Supplemental Material:

Fig. S1:

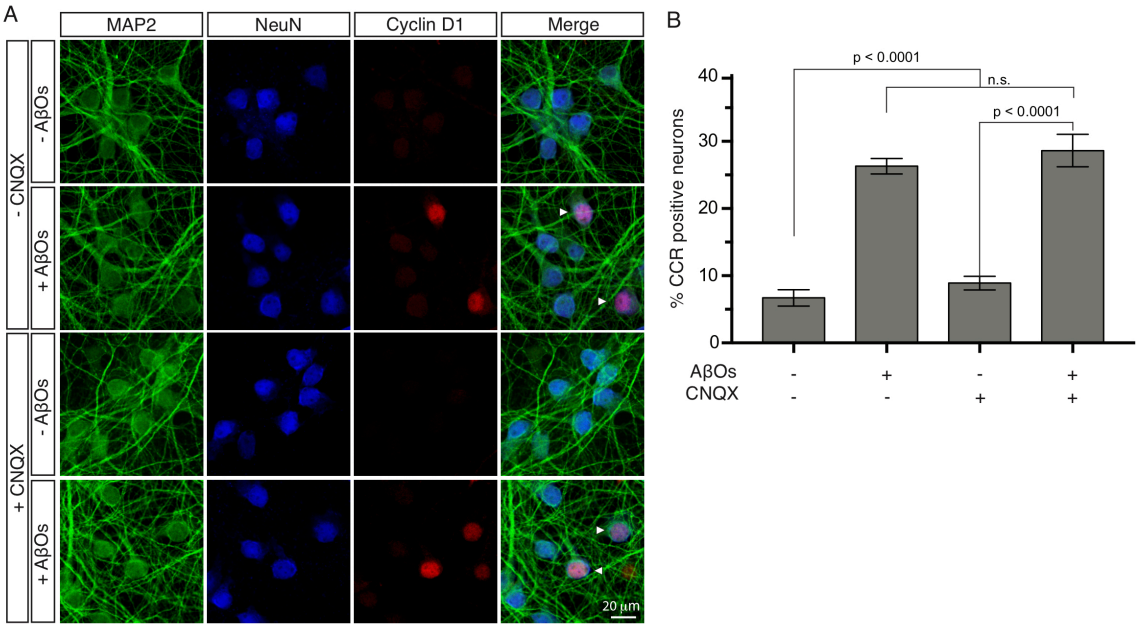

Fig. S1: The selective α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAR) inhibitor, CNQX, does not inhibit CCR. (A) Primary cortical neurons were treated overnight with AβOs with or without 10 μM CNQX. After 18 hours of exposure to AβOs, neurons were stained for MAP2, NeuN, and Cyclin D1 to mark neurons that had re-entered the cell cycle. (B) Quantification of the immunofluorescence results and statistical analysis by one-way ANOVA demonstrates that AMPAR inhibiton by CNQX does not block AβO-induced CCR. AMPAR therefore does not appear to modulate the AβO-induced calcium entry that drives CCR. Error bars indicate s.e.m.

Supplemental Fig. 2:

