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# Author Correction: Amylin and pramlintide modulate $\gamma$ -secretase level and APP processing in lipid rafts

Youssef M. Mousa, Ihab M. Abdallah, Misako Hwang, Douglas R. Martin & Amal Kaddoumi 

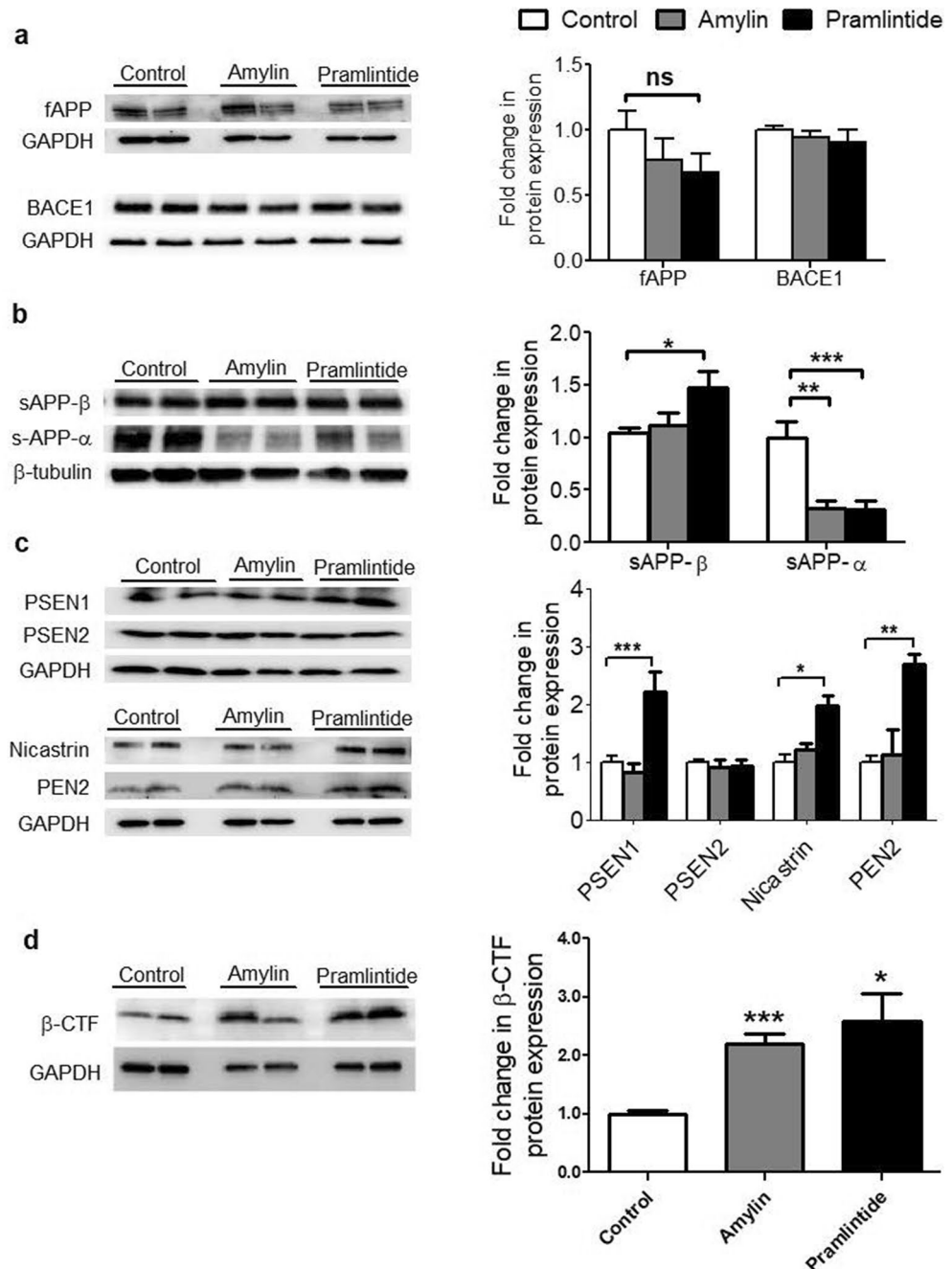
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This Article contains errors.

