

Supplemental figures:

Figure 1: Localization of CaMKII isoforms at the NMJ.

The animal was perfused with 2% PFA and the sternomastoid muscle was removed and cleaned from connective tissues. Whole muscle was then immunostained with commercially available antibodies against CaMKII β , γ and δ . Muscles were then incubated with a secondary antibody conjugated to Alexa 488 and BTX-Alexa 594 (red) to label nAChRs and synapses were then scanned with confocal microscopy. **A)** Example of a stack of 24 confocal images (0.5 μ m intervals) of an NMJ that was labeled with anti-CaMKII δ and collapsed onto a single plane; AChRs in the postsynaptic membrane (**B**); **C)** Overlay of the Alexa 488 fluorescence in green and the nAChR in red. **D-F)** Representative image of a neuromuscular junction immunostained with anti-CaMKII γ . **G-I)** Representative image of a NMJ immunostained with anti-CaMKII β . Note that all CaMKII antibodies show similar pattern of fluorescence labeling (nerve and postsynaptic membrane) at the synapse. Images were adjusted in Photoshop (Adobe) to maximize contrast.

Figure 2: Overexpression of CaMKII β m has no effect on the recycling of receptors at the NMJ.

The sternomastoid muscle was electroporated with CaMKII β m and one week after electroporation the sternomastoid muscle was labeled with BTX-biotin followed by a saturating dose of Alexa 647. Five days later, the sternomastoid muscle was bathed with streptavidin Alexa 594 to label recycled AChRs. The Alexa 594 fluorescence of recycled receptors on electroporated muscles was quantified and compared to non electroporated muscles. **A)** Example of an expressed CaMKII β m at the NMJ of an electroporated muscle as shown by white lines; **B)** Overlay of labeled recycled AChRs at electroporated and non electroporated muscle cells; **C)** Fluorescence quantification of labeled recycled receptors on the electroporated and non electroporated muscle fibers; **D)** Graph summarizing data obtained from many synapses. Note that the average recycled receptor density at synapses expressing CaMKII-GFP and synapses from non electroporated synapses was similar (P value: 0.2, student test)

Figure 3: Treatment of the sternomastoid muscle with KN93, or stimulation plus KN93 has no effect on BTX-biotin dissociation rate from receptors.

The sternomastoid muscle was labeled with BTX-biotin followed by a saturating dose of streptavidin. Five days later, the mouse anaesthetized and subjected to stimulation with or without KN93 during 8 hours. The sternomastoid muscle was removed and AChR-Biotin-complexes were pulled down with Neutravidin. The same amount of proteins was subjected to SDS and immunoblotted with anti-AChR subunit alpha. **A)** Representative blots show that treated stimulated with KN93, treated unstimulated with KN93, and untreated unstimulated sternomastoid muscles have similar amount of pulled down AChRs. **B)** Graph shows the mean densitometry quantification from three independent experiments. Quantification of scanned blots was performed by using Image J (NIH Image Software).

