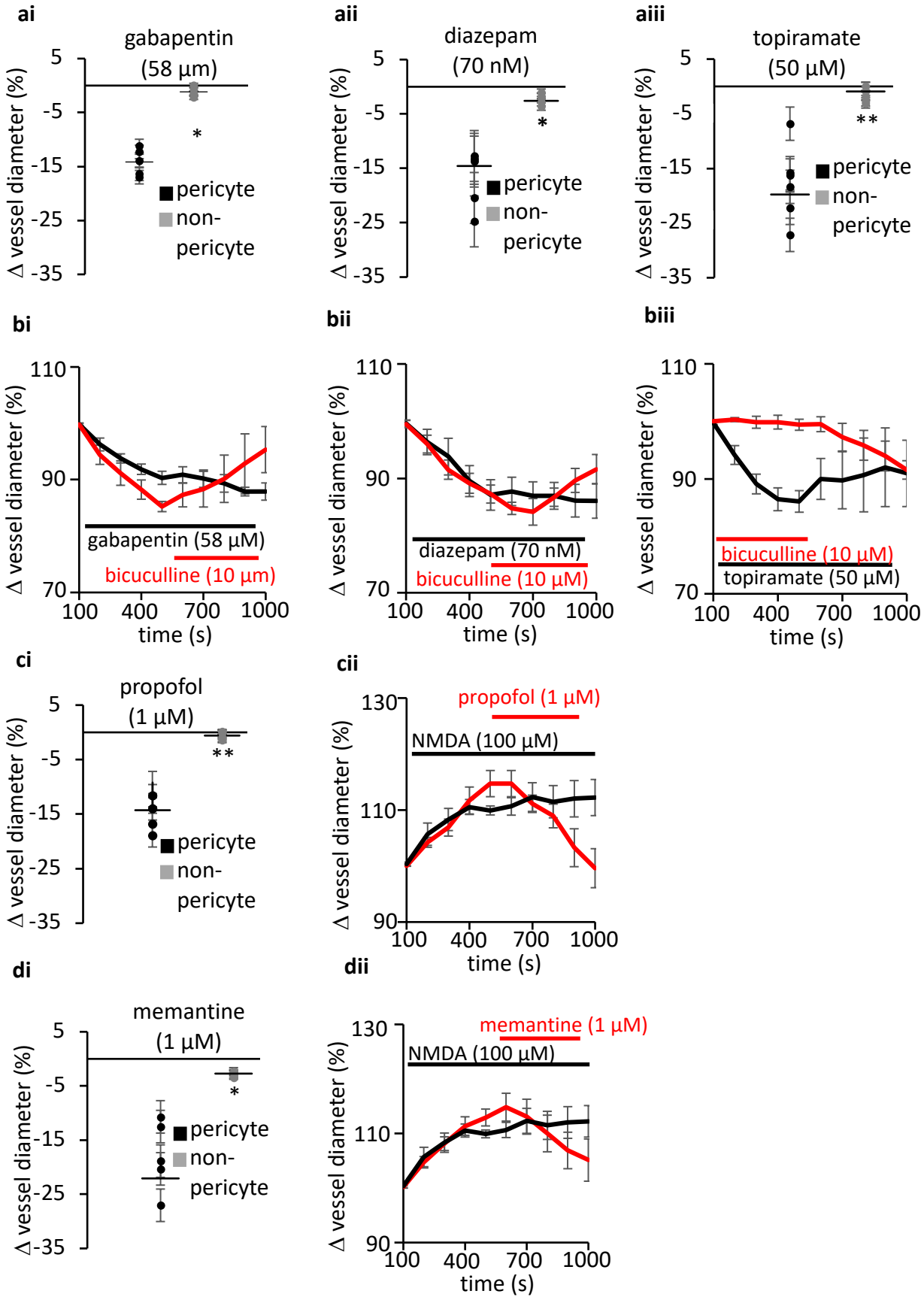
ai
gabapentin
(58 µm)
Δ vessel diameter (%)
5
-5
-15
-25
-35
*
pericyte
non-
pericyte
aii
diazepam
(70 nM)
*
pericyte
non-
pericyte
aiii
topiramate
(50 µM)
**
pericyte
non-
pericyte
bi
110
90
70
gabapentin (58 µM)
bicuculline (10 µm)
100
400
700
1000
time (s)
bii
diazepam (70 nM)
bicuculline (10 µM)
biii
bicuculline (10 µM)
topiramate (50 µM)
ci
propofol
(1 µM)
**
cii
propofol (1 µM)
NMDA (100 µM)
130
110
90
di
memantine
(1 µM)
dii
memantine (1 µM)

Figure 6 | Pericyte-mediated regulation of MBF in response to modulators of $GABA_A$Rs and NMDARs. Data was taken from time series experiments in which naïve kidney slices were exposed a variety of different compounds. Scatter plots show mean data for gabapentin (58 nM; **ai**), diazepam (70 μM; **aii**), topiramate (10 μM; **aiii**), propofol (1 μ; **ci**) and memantine (1 μM; **di**) induced pericyte-mediated constriction of vasa recta capillaries. **bi-iii** all show representative traces of gabapentin, diazepam and topiramate-evoked vasoconstriction of vasa recta (respectively; black lines), which is attenuated by bicuculline (10 μM) for all agents (red lines). **cii,** Representative trace showing the NMDA-evoked (100 μM) dilation of vasa recta by pericytes (black line) is attenuated by propofol (1 μM; red line). **dii,** Representative trace showing NMDA-evoked dilation of vasa recta by pericytes (black line) is attenuated by memantine (1 μM; red line). Data shown from male Sprague-Dawley rats as mean ± s.e.m, n ≥ 3 pericytes. Statistics were calculated in GraphPad PRISM (5.0). Statistical significance between pericyte and non-pericyte sites were determined using: a Student's t-test for pericyte versus non-pericyte sites, $^{**}P < 0.01$; $^{*}P < 0.05$