

Dissociation of ERK signalling inhibition from the anti-amyloidogenic action of synthetic ceramide analogues

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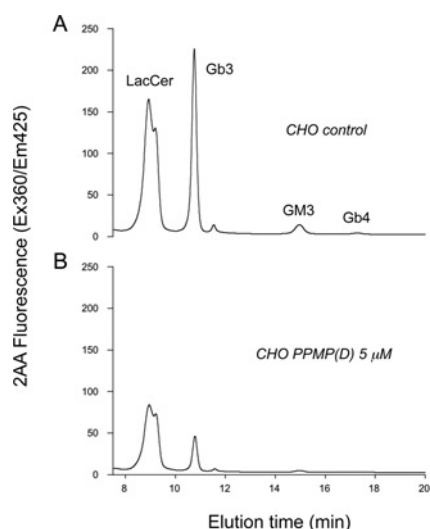


Figure S1 Inhibition of CHO-APP GSL levels by D-PPMP

CHO-APP cells were incubated for 48 h in the absence (upper profile, 'CHO control') or presence (lower profile, 'CHO PPMP(D) 5 μM') of 5 μM D-PPMP. Lipids were extracted and GSL glycans were analysed by HPLC as described in the legend to Figure 8. Gb3, globotriaosylceramide; Gb4, globotetraosylceramide; GM3, ganglioside G_{M3}.

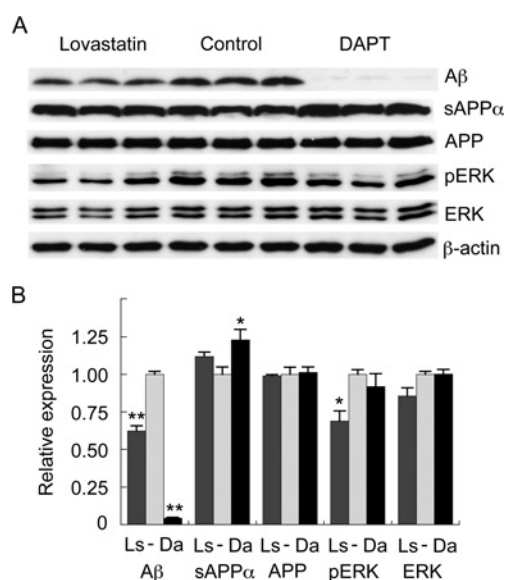


Figure S2 Influence of lovastatin and DAPT (two anti-amyloidogenic non-GSL-modifying compounds) on ERK activation

CHO-APP cells were treated with lovastatin (1 μM) or DAPT (1 μM) for 48 h, and secreted Aβ and sAPPα and cellular APP and total ERK and pERK (A) were measured by Western blotting with β-actin used as a loading control. Absorbance measurements of the Western blots are shown in the histograms (B): grey bars, control; dark grey bars, lovastatin treated; and black bars, DAPT treated. Results are mean ± S.E.M. values; *P < 0.05, **P < 0.01.

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