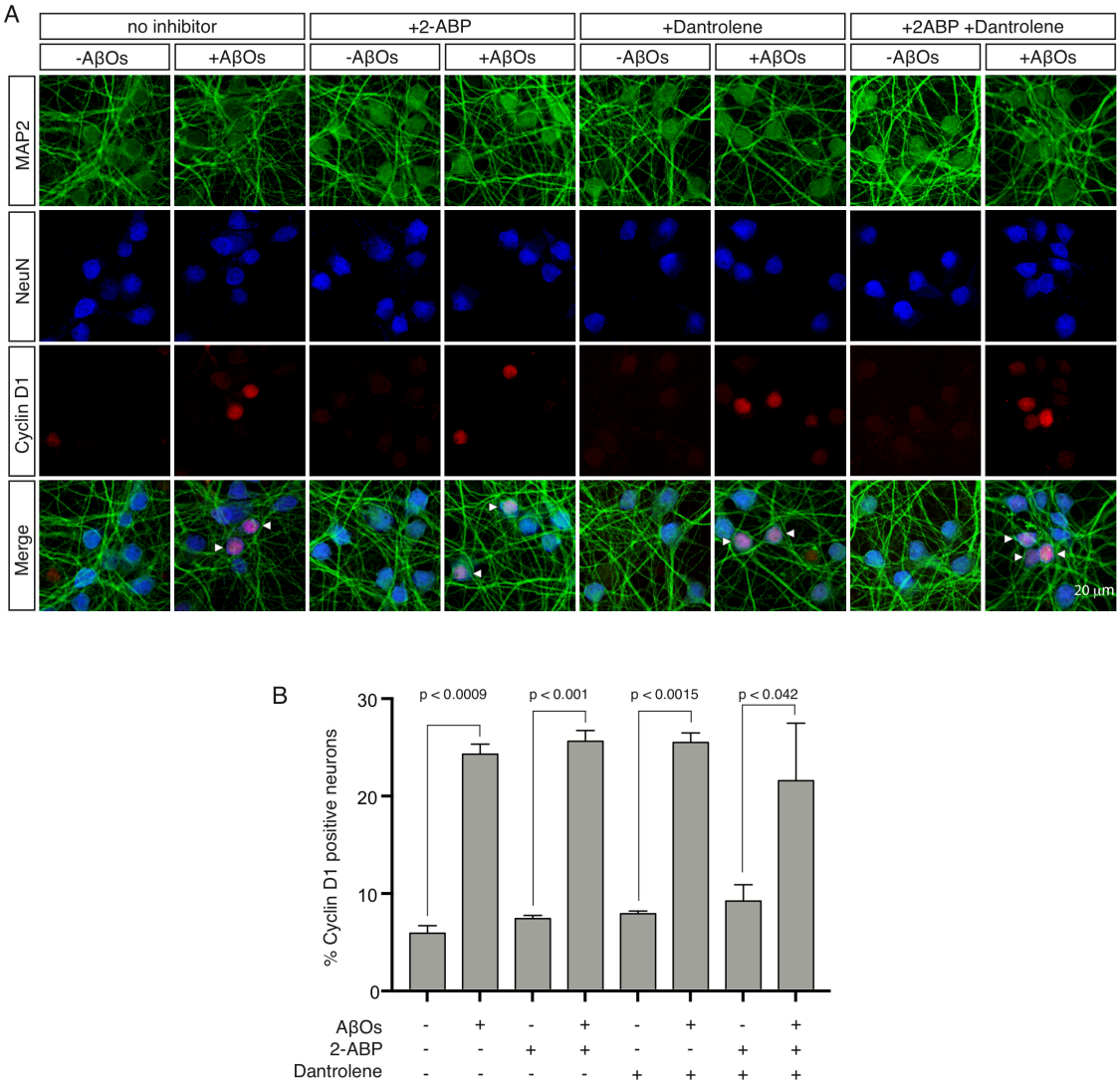

Fig. S2: Inhibitors of endoplasmic reticulum (ER) IP_3 and ryanodine receptors do not block activation of CCR. (A) Primary cortical neurons were treated overnight with AβOs with or without 50 µM 2-aminoethoxydiphenyl borate (2-APB) to block IP_3 receptors, 20 µM dantrolene to block ryanodine receptors, or both inhibitors. After 18 hours of exposure to AβOs, neurons were stained for MAP2, NeuN, and Cyclin D1 to mark neurons that had re-entered the cell cycle. (B) Quantification of the immunofluorescence results. Neither drug, alone or in combination, blocked AβO-induced CCR. Calcium release by the ER therefore does not contribute significantly to the AβO-induced calcium release that drives CCR. Indicated p values were calculated by one-way ANOVA using the Bonferroni post-hoc test. Error bars indicate s.e.m.