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Supplemental Information

β -Amyloid Clustering around ASC Fibrils

Boosts Its Toxicity in Microglia

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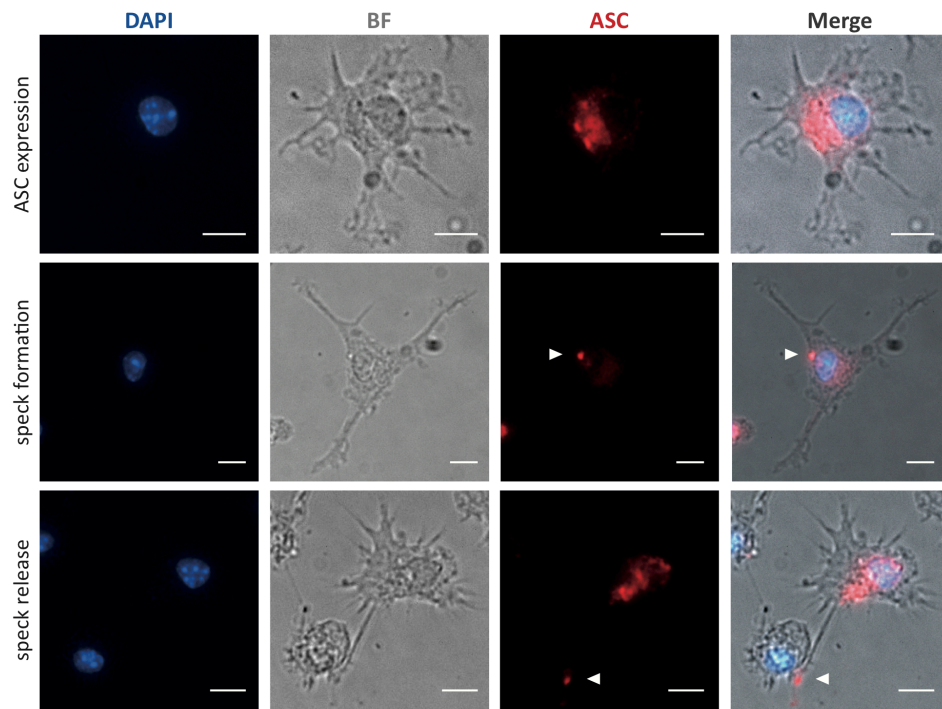


Fig. S1 | Exogenous ASC induces expression and specking of endogenous ASC. Related to Fig. 2 and Fig. 3. Immunocytochemical staining of ASC using a mouse-specific antibody (D2W8U). Top row: ASC expression, middle row: ASC speck formation, bottom row: ASC speck release. Bright-field (BF). Images were taken at 60 X magnification. Scale bar, 10 μ m.

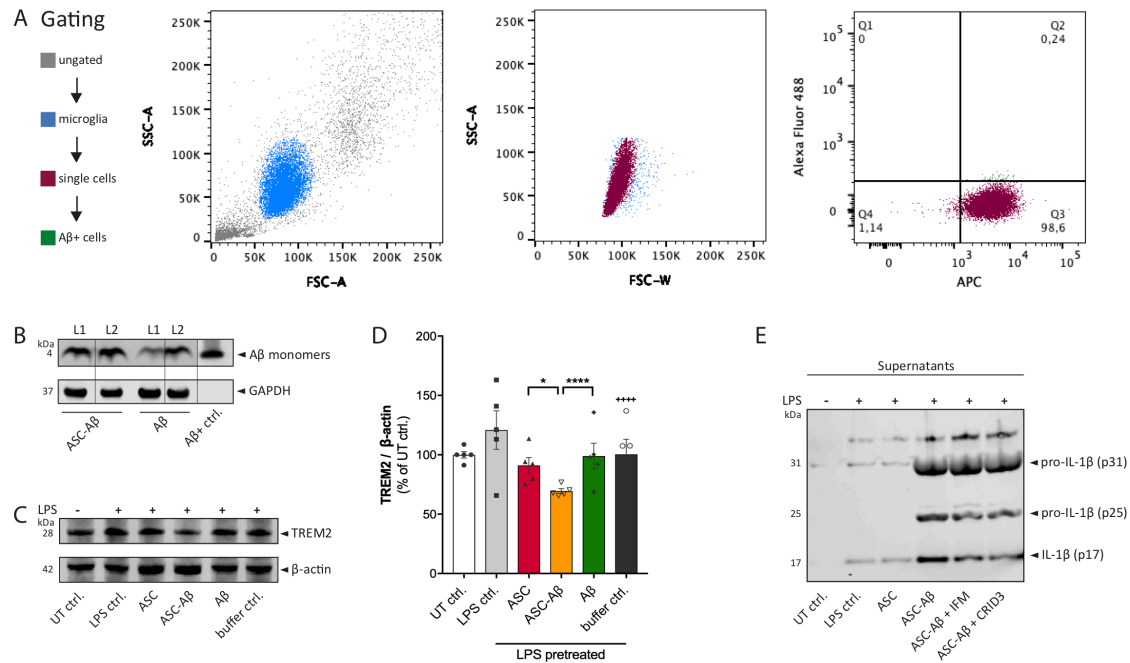


Fig. S2 | FACS gating. Treatment with ASC-Aβ composites reduces TREM2 expression in primary microglia. Related to Fig. 3 and Fig. 5.

All experiments displayed were performed using primary WT microglia. **A** FACS gating. Ungated (grey) → CD11b+ population, considered microglia (blue) → singlets (purple) → Aβ+ single microglia in Q2 (green). **B** Western blot of microglia cell lysates primed for 3 h with 100 ng/ml LPS and exposed to Aβ alone or ASC-Aβ composites for 12 h, stained for Aβ (82E1). **C**, **D** TREM2 expression detected in microglia lysates by western blot after 12 h of treatment post LPS-priming. Data were collected from two (**B**) or five (**C**, **D**) independent experiments (n = 2, n = 5). **(E)** Representative immunoblot for IL-1β in microglia supernatants treated with ASC-Aβ without or with co-application of the NLRP3 inflammasome inhibitors IFM-2384 or CRID3. All graphs are presented as mean ± SEM and were analysed by unpaired t-test. Levels of significance are indicated as *p < 0.05; **p < 0.01; ***p < 0.001; ****p < 0.0001. Star-symbol indicates significance between groups connected by lines; plus-symbol indicates significance between ASC-Aβ composites and volume equal buffer ctrl. treated groups.