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Supplemental information

Anoxia-induced hippocampal LTP is regeneratively produced by glutamate and nitric oxide from the neuro-glial-endothelial axis

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SUPPLEMENTAL INFORMANTION

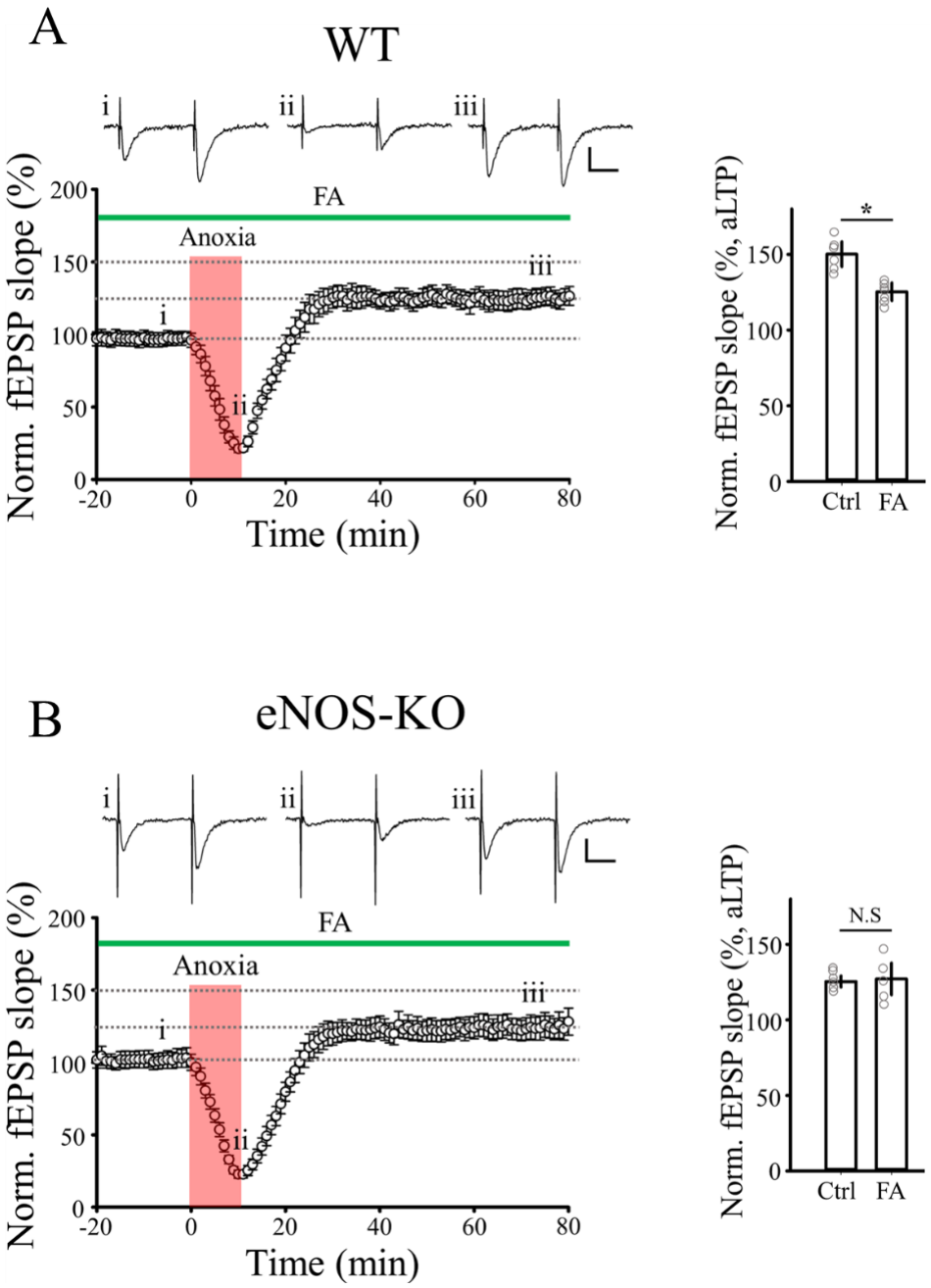

Fig S1. FA selectively blocks eNOS-dependent LTP. Related to Figure 5.

(A) In slices from WT mice, FA (10 mM) reduced aLTP of fEPSPs to 125.3 ± 6% (n = 7) in comparison to controls (150 ± 8%, n = 7, $p < 0.05$, Student's *t*-test). (B) FA had no effect on aLTP in slices from eNOS-KO mice (Ctrl 125 ± 4%, n = 6, FA 127 ± 10 %, n = 5, Student's *t*-test). *$p < 0.05$, N.S, no significance. Bar graphs (A, B) indicate aLTP magnitude in the presence of FA and control (contr) in the absence of FA. Scale bars: 0.2 mV, 20 ms.

