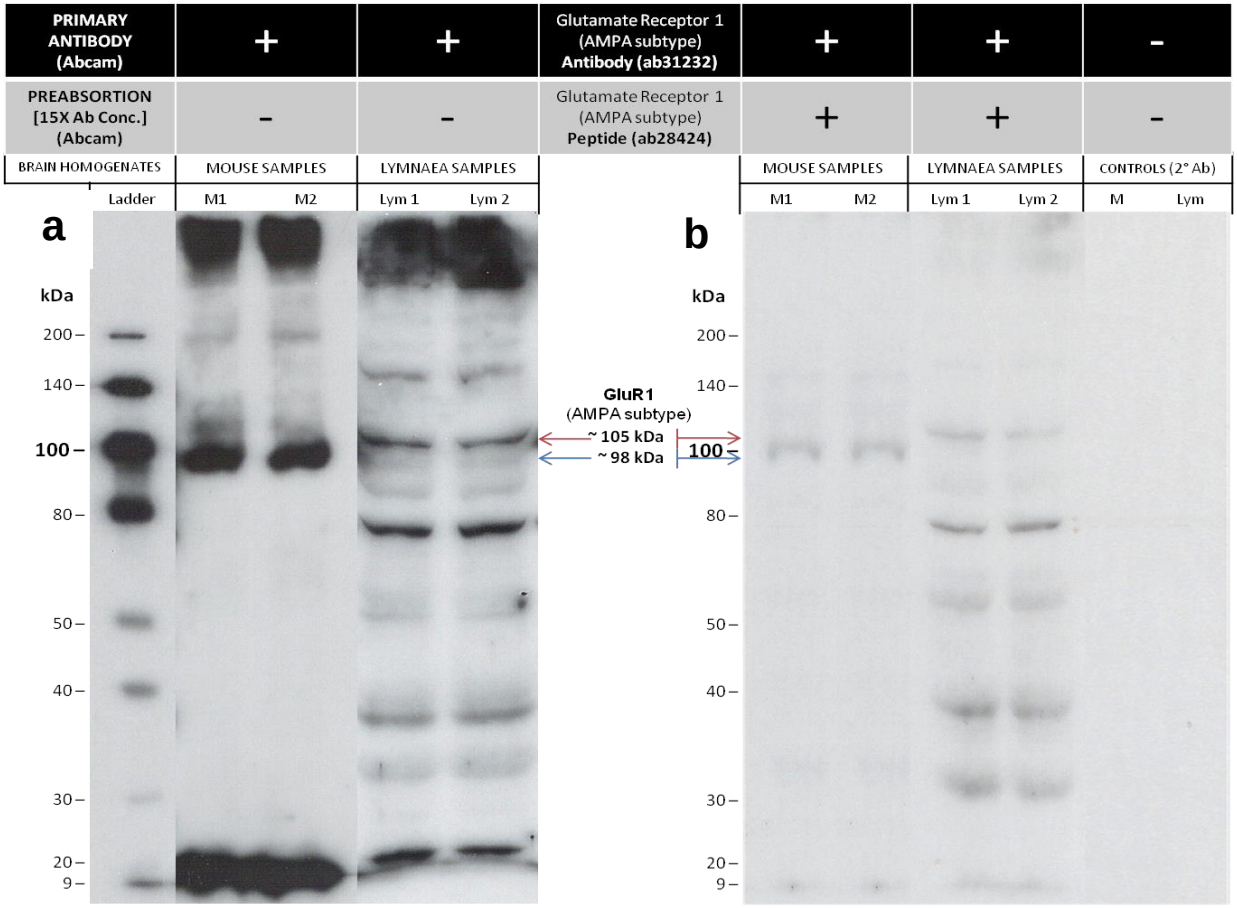
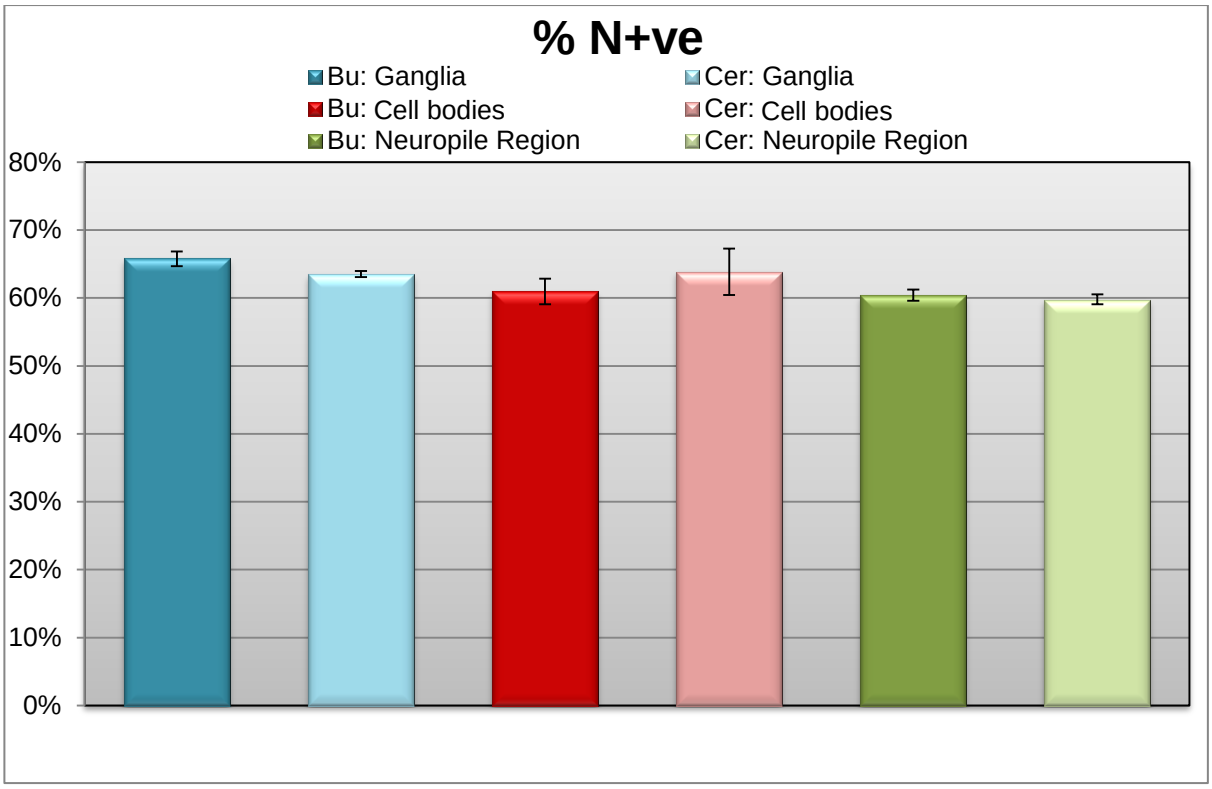
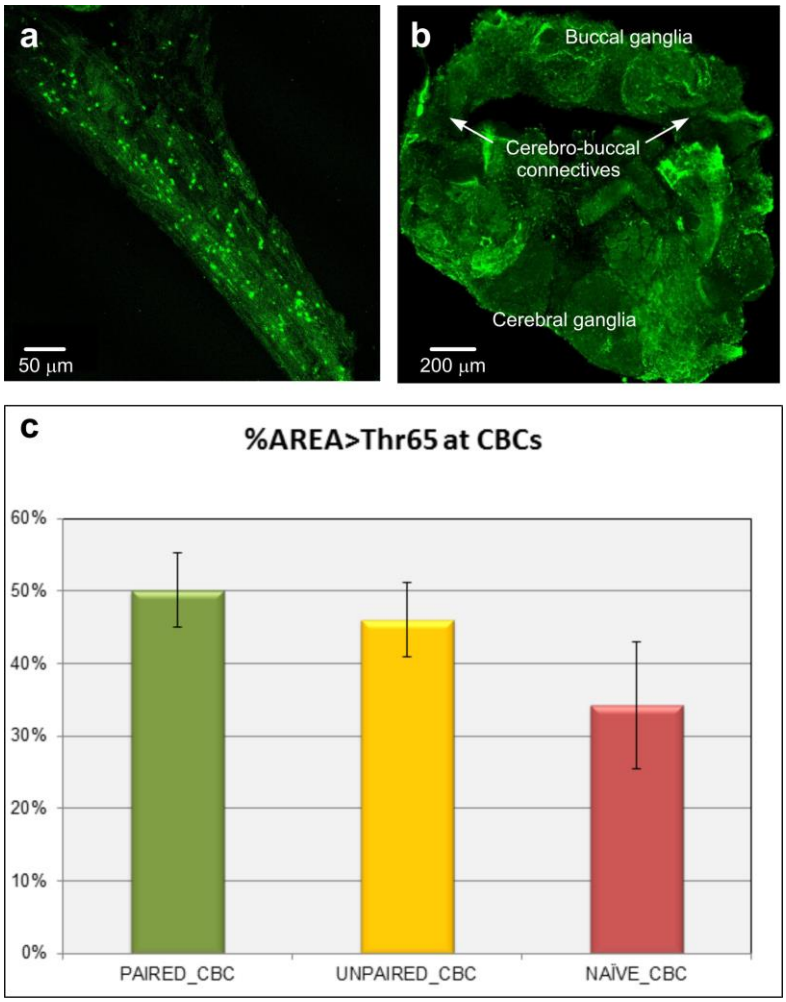


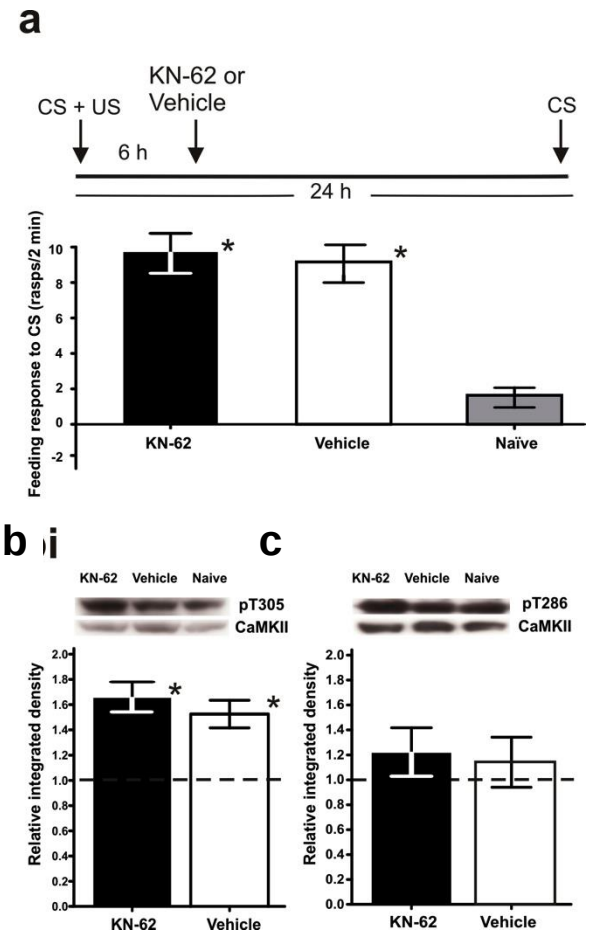
Supplementary Figure 1. Western blot detection of mouse GluA1 and Lym-eGluR1[1,2] (with a rabbit GluA1 antibody [ab31232], and the specificity of detection. A set of mouse (M1, M2 and M) and *Lymnaea* (Lym1, Lym2 and Lym) protein samples were resolved by SDS-PAGE (8.25 %) and electro-blotted onto PVDF membrane. **(a)** The primary antibody detected a very prominent band of ~ 98 kDa in the mouse samples (M1 and M2). Although the same band is also detectable in *Lymnaea* samples (Lym1 and Lym2, blue arrow), it is very low in intensity compared to the band expressed at ~105 kDa (red arrow). **(b)** Pre-incubation with the corresponding synthetic peptide [AB28424] most strongly suppressed the expression of the ~105 kDa and ~98 kDa bands in the *Lymnaea* samples (Lym1 and Lym2), and almost completely suppressed the ~98 kDa band in the mouse samples (M1 and M2). Controls not exposed to primary antibody or synthetic peptide showed no band expression in either sample.



