


CORRECTION

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# Correction: CD2AP deficiency aggravates Alzheimer's disease phenotypes and pathology through p38 MAPK activation

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**Correction:** *Translational Neurodegeneration* (2024) 13:64

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incorrect lane image of p-tau 396. The Fig. 3 is corrected from:

Following publication of the original article [1], the authors reported an error in the Fig. 3h, which presented

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The original article can be found online at <https://doi.org/10.1186/s40035-024-00454-5>.

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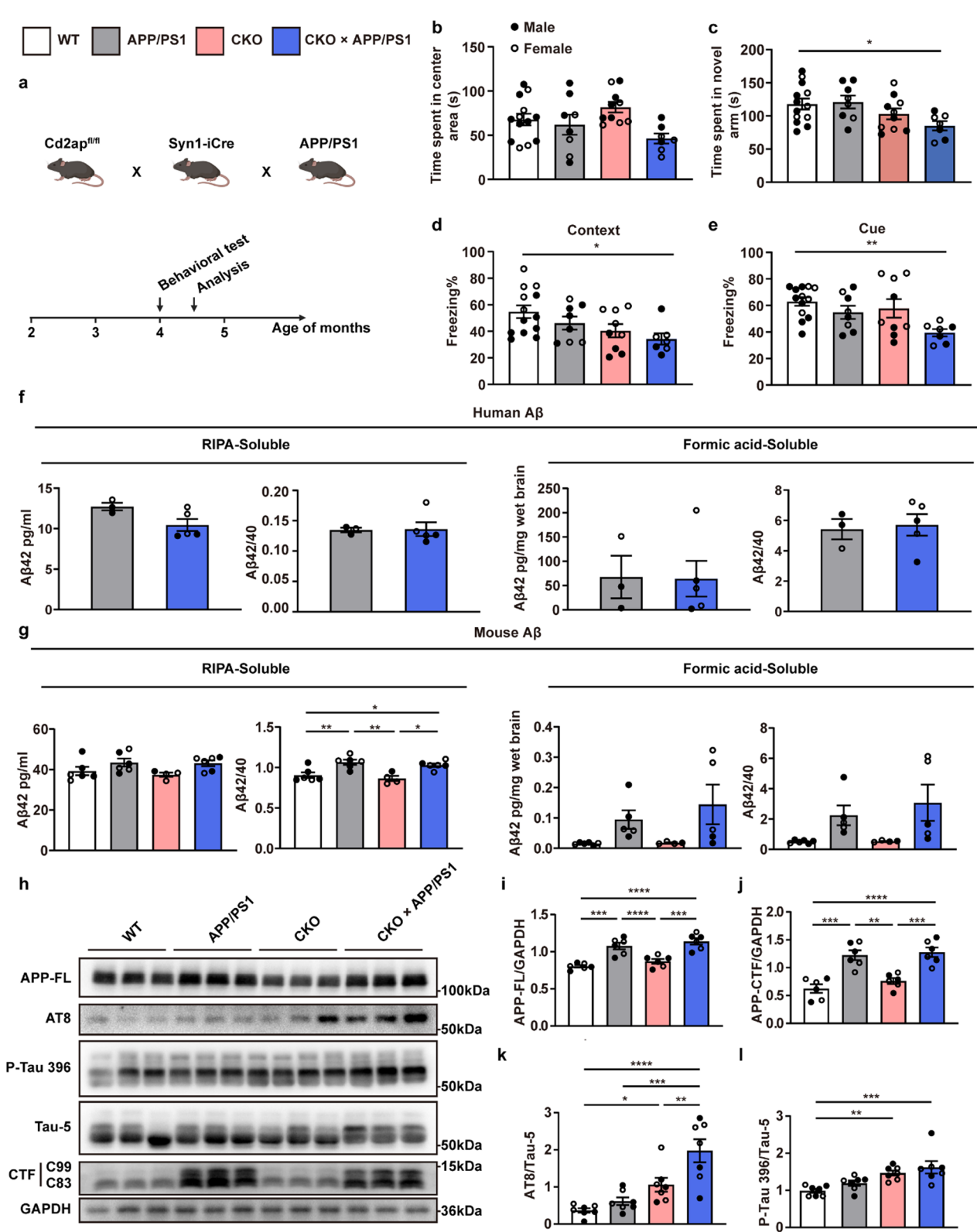
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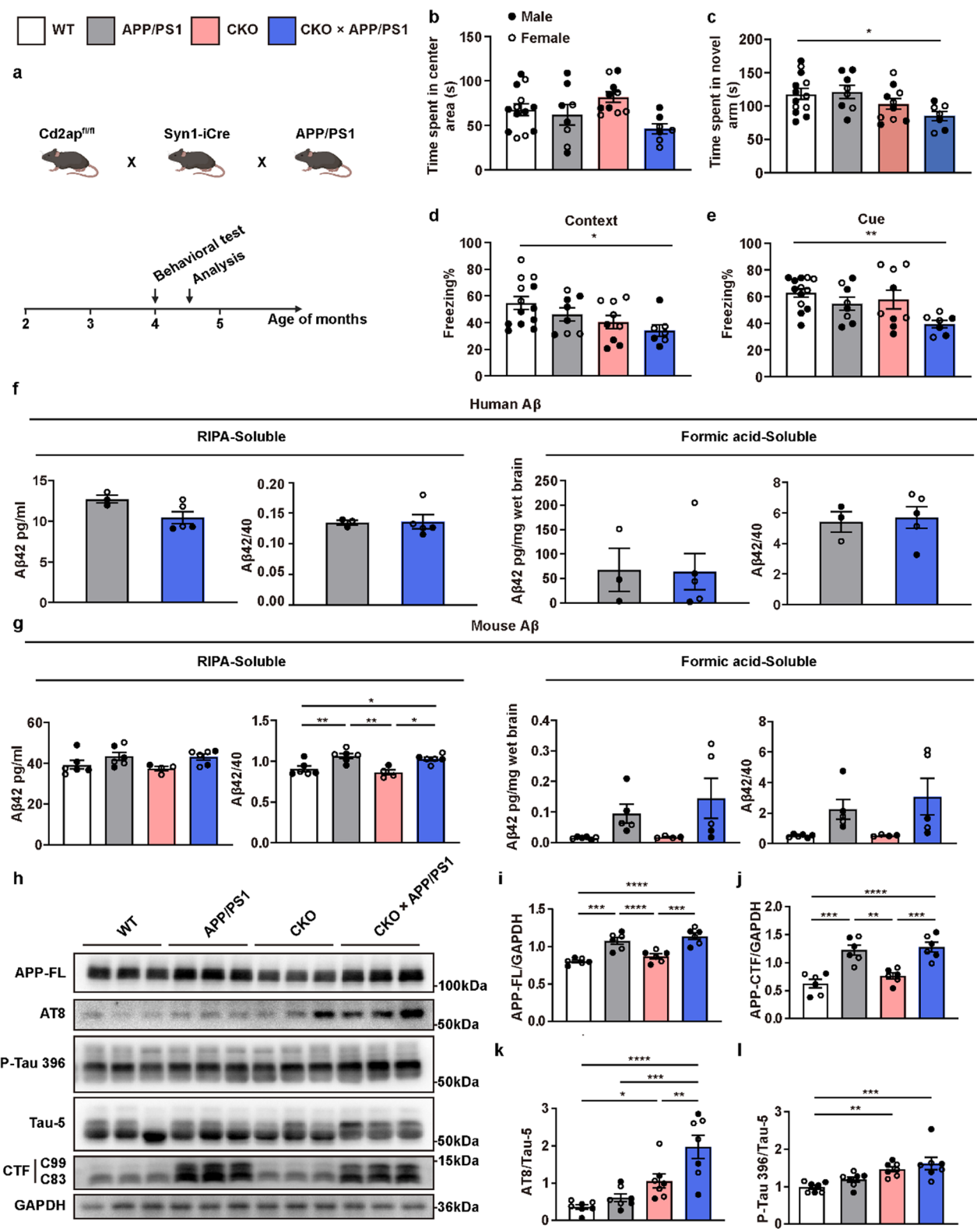
(See figure on next page.)

