Supplementary Figure 4. Electrophysiology on wild-type GluRδ2 and the lurcher mutant GluRδ2^lc receptor.

Representative current traces obtained from a two-electrode voltage-clamp recording on (A) uninjected Xenopus oocytes, (B) wild-type GluRδ2 receptor injected oocytes, or (C) GluRδ2^lc injected oocytes. Application of 1 mM D-serine (black bars) and 100 µM NASP (white bar) are indicated at the time indicated by the bars above the current trace. (D) Application of NASP alone and co-application of D-serine + NASP inhibited current at GluRδ2^lc by 1.37 ± 0.04 (N=5) and 1.40 ± 0.04 (N=5), respectively, relative to the current inhibition produced by application of D-serine alone.

When expressed in oocytes, GluRδ2^lc conductance is manifest as a shift in the amplitude of the holding current required to clamp the oocyte at negative membrane potentials compared to wild-type GluRδ2 or un-injected oocytes (data not shown). As GluRδ2^lc conductance is sensitive to bath application of the channel blocker 1-naphthyl acetyl spermine (NASP), application of NASP can be used to assess the contribution of the GluRδ2^lc current to the increased holding current observed in these oocytes. Changes in holding current to application of D-serine (1 mM) or NASP (100 µM) in uninjected oocytes or oocytes expressing wild-type GluRδ2 were not seen. Responses to D-serine (1 mM) were only observed in oocytes injected with RNA encoding GluRδ2^lc and in which the holding current was sensitive to NASP (100 µM). These data demonstrate that expression of GluRδ2^lc is a requirement for inhibition of holding current upon application of D-serine. Furthermore, D-serine (1 mM) is unable to elicit additional current response at GluRδ2^lc when applied together with NASP (100 µM), hereby demonstrating that the effect of D-serine is specific for GluRδ2^lc.