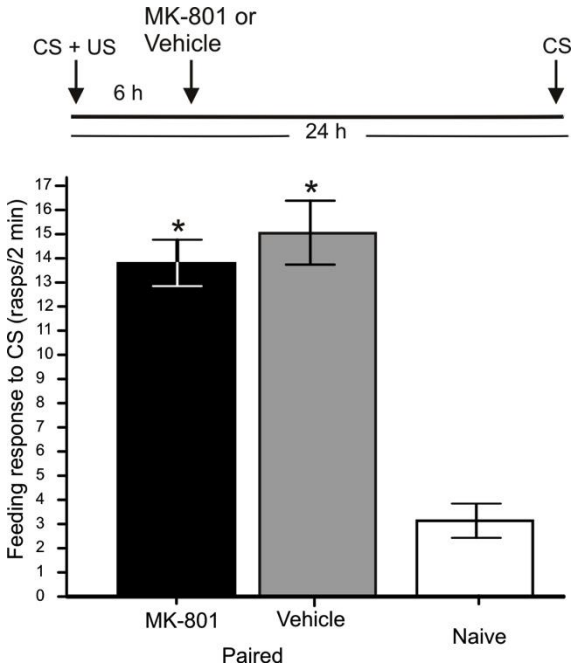
Supplementary Figure 2. Results of the intensity correlation analysis (ICA) of Glu-A1 and PSD-95 co-colocalization. Means (±SEM) percentage values of green (GluA1) and red (PSD-95) pixel pairs that have only positive PDM (**P**roduct of **D**ifferences from the **M**ean) values, over the total number of pixel pairs (%N+ve) is shown.



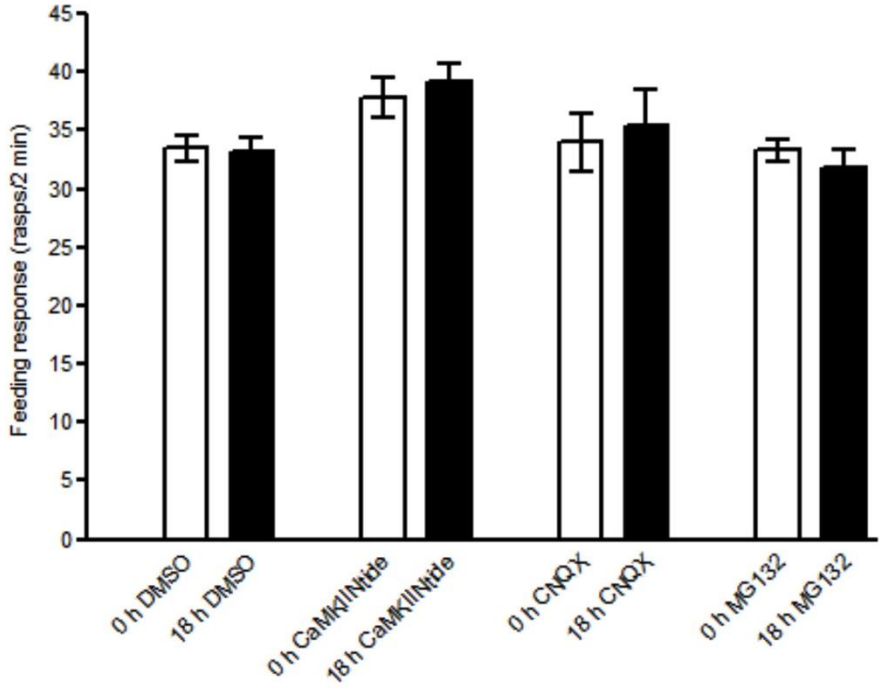
Supplementary Figure 3. GluA1 receptor expression in the cerebro-buccal connective and connective tissue surrounding the 'learning ganglia'. (**a**) Glu-A1 immunoreactive punctae in the cerebro-buccal connective (CBC). (**b**) Glu-A1 immunoreactivity in the connective tissue surrounding the buccal and cerebral ganglia interconnected by the cerebro-buccal connectives. This image is from a whole-mount preparation. Please note that the same type of preparation was used in the western blot experiments. (**c**) Increased GluA1 immunoreactivity in CBCs from Unpaired and Paired animals (*N*=5 each) compared to CBCs from Naïve animals (*N*=5). Although an ANOVA did not identify a source of significant difference among the 3 groups, there is a tendency for both the Unpaired and Paired values to be higher compared to the Naïve value.



