

Supplementary Data

Brain Pericytes ABCA1 Expression Mediates Cholesterol Efflux but not Cellular Amyloid- β Peptide Accumulation

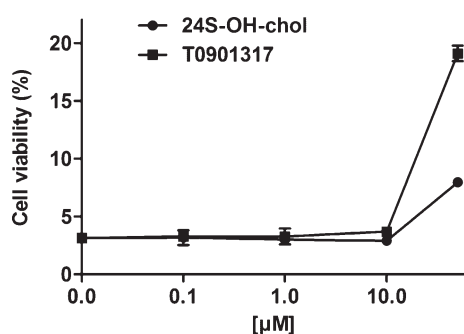
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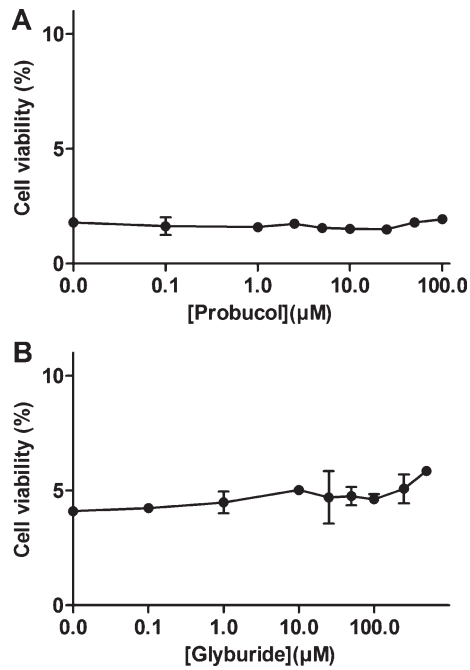
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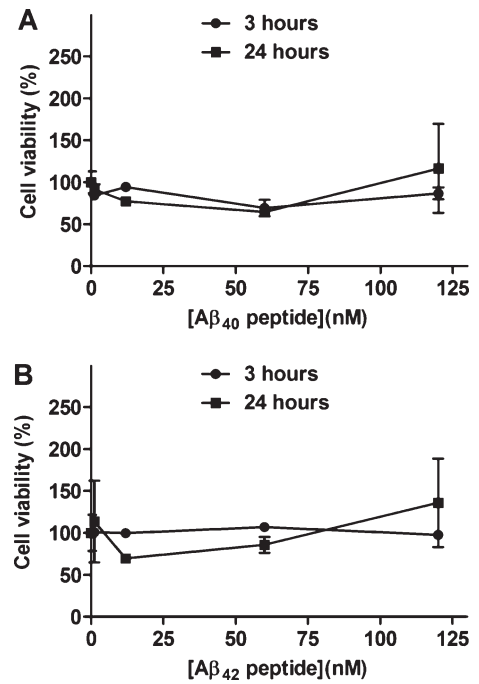


Supplementary Figure 1. The effect of LXR agonists on pericyte death. Brain pericytes were incubated 24 h in 0.1% BSA/DMEM containing different concentrations of 24S-OH-cholesterol (black circles) or TO901317 (black squares). Cell death was estimated using a lactate dehydrogenase (LDH) assay and the total lysis value (5426 ± 590 of relative fluorescent unit (RFU)) was obtained with a full-kill control condition. Data show the mean \pm s.d. for one of two representative experiments performed in triplicate.

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Supplementary Figure 2. The effect of the ABCA1 inhibitors probucol (A) and glyburide (B) on pericyte death. Brain pericytes were incubated 8 h in 0.1% BSA/DMEM containing different concentrations of probucol (0–100 μM) or glyburide (0–500 μM). Cell death was estimated using an LDH assay and the total lysis value was obtained with a full-kill control condition (3238 ± 585 and 6266 ± 346 RFU for probucol and glyburide experiments, respectively). Data show the mean \pm s.d. for one of two representative experiments performed in triplicate.



Supplementary Figure 3. The effect of the A β_{40} and A β_{42} peptides on pericyte death. Brain pericytes were incubated 3 h and 24 h in 0.5% BSA/DMEM containing different concentrations of A β peptides (0–120 nM). Cell death was estimated using an LDH assay and the total lysis value was obtained with a full-kill control. Results represent the percentage of cell viability compared with the control condition. Data show the mean \pm s.d. for one of two representative experiments performed in triplicate.