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Supplementary Data

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

Julien Saint-Pol[a,b,c], Elodie Vandenhaute[a,b,c], Marie-Christine Boucau[a,b,c], Pietra Candela[a,b,c], Lucie Dehouck[a,b,c], Roméo Cecchelli[a,b,c], Marie-Pierre Dehouck[a,b,c], Laurence Fenart[a,b,c] and Fabien Gosselet[a,b,c,*]
[a]*Univ Lille Nord de France, Lille, France*
[b]*UArtois, LBHE, Lens, France*
[c]*IMPRT-IFR114, Lille, France*

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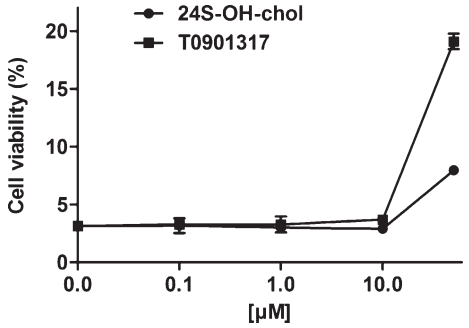

Supplementary Figure 1. The effect of LXR agonists on pericyte death. Brain pericytes were incubated 24 h in 0.1% BSA/DMEM containing different concentrations of 24S-OH-chol (black circles) or TO910317 (black squares). Cell death was estimated using a lactate dehydrogenase (LDH) assay and the total lysis value (5426 ± 590 of relative fluorescent unit (RFU)) was obtained with a full-kill control condition. Data show the mean ± s.d. for one of two representative experiments performed in triplicate.

*Correspondence to: Dr. Fabien Gosselet, Université d'Artois, Laboratoire de Physiopathologie de la Barrière Hémato-encéphalique, EA 2465 – IMPRT 114, Faculté Jean Perrin, Rue Jean Souvraz, S.P. 18, F-62300 Lens, France. Tel.: +33 321 791780; Fax: +33 321 791736; E-mail: fabien.gosselet@univ-artois.fr.

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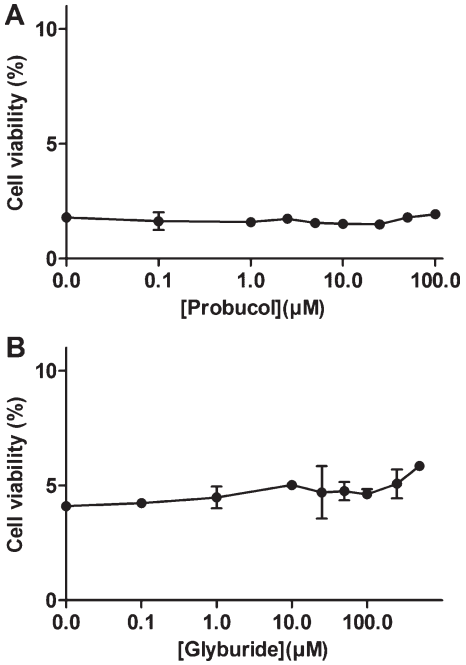
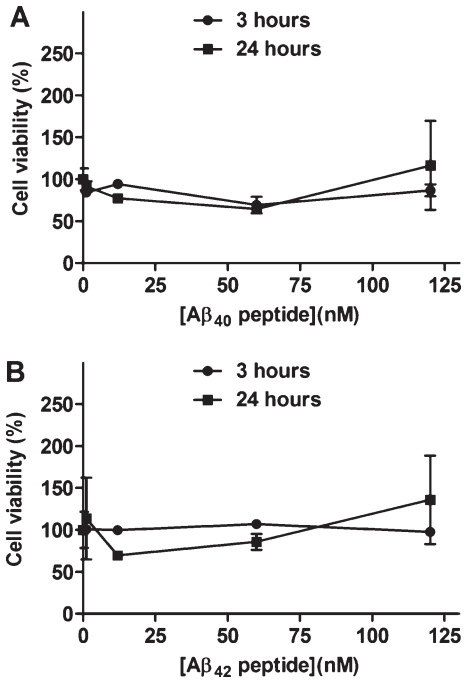


Supplementary Figure 2. The effect of the ABCA1 inhibitors probucol (A) and glyburide (B) on pericyte death. Brain pericytes were incubated 8 h in 0.1% BSA/DMEM containing different concentrations of probucol (0–100 μM) or glyburide (0–500 μM). Cell death was estimated using an LDH assay and the total 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 ± s.d. for one of two representative experiments performed in triplicate.



Supplementary Figure 3. The effect of the $A\beta_{40}$ and $A\beta_{42}$ peptides on pericyte death. Brain pericytes were incubated 3 h and 24 h in 0.5% BSA/DMEM containing different concentrations of Aβ 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 ± s.d. for one of two representative experiments performed in triplicate.