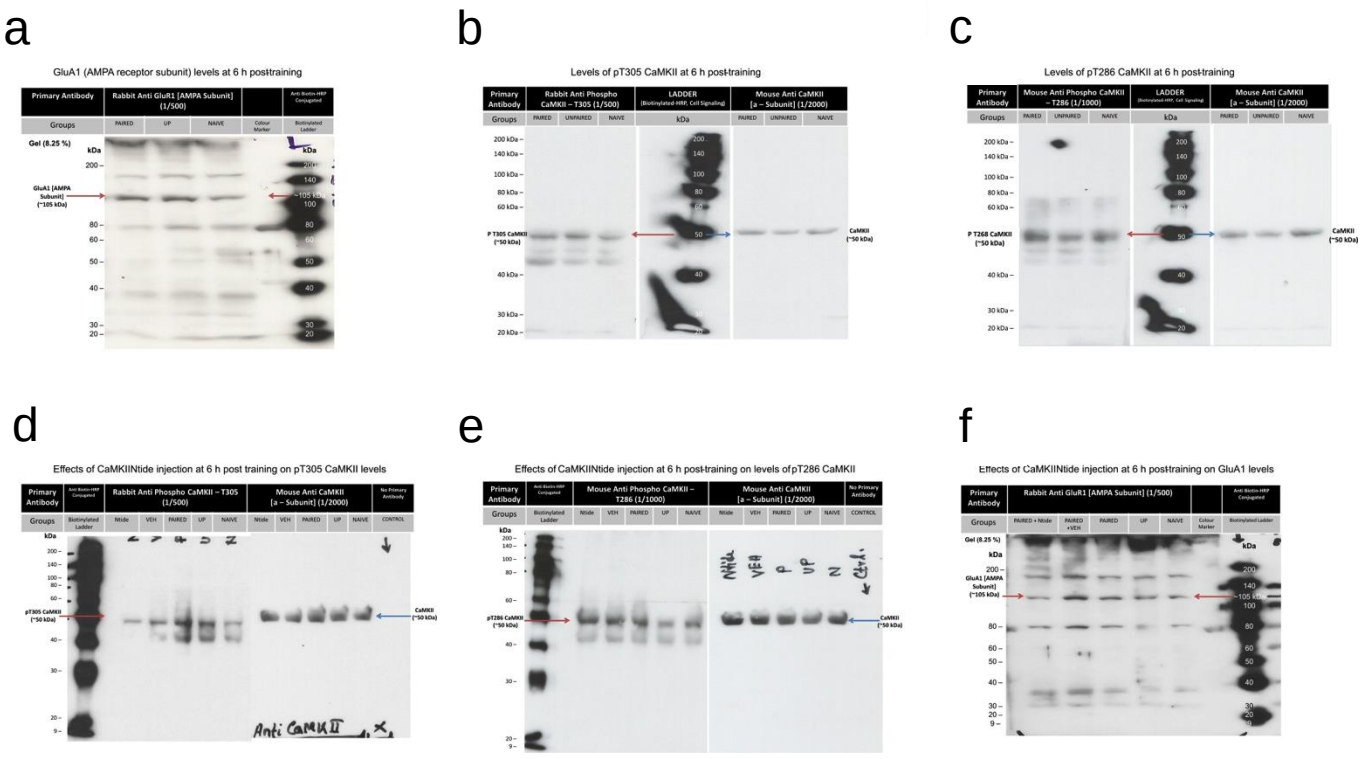
Supplementary Figure 4. KN-62 treatment at 6 h after classical conditioning does not affect ongoing memory consolidation or the learning-induced phosphorylation state of CaMKII in *Lymnaea*. (a) Results of the 24 h memory test conducted concurrently with the biochemical assays shown in b. Timeline for the experiment is shown above the diagram. At the 24 h test with the CS, both the classically conditioned and 6 h later KN-62 treated (*N*=61) and the classically conditioned and 6 h later vehicle-treated animals (*N*=62) showed a significantly higher (asterisks) feeding response to the CS compared to the Naïve animals (*N*=53). ANOVA: *P*<0.0001. Tukey's tests: KN-62 versus Naive and Vehicle versus Naive, both *P*<0.05; KN-62 versus Vehicle, *P*>0.05. **(b)** KN-62 treatment at 6 h after classical conditioning has no effect on the learning-induced phosphorylation state of CaMKII. Examples of pT305-CaMKII, pT286-CaMKII and CaMKII western blot bands from the same KN-62 treated, Vehicle treated and Naïve samples (each containing protein extracts from the buccal and cerebral ganglia and cerebro-buccal connectives of 12 animals) are shown above the graphs. The means (±SEM) of pT305-CaMKII (**b**) or pT286-CaMKII (**c**) over CaMKII levels are shown (relative integrated density). The values thus calculated for KN-62 treated samples (**b**, *N*=6; **c**, *N*=6) and Vehicle treated samples (**b**, *N*=6; **c**, *N*=6) were normalized to the mean of the density values obtained from naïve samples (**b**, *N*=14; **c**, *N*=12), providing a baseline of 1 (dashed line). Both KN-62 and Vehicle treatment 6 h after paired training leave the significantly higher than baseline level (increased by training, see **Fig. 6a**), of pT305-CaMKII unaffected (**b**). Asterisks indicate significant differences compared to the naïve baseline. One sample t tests for data in **b**: KN-62 versus naïve baseline, *P*<0.009; Vehicle versus naïve baseline, *P*<0.02. There is no statistically significant difference between the normalized data from the KN-62 and Vehicle group (two-sample t-test, *P*=0.15). T286 phosphorylation remains unaffected by paired training (also see **Fig. 6b**) and also by treatment with either KN-62 or vehicle (**c**). One sample t tests for data in **c**: KN-62 versus naïve baseline, *P*=0.45; Vehicle versus naïve baseline, *P*=0.23. There is no statistically significant difference (Two-sample t-test, *P*= 0.92) between the normalized data from the KN-62 and Vehicle group either.