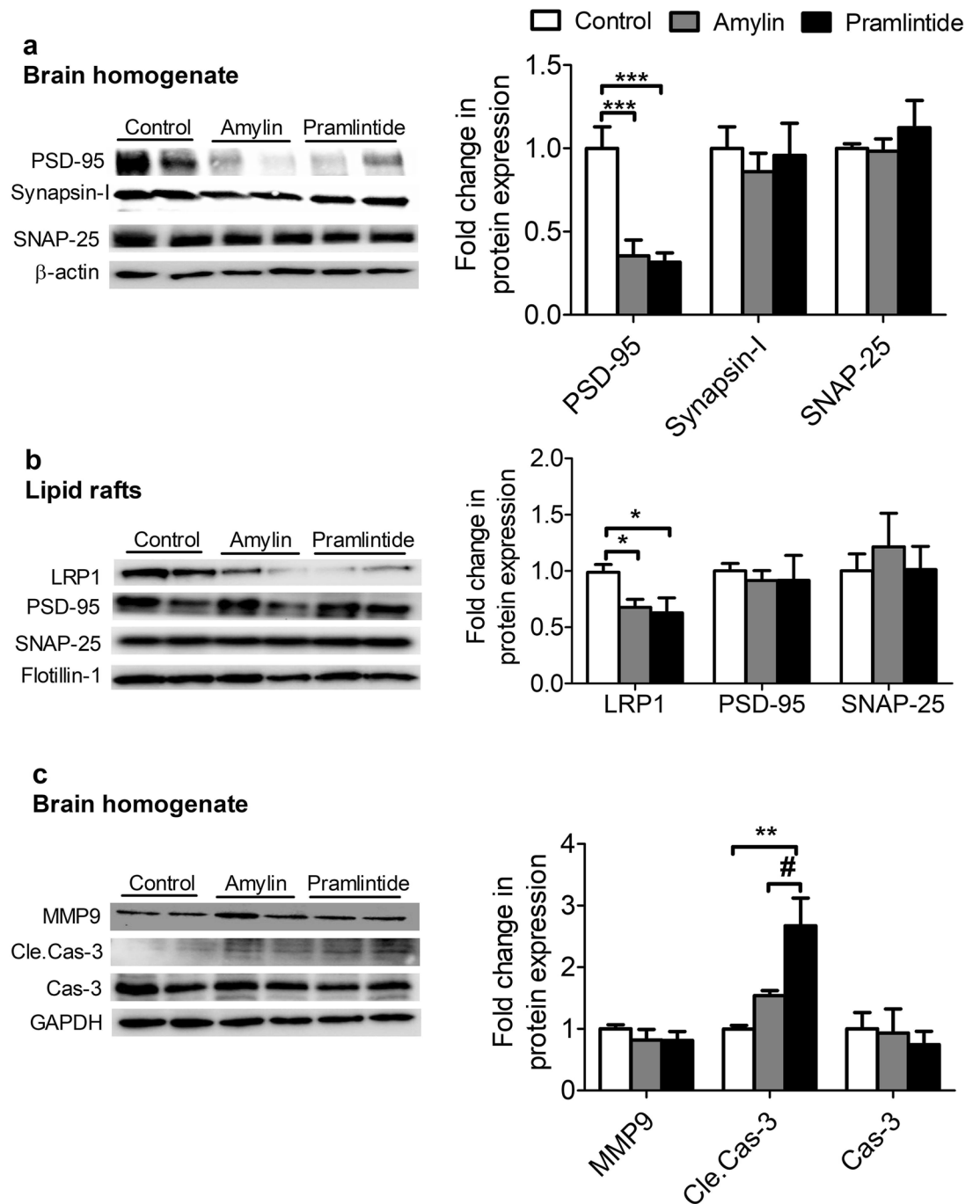
In Figure 2, Figure 2d is missing, which shows fold change in  $\beta$ -CTF protein expression. The correct Figure 2 appears below as Fig. 1.

In Figure 5b, which shows fold change in protein expression in lipid rafts, 'LRP1' is not represented. The correct Figure 5 appears below as Fig. 2.

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**Figure 1.** Effect of amylin and pramlintide on APP processing in total brain homogenate. (a) Representative Western blot and densitometry analysis of full-length APP (fAPP) and BACE1 demonstrated amylin and pramlintide did not alter full-length APP (fAPP) and BACE1 in mice brain homogenates. The fAPP and BACE1 levels were normalized to GAPDH level. (b) Representative Western blot and densitometry analysis of sAPP-β and sAPP-α in mice brains demonstrated pramlintide significantly increased sAPP-β compared to control, whereas reduction in the level of sAPP-α was observed after treatment with amylin and pramlintide. The levels of sAPP-β and sAPP-α were normalized to the level of β-tubulin. sAPP-β and sAPP-α were ran on different gels due to molecular weight similarity. (c) Representative Western blot and densitometry analysis of γ-secretase subunits in mice brains demonstrated pramlintide caused a significant increase in PEN2 subunit when compared to control and amylin; however, neither peptide influenced the other γ-secretase subunits PSEN1, PSEN2 and nicastrin. The level of γ-secretase subunits was normalized to level of GAPDH in each corresponding lane. PSEN1 and PSEN2 were ran on different gels due to molecular weight similarity. (d) Representative Western blot and densitometry analysis of β-CTF in mice brains demonstrated amylin and pramlintide caused a significant increase in β-CTF when compared to control. Data is presented as mean ± SEM, and the densitometry analysis is from n = 6 mice in each group. The western blot results are representative results from two different mice from each group. Data is presented as mean ± SEM and the statistical significance for all result was assessed by student t-test, with \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$  compared to control group; \* $p < 0.05$  compared to pramlintide, and ns = not significant.



**Figure 2.** Effect of amylin and pramlintide on A $\beta$ -related pathology. (a) Representative Western blot and densitometry analysis of synaptic markers in mice brain homogenates showed amylin and pramlintide significantly reduced the level of PSD-95, without affecting SNAP-25 and synapsin-1 in total brain homogenate. Data were normalized to  $\beta$ -actin. (b) Representative Western blot and densitometry analysis of synaptic markers and LRP1 in lipid rafts. Amylin and pramlintide had no effect on PSD-95 and SNAP-25 levels in lipid rafts; however, both peptides decreased the level of LRP1. All proteins from lipid rafts were normalized to flotillin-1. (c) A representative Western blot and densitometry analysis demonstrated that amylin and pramlintide significantly increased cleaved caspase-3 (Cle.Cas-3) compared to amylin and control group without affecting the levels of total caspase-3 (Cas-3) and MMP9. MMP9 was ran on different gel due to molecular weight similarity. All proteins were normalized to their corresponding housekeeping proteins. The densitometry analysis is from  $n = 6$  mice in each group. The western blot results are representative results from two different mice from each group. Data is presented as mean  $\pm$  SEM and the statistical significance for all result was assessed by student test, with  $*p < 0.05$ ,  $**p < 0.01$ , and  $***p < 0.001$ .



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