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Publisher Correction: Exploring the significance of caspase-cleaved tau in tauopathies and as a complementary pathology to phospho-tau in Alzheimer's disease: implications for biomarker development and therapeutic targeting

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Publisher Correction to: ***acta neuropathol commun*** **12, 36 (2024)**
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Following the publication of the original article [1], it was noted that due to a typesetting error the figure legends were paired incorrectly. The figure legends for Figs. 1 and 2 were wrongly given as captions for Fig. 2, 1 respectively.

The publisher apologizes for the inconvenience caused.

The correct figures and captions have been included in this correction, and the original article [1] has been corrected.

The online version of the original article can be found at https://doi.org/10.1186/s40478-024-01744-9.

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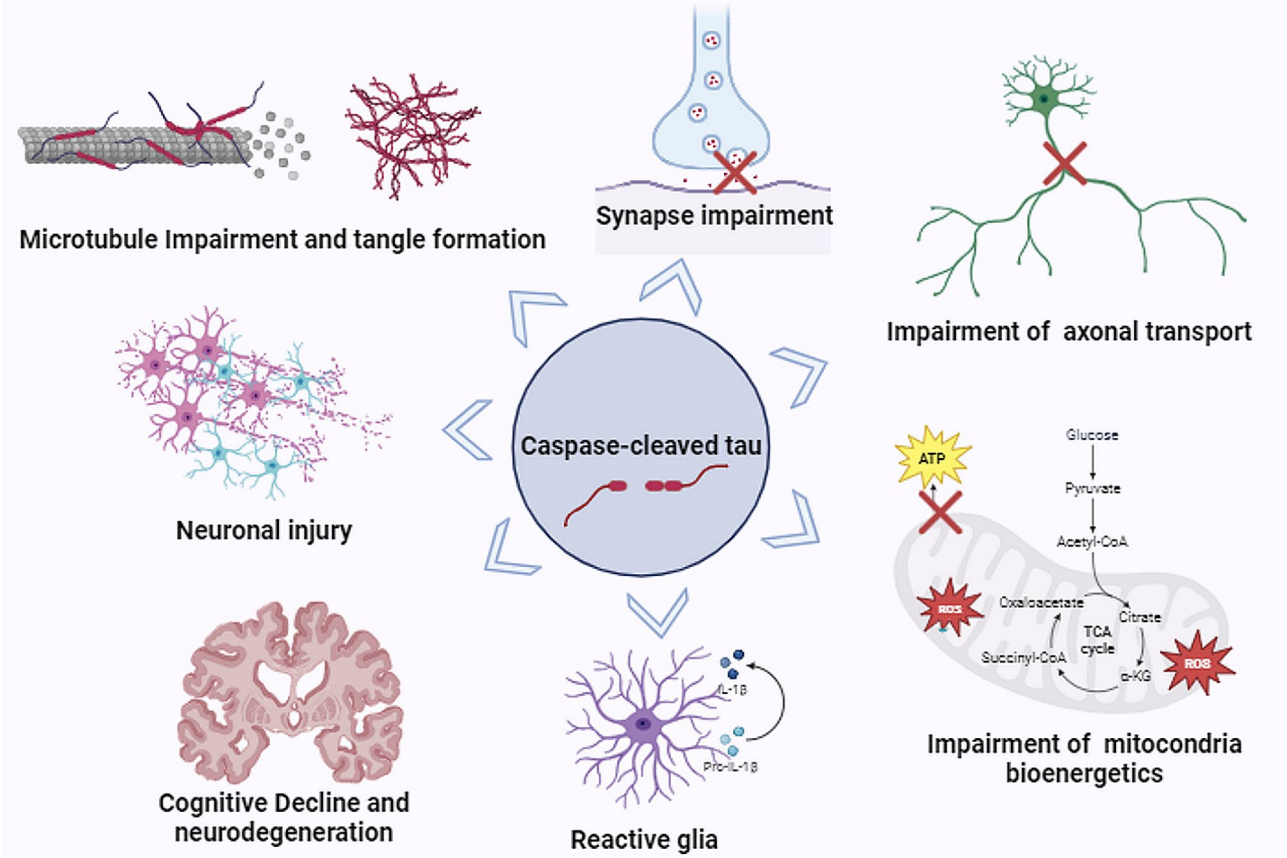

Fig. 1 Pathological mechanisms induced by caspase-cleaved tau

Fig. 2 Putative sites caspase-cleaved tau. Caspases 1, 3, 6, 7, and 8 cleave tau at D421. Caspase-2 cleaves tau also at D65 and D314, caspase-3 cleaves tau also at D25, caspase-6 cleaves tau also at D402 and D13. Tau consists of four domains: the projection domain (M1–Y197), a proline-rich region (P1 and P2), the microtubule-binding repeats (R1, R2, R3, R4), and a C-terminus domain (K369–L441). Amino acids 1-441

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References

1. Rizzi L, Grinberg LT (2024) Exploring the significance of caspase-cleaved tau in tauopathies and as a complementary pathology to phospho-tau in Alzheimer's disease: implications for biomarker development and therapeutic targeting. acta Neuropathol Commun 12:36. https://doi.org/10.1186/s40478-024-01744-9

Publisher's Note