Molecular Neurodegeneration

CORRECTION

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Correction: Blood–brain barrier-associated pericytes internalize and clear aggregated amyloid-β42 by LRP1-dependent apolipoprotein E isoform-specific mechanism

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After publication of the first correction [1] to the original manuscript [2] regarding Fig. 4b, errors were noticed in the corrected Fig. 4B representative images for anti-LRP1 and RAP conditions:

- In the merged column, representative images with similar pattern were noticed in anti-LRP1 and si.*Lrp1* conditions, and in the Cy3-Aβ42 column, representative images with similar pattern were noticed in si.*Lrp1* and RAP conditions.
- The anti-LRP1 merged image, an incorrect cell tracker image was used for the merged overlay image. The merged image for anti-LRP1 has been corrected using images from the anti-LRP1 Cell tracker and Cy3-Aβ42 channels as originally presented in Fig. 4B.
- The RAP Cy3-Aβ42 image is incorrect and was also incorrectly used for the RAP merged image. The authors have identified the correct RAP Cy3-Aβ42 image and replaced both the Cy3-Aβ42 and merged RAP images.

The single channel si.*Lrp1* Cy3-Aβ42 image and si.*Lrp1* merged image are both correct, and no change is needed. Importantly, these errors only pertain to the incorrect representative images in Fig. 4B and have no impact on the analysis or conclusions presented in the paper.

The corrected version of the entire Fig. 4 is shown ahead, and the authors apologize for these unintentional errors.

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The original articles can be found online at https://doi.org/10.1186/s13024-018-0286-0 and https://doi.org/10.1186/s13024-022-00573-5.

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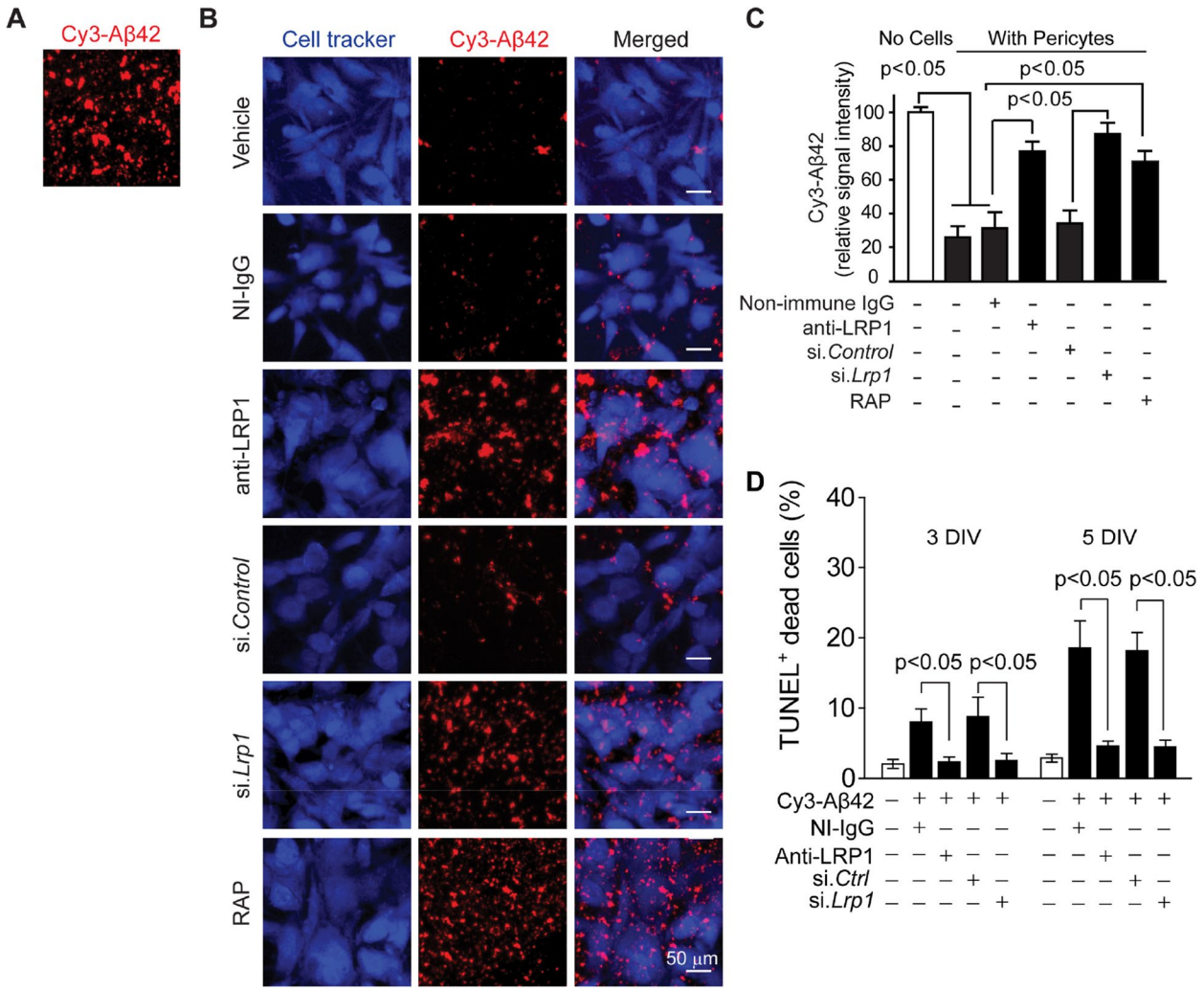

Fig. 4 LRP1 mediates clearance of aggregated Cy3-Aβ42 by mouse pericytes. **a**-**b** Multiphoton/confocal laser scanning microscopy of multi-spot glass slides coated with Cy3-Aβ42 without cells (**a**), and with primary mouse brain pericytes cultured for 5 days in the presence of NI-IgG or anti-LRP1, after si.*Lrp1* silencing compared to scrambled si.*Control*, and with RAP or vehicle (**b**). Scale bar, 50 µm. **c** Quantification of Cy3-Aβ42 relative signal intensityon multi-spot slides after 5 days without cells (open bar on the left) and with pericytes in the presence of vehicle (control), NI-IgG and anti-LRP1, after silencing with scrambled si.*Control* or si.*Lrp1*, and in the presence of RAP. $N = 4$ independent cultures (biological replicates, see Methods); mean ± s.e.m.; $p < 0.05$ by One-way ANOVA followed by Bonferroni post-hoc test. d Quantification of TUNEL + pericyte cell death at 3 and 7 days after seeding on multi-spot glass slides coated with Cy3-Aβ42 in the presence and absence of NI-IgG and anti-LRP1, and after si.*Lrp1* silencing or si.*Ctrl* as in (**b**). $N = 3$ independent cultures per group; mean ± s.e.m.; $p < 0.05$ by One-way ANOVA followed by Bonferroni post-hoc test

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References

1. Ma Q, Zhao Z, Sagare AP, et al. Correction: Blood-brain barrier-associated pericytes internalize and clear aggregated amyloid-β42 by LRP1-dependent apolipoprotein E isoform-specific mechanism. Mol Neurodegener. 2022;17:71. https://doi.org/10.1186/s13024-022-00573-5.
2. Ma Q, Zhao Z, Sagare AP, et al. Blood-brain barrier-associated pericytes internalize and clear aggregated amyloid-β42 by LRP1-dependent apolipoprotein E isoform-specific mechanism. Mol Neurodegener. 2018;13:57. https://doi.org/10.1186/s13024-018-0286-0.