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Corrigendum

Sulfatase modifying factor 1 trafficking through the cells: from endoplasmic reticulum to the endoplasmic reticulum

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Authors' statement

Following a note from the authors to *EMBO Journal* and subsequent investigation by the Journal, the following errors are reported:

Figure 1D: We were made aware that the band marked as WT may have been spliced into this panel; however, this cannot be confirmed or disproved due to the absence of source data and the low resolution of the figure. This experiment confirms the results of experiments in Figs 1B and C, and 4C and D and F, and 5. Furthermore, the uptake of SUMF1 protein was also confirmed by mass spectrometry analysis (Supplementary Figure 1). This control was also intended to provide a reference for antibody recognition, which is also provided by the Western blot of protein extracts and media harvested from HepG2 cells (Fig 1D upper panels). Therefore, the possible splice does not affect the main conclusions of the paper.

Figure 4C (top panel): We were made aware of the overcontrasting of the Western blot result (4C; top panel). Considering other pieces of evidence in this paper (Fig 4A), our own additional results and the results from others (Dierks *et al,* 2003, Landgrebe *et al,* 2003, Preusser-Kunze *et al*, 2005), where it was demonstrated that N141 is the only residue where SUMF1 is glycosylated, our findings have been validated in other contexts and the possible overcontrast of the Western blot data does not alter the conclusion of the paper.

Figure 4E: Fig 4E includes the analysis of Xenopus SUMF1 localization into the ER of HeLa cells, which appeared to be similar to the localization observed for SUMF1N141A (Fig 4B). We have come to realize that two areas of the immunofluorescence picture in the anti-ERAB and merged panels of Fig 4E were blocked out. Although we cannot discern the data underlying the blocked areas because source data are not available, the correct localization of Xenopus SUMF1 in the ER was observed in other experiments (please see https://figshare.com/articles/xSumf1_localization_pdf/4244378) and confirmed by the observation that Xenopus SUMF1 was enzymatically active after expression in HeLa cells and it was correctly secreted but not taken up (Fig 4F–G). Overall, the results from the studies regarding Xenopus SUMF1 recapitulated what was observed for human SUMF1N141A (Fig 4B–D), and therefore, the conclusion of the paper stands. Nonetheless, we withdraw this figure panel, as it cannot be regarded as reliable in the absence of source data.

Figure 6B (upper panels): We were made aware of possible band insertions in the two right-hand lanes of the SUMF1 panel as well as a possible duplication of the two right-hand lanes of the beta-tubulin panel. Since source data are not available, we cannot confirm or disprove these aberrations; however, the general conclusion of this experiment stands, because it is confirmed by the data presented in Fig 6A and B, bottom panels. Specifically, 6B includes experiments showing that the uptake of SUMF1 is mediated by the Mannose receptor in MEFs. This result is confirmed by the competition uptake experiments performed in human cells (Fig 6A) and in $MPR^{-/-}$MEFs (6B bottom panels) where Mannan was used to inhibit the MR. We withdraw Fig 6B upper panels, as it cannot be regarded as reliable in the absence of source data.

Supplementary figure legend SI2A: While for most proteins, the samples corresponding to the medium and cellular pellets were run in the same gels; for SUMF2, the media and cell pellets were loaded in different gels, the Western blots were processed in parallel, and the two sets of lanes were collated together. This was not properly described in the Supplementary figure legend 2 which in the 5th line should read "The protein extracts and media were loaded in different gels and the filters were decorated with different antibodies: anti-SUMF2, anti-PDI, anti-ERK and anti-beta-tubulin."

All authors concur with this statement and wish to apologize for the inconvenience caused.

Editors' statement

In light of the potential image aberrations noted in the author's statement, and since source data were not available for the figure panels in question, Figs 4E and 6B, upper panels, are herewith withdrawn.

We alert readers to the fact that a related paper in *EMBO reports* is also subject to a correction:

Zito E, Fraldi A, Pepe S, Annunziata I, Kobinger G, Di Natale P, Ballabio A, Cosma MP (2005) Sulphatase activities are regulated by

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the interaction of sulphatase-modifying factor 1 with SUMF2. *EMBO Rep* 2005 6: 655–660.

References

Dierks T, Schmidt B, Borissenko LV, Peng J, Preusser A, Mariappan M, von Figura K (2003) Multiple sulfatase deficiency is caused by mutations in the gene encoding the human C(alpha)-formylglycine generating enzyme. *Cell* 113: 435 – 444

Landgrebe J, Dierks T, Schmidt B, von Figura K (2003) The human SUMF1 gene, required for posttranslational sulfatase modification, defines a new gene family which is conserved from pro- to eukaryotes. *Gene* 316: 47 – 56

Preusser-Kunze A, Mariappan M, Schmidt B, Gande SL, Mutenda K, Wenzel D, von Figura K, Dierks T (2005) Molecular characterization of the human Calpha-formylglycine-generating enzyme. *J Biol Chem* 280: 14900 – 14910

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