**Fig. 3** Neuronal *Cd2ap* deletion aggravated cognitive function and pathological features in APP/PS1 mice. **a** Scheme of the experimental mouse timeline. Briefly, a series of behavioral tests were performed in 4-month-old mice, and subsequent pathological analyses were conducted in 4.5-month-old mice. **b** No significant difference in time spent in the center area was observed in the open field test.  $n = 13$  (WT, female  $n = 5$ , male  $n = 8$ ),  $n = 8$  (APP/PS1, female  $n = 3$ , male  $n = 5$ ),  $n = 10$  (CKO, female  $n = 5$ , male  $n = 5$ ),  $n = 7$  (CKO  $\times$  APP/PS1, female  $n = 4$ , male  $n = 3$ ). **c** CKO  $\times$  APP/PS1 mice spent less time in the novel arm in the Y-maze novel arm preference test compared to WT mice.  $n = 13$  (WT, female  $n = 5$ , male  $n = 8$ ),  $n = 8$  (APP/PS1, female  $n = 3$ , male  $n = 5$ ),  $n = 10$  (CKO, female  $n = 5$ , male  $n = 5$ ),  $n = 7$  (CKO  $\times$  APP/PS1, female  $n = 4$ , male  $n = 3$ ). **d, e** CKO  $\times$  APP/PS1 mice showed significantly decreased contextual and cue-related freezing compared to WT mice.  $n = 13$  (WT, female  $n = 5$ , male  $n = 8$ ),  $n = 8$  (APP/PS1, female  $n = 3$ , male  $n = 5$ ),  $n = 9$  (CKO, female  $n = 5$ , male  $n = 4$ ),  $n = 7$  (CKO  $\times$  APP/PS1, female  $n = 4$ , male  $n = 3$ ). **f** ELISA analysis of A $\beta$  showed that neuronal *Cd2ap* deletion had no obvious influence on human A $\beta$  level.  $n = 3$  (APP/PS1, female  $n = 1$ , male  $n = 2$ ),  $n = 5$  (CKO  $\times$  APP/PS1, female  $n = 3$ , male  $n = 2$ ). **g** ELISA analysis of A $\beta$  showed that neuronal *Cd2ap* deletion had no obvious influence on murine A $\beta$  level.  $n = 6$  (WT, female  $n = 2$ , male  $n = 4$ ),  $n = 6$  (APP/PS1, female  $n = 3$ , male  $n = 3$ ),  $n = 4$  (CKO, female  $n = 3$ , male  $n = 1$ ),  $n = 6$  (CKO  $\times$  APP/PS1, female  $n = 4$ , male  $n = 2$ ). **h-j** In 4.5-month-old mice, Immunoblots revealed that neuronal *Cd2ap* deletion had no obvious influence on the full-length APP (APP-FL) and APP-CTF proteins.  $n = 6$  (WT, female  $n = 3$ , male  $n = 3$ ),  $n = 6$  (APP/PS1, female  $n = 3$ , male  $n = 3$ ),  $n = 6$  (CKO, female  $n = 3$ , male  $n = 3$ ),  $n = 6$  (CKO  $\times$  APP/PS1, female  $n = 3$ , male  $n = 3$ ). **k, l** In 4.5-month-old mice, Immunoblots revealed that neuronal *Cd2ap* deletion led to significantly increased ptau202/205 (AT8) and p-tau396 level, especially in the CKO  $\times$  APP/PS1 mice.  $n = 7$  (WT, female  $n = 3$ , male  $n = 4$ ),  $n = 7$  (APP/PS1, female  $n = 3$ , male  $n = 4$ ),  $n = 7$  (CKO, female  $n = 4$ , male  $n = 3$ ),  $n = 7$  (CKO  $\times$  APP/PS1, female  $n = 4$ , male  $n = 3$ ). All data are presented as mean  $\pm$  SEM. Unpaired *t*-test with two-tailed analysis (**f**), one-way ANOVA with Turkey's multiple comparison tests for multiple comparisons (**b-e, g, i, j, k**), Kruskal–Wallis tests with Dunn's multiple comparison tests (**l**). \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ , \*\*\*\* $P < 0.0001$



**Fig. 3** (See legend on previous page.)

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This correction does not affect the description of the results or the conclusion of this work.

The original article [1] has been updated.

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## Reference

1. Xue YY, Zhang ZS, Lin RR, et al. CD2AP deficiency aggravates Alzheimer's disease phenotypes and pathology through p38 MAPK activation. *Transl Neurodegener*. 2024;13:64. <https://doi.org/10.1186/s40035-024-00454-5>.