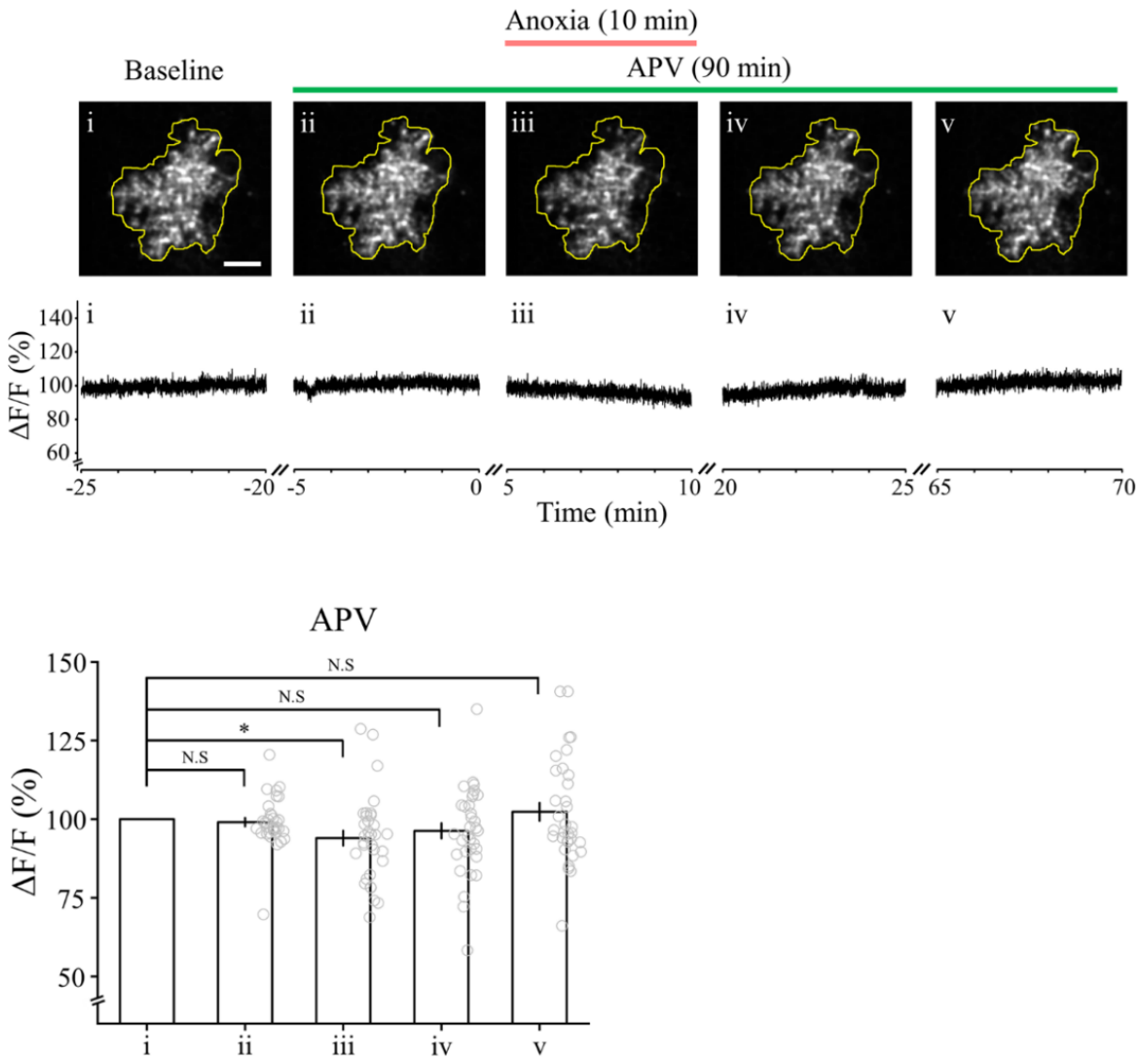

Fig S2. APV blocks anoxia-induced Ca^{2+} elevation in astrocytes. Related to Figure 6.

Two-photon Ca^{2+} imaging of GCaMP6f-expressing astrocytes. Bath-application of APV alone (100 μM for 90 min from 20 min before anoxia) had no effect on the astrocytic Ca^{2+} signal (ii). Anoxic insult (10 min from time 0) caused a transient reduction of Ca^{2+} signal (iii), but it recovered to the baseline within 10 min (iv) without showing Ca^{2+} signal potentiation (v) in the presence of APV. Bar graphs for astrocytic Ca^{2+} signal quantification in APV application experiments. APV significantly reduced astrocytic Ca^{2+} signal after anoxia (iii, 91.41 ± 2.2%, 11 slices, n = 33, one-way repeated-measures ANOVA: $F_{(4, 160)}$ = 9.95) with no potentiation thereafter (iv, see below). *p < 0.05, N.S, not significance. Ca^{2+} signals from 1-3 astrocytes are simultaneously monitored and quantified.