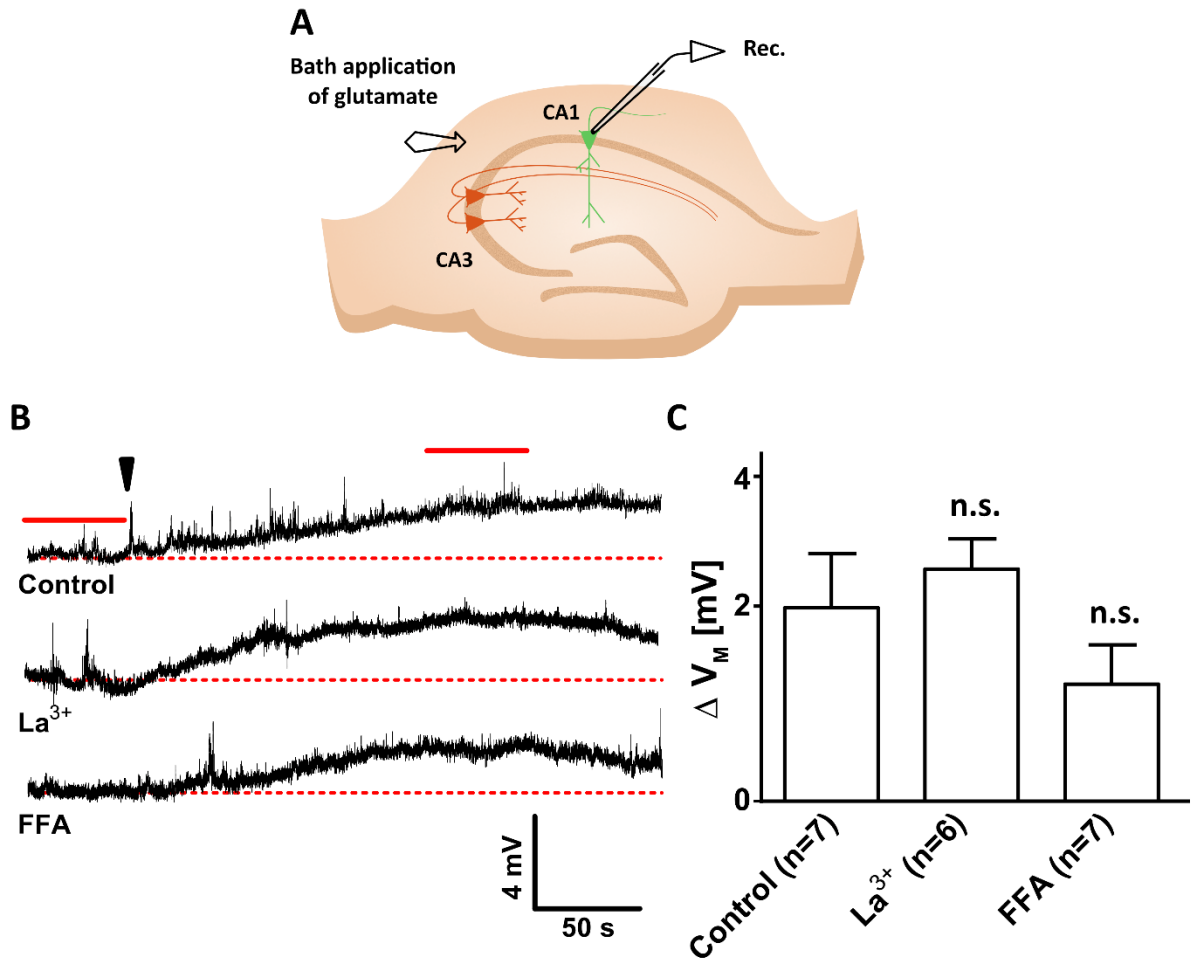
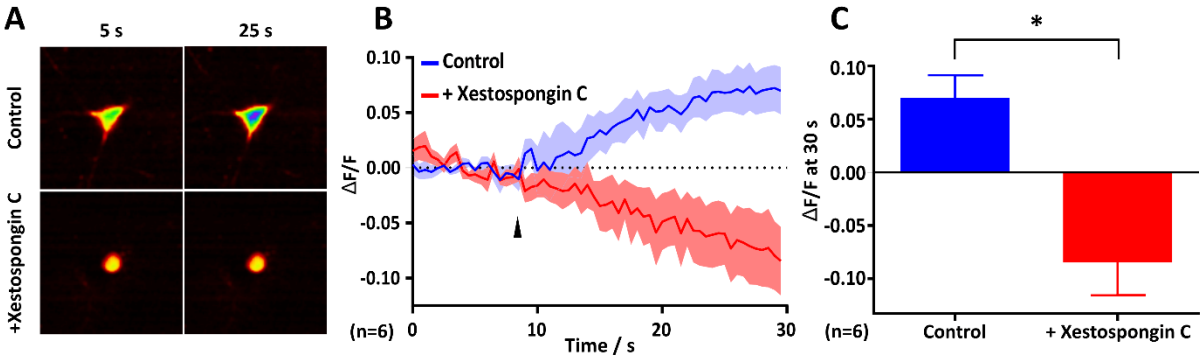



**Supplementary Figure 1 mGluR1-mediated depolarisation unlikely to occur via transient receptor potential (TRP) channel activation. A.** Diagram showing the experimental configuration, whole cell current clamp recordings were performed on CA1 pyramidal neurons whilst mGluR1 was pharmacologically isolated and L-glutamate was bath applied. **B.** Representative voltage recordings during application of extracellular glutamate (180 s, black arrow indicates where glutamate enters bath) **C.** columns show mean Δ $V_M$ of CA1 pyramidal neurons before and after glutamate perfusion (indicated by red bars in B) for each pharmacological manipulation undertaken. Drugs were added to the bath solution at the following concentrations: $La^{3+}$ (100 µM), flufenamic acid (FFA) (20 µM). Error bars display SEM. n.s. = non-significant difference in mean membrane potential compared to control.




**Supplementary Figure 2 Xestospongin C inhibits somatic $IP_3$ mediated $Ca^{2+}$ release in CA1 pyramidal neurons in the hippocampus.** CA1 pyramidal neurons were filled with the $Ca^{2+}$ indicator OGB-1 (1 mM) via patch pipette for one minute. After recovery, a series of confocal images were collected whilst cells were re-patched (at 9 s) with internal solution containing $IP_3$ (100 µM) under control conditons or with pre-incubation with the $IP_3$ receptor antagonist Xestospongin C (2 µM, 20 minutes). **A.** Representative confocal images taken before (5 s) and after break through (25 s). **B.** ΔF/F over the imaging time course, break through with $IP_3$ containing internal solution is shown by the arrow at 9 s. **C.** Columns show mean ΔF/F at 30 s. Error bars show SEM (n = 6 for both groups). Significant differences are displayed by asterisks, where * = $P < 0.05$.