Figure S1

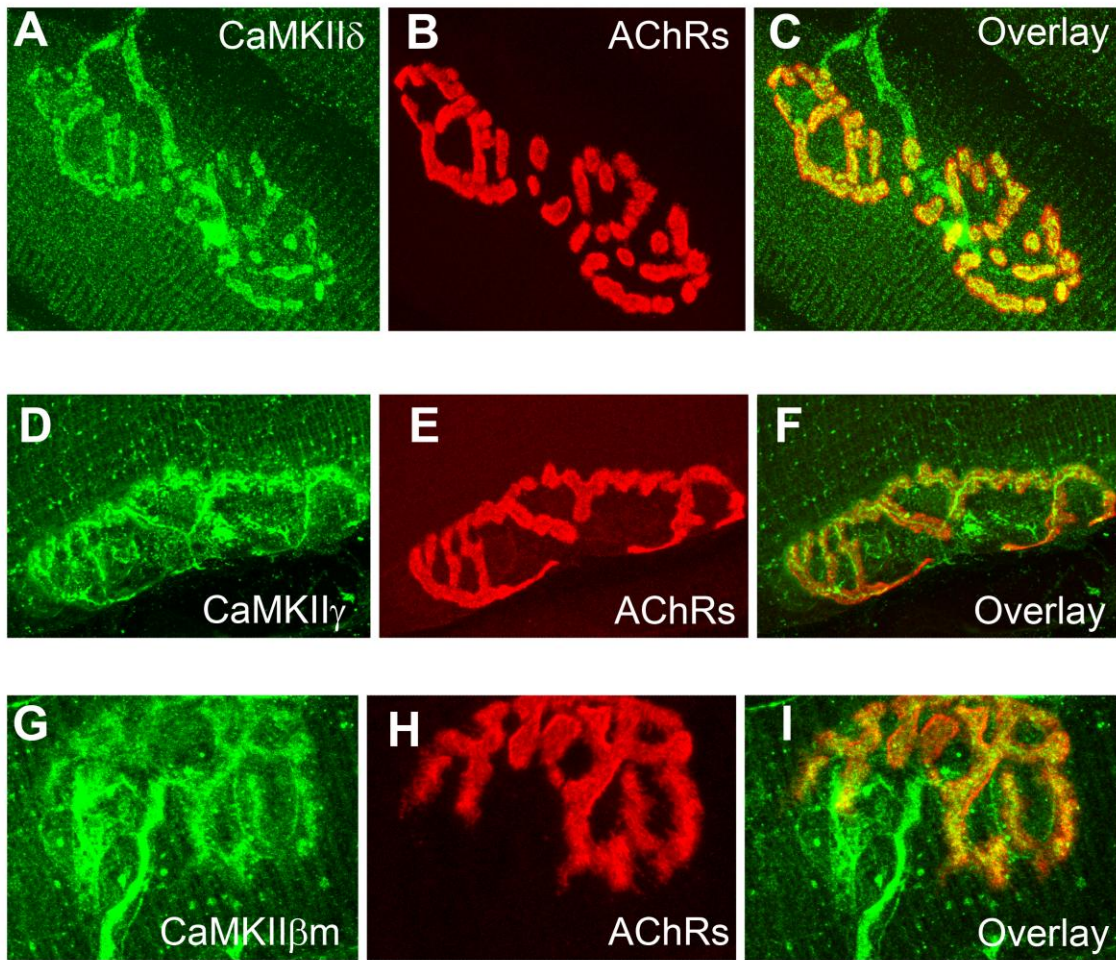


Figure S2

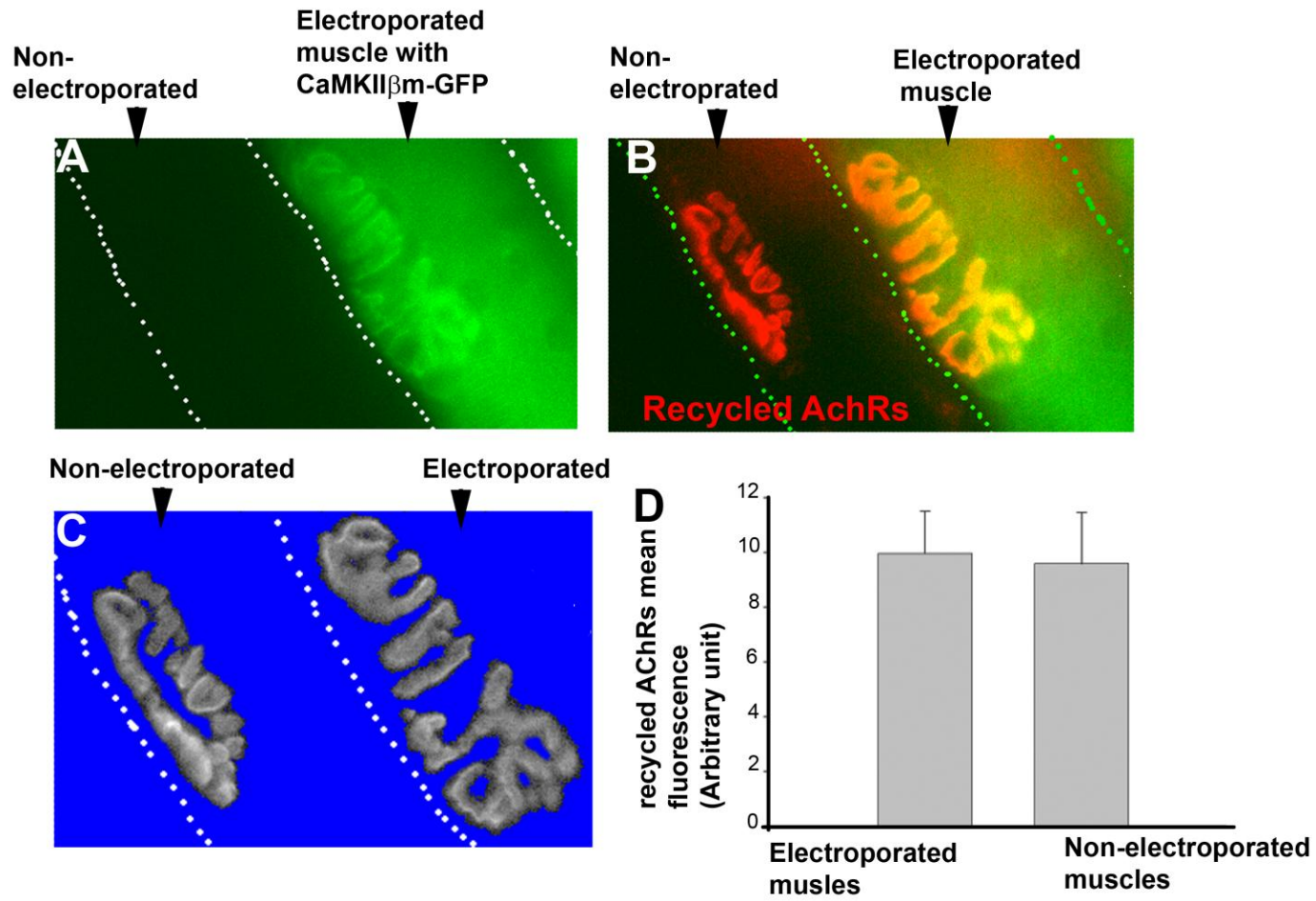


Figure S3

