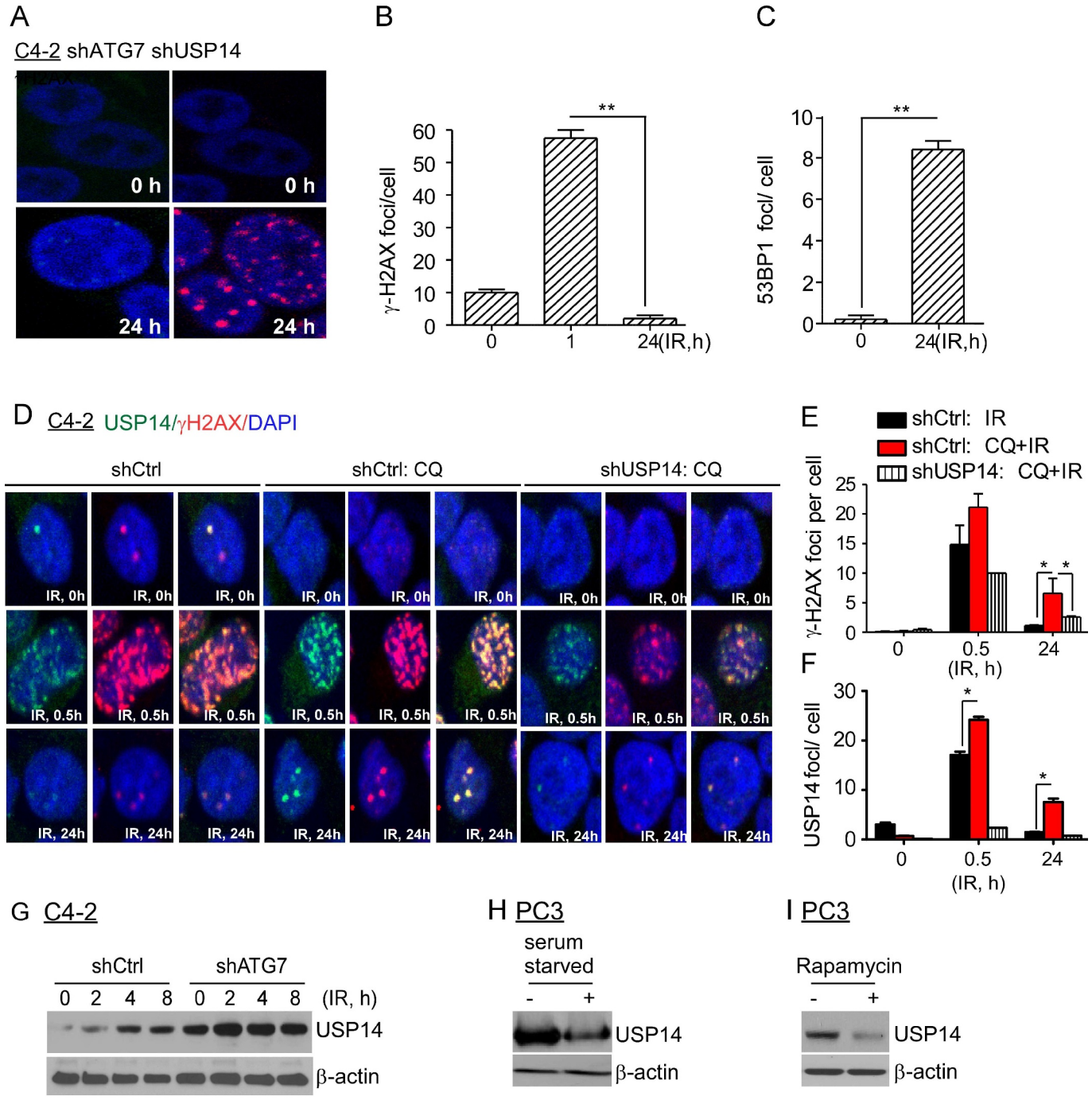

Figure 2. USP14 disrupts DDR signaling in autophagy-deficient cells. (a–c) Confocal immunostaining and graphical representation of γH2AFX and TP53BP1 foci following IR treatment in C4-2 cells co-expressing sh*ATG7* and sh*USP14*. Nuclei were stained with DAPI. (d–f) Confocal immunostaining and graphical representation of γH2AFX and USP14 foci following IR+/− CQ treatment in C4-2 cells expressing sh*Ctrl* and sh*USP14*. Nuclei were stained with DAPI. Data shown are the means ± SEM (n = 2); P < 0.05 *, P < 0.01 **. (g) Western blot analysis for USP14 in sh*Ctrl*-expressing vs sh*ATG7*-expressing C4-2 cells following IR treatment for the indicated time. ACTB/β-actin was used as a loading control. Western blot analysis for USP14 in PC3 cells treated with (h) serum starvation and (i) rapamycin. ACTB/β-actin was used as a loading control.