

Figure 4 | Glutamate signalling pathways. Data was taken from time series experiments in which naïve kidney slices were exposed to glutamate (glut) or glycine (gly) alone (**a**-**d**) or in the presence of other comounds (**e**-**l**). **a**, **c**, Representative traces of percentage change in vessel diameter (blue trace) and percentage change in DAF-FM fluorescence (red trace) in response to exposure of vasa recta to glutamate (10 μ M) and glycine (1 mM). DAF-AM signal before (**bi** and **di**), during (**bii** and **dii**) and after (**biii** and **diii**) superfusion with glutamate or glycine. White lines denote the vessel wall, yellow circle = pericyte and red brackets show where vessel diameter was measured, white scale bar = 10 μ m . **e**, Glutamate-evoked dilation was significantly attenuated by L-NNA (100 μ M). **f**, representative trace showing percentage change in vessel diameter in response to exposure to glutamate and LNNA. ODQ (10 μ M) failed to significantly attenuate the glutamate-evoked dilation. Data shows the representative trace). Both PPOH (9 μ M; **I**, mean data, **j**, representative trace) and L-161,982 (1 μ M; **k**, mean data, **l**, representative trace) significantly attenuated the glutamate mediated dilation. Data shown from male Sprague Dawley rats as mean \pm s.e.m, n \geq 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 comparison between drugs. ***P < 0.001; **P < 0.01, * P < 0.05