

Supplementary Fig. 1. Quantitation of p47^{phox} and p67^{phox} expression. (A) and (B) Quantitative analysis (n=3 for each group) of p47^{phox}-GFAP and p67^{phox}-GFAP co-localization in *App*Tg and *App*Tg/*Cebpd*^{-/-} mice using ImageJ software. (C) and (D) Quantitative analysis of p47^{phox} and p67^{phox} protein level by IL-1β-treated wild-type and *Cebpd*^{-/-} mice primary astrocyte. The bands were quantified using ImageJ software. n.s. not significant. (*p<0.05, **p<0.01, Student's *t*-test)



Supplementary Fig. 2. Cebpd activates *Nrf2* expression in primary astrocytes. Overexpression of HA/Cebpd (HA-Cd) in primary astrocytes could increase *Nrf2* expression. The total RNA was harvested from Cebpd-overexpressed cells and Q-PCR was conducted with *Nrf2* primers. (*p<0.05, Student's *t*-test)

(A)

(B)



Primary astrocyte

IL-1 β Control SOD1 Control **GFAP** + **SOD1** Control IL-1β IL-1β **GFAP** + SOD1 IL-1 β

Supplementary Fig. 3. Compared with primary astrocytes, neuronal Sod1 expression was not increased after IL-1 β treatment. (A) The astrocytic *Sod1* transcription was highly increased after IL-1 β treatment, while the neuronal *Sod1* transcription was not induced by IL-1 β . The total RNA was harvested from IL-1 β -treated primary cells and Q-PCR was conducted with specific primers. (B) Sod1 immunoreactivity co-localized with GFAP, a specific neuron marker, and was highly increased by IL-1 β , while Sod1 co-localized with MAP2, a specific neuron marker, was not significantly induced after IL-1 β treatment. Primary neurons and astrocytes were treated with IL-1 β and subjected to immunofluorescence with anti-GFAP, anti-MAP2 and anti-Sod1 antibodies. Similar results were obtained from two independent experiments, each performed in triplicate, and the data shown here were from one representative assay. n.s. not significant. (***p<0.001, One-way ANOVA)



Supplementary Fig. 4. IL-1 β -induced transcription of *Cebpd* was increased in primary astrocytes compared with primary neuronal cells. The total RNA was harvested from IL-1 β -treated cells and Q-PCR was conducted with specific primers. (*p<0.05, Student's *t*-test)



Supplementary Fig. 5. IL-1β increases mitochondrial superoxide production in primary astrocytes.

(A) Purity of primary astrocytes culture. Primary mouse astrocytes were stained with astrocyte specific marker, GFAP (green), and microglia specific marker, Iba1 (red). (B) Primary astrocytes were treated with 5 ng/ml IL-1 β . After 6 h, the mitochondrial superoxide production was detected with mitochondrial superoxide indicator by immunofluorescent staining.