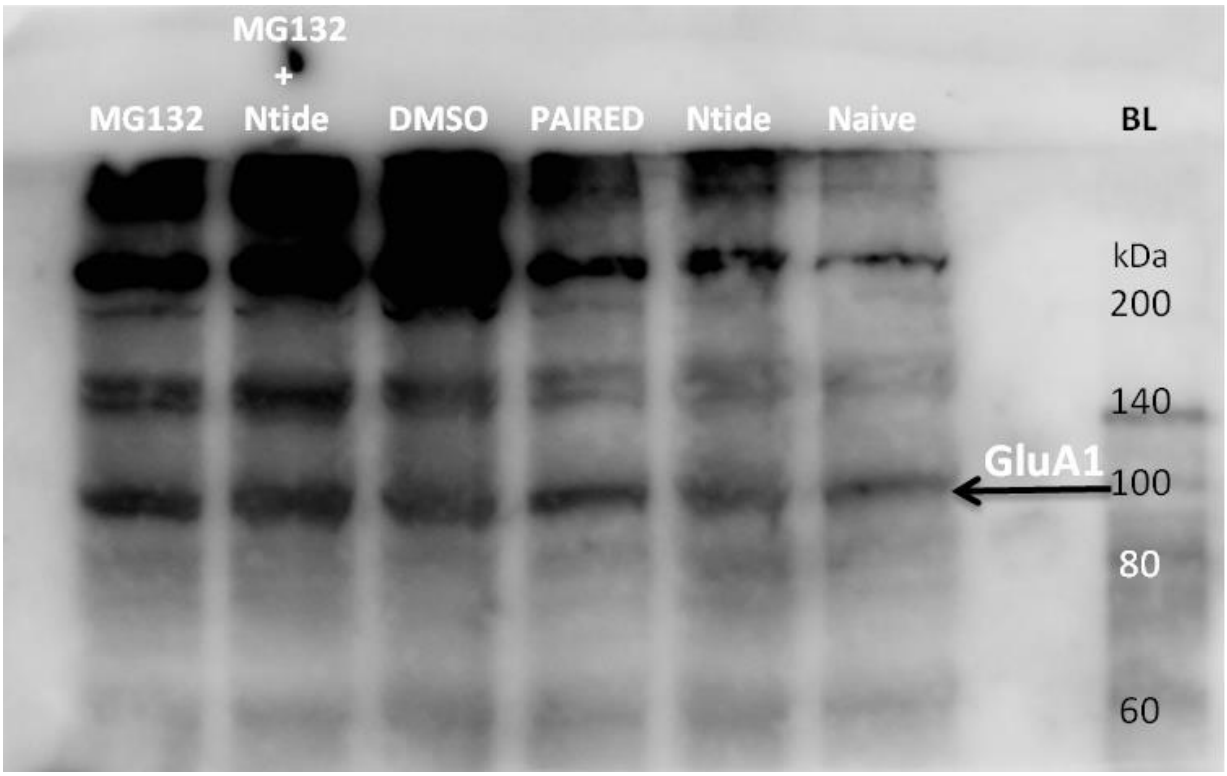
Supplementary Figure 5. Blocking NMDA receptors at 6 h after classical conditioning does not affect ongoing memory consolidation in *Lymnaea*. Results of the 24 h memory test are shown with the timeline for the experiment above the diagram. Means (±SEM) are shown. At the 24 h test with the CS, both the classically conditioned and 6 h later MK-801 treated (*N*=32) and the classically conditioned and 6 h later vehicle-treated animals (*N*=32) showed a significantly higher (asterisks) feeding response to the CS compared to the Naïve animals (*N*=32). One-way ANOVA: $P<0.0001$. Tukey's tests: MK-801 vs Naive and Vehicle versus Naive, both $P<0.05$; MK-801 versus Vehicle, $P>0.05$.



Supplementary Figure 6. CaMKIINtide, CNQX or MG132 do not affect the ability of animals to generate a full feeding motor program at 18 h post-treatment. The 18 h post-injection time was used because in the learning experiments drugs were injected at 6 h post-training and the feeding response to the CS was tested at 24 h post-training. Six hours after testing them for their feeding response to sucrose, animals (*N*=10 in each group) were injected with CaMKIINtide (4 μM final concentration), CNQX (20 μM final concentration), MG132 (1 μM final concentration) or DMSO in saline (vehicle, 0.2% final concentration). Eighteen hours after the administration of the drugs and the vehicle, respectively, animals from each group were re-tested for their feeding response to sucrose. These tests revealed normal feeding responses in all drug-treated groups (paired t-tests, *P* values between 0.17 and 0.86), confirming that neither the drugs nor the vehicle affected the animals' ability to generate a full feeding motor program in response to the appropriate sensory stimulus. Means (±SEM) are shown.



Supplementary Figure 7. Full-length blots for the example immunoblot images shown in Fig. 2 (**a**), Fig. 6a and b, respectively (**b, c**), Fig. 7b and c, respectively (**d, e**) and Fig. 8 (**f**).



Supplementary Figure 8. Full-length blot for the example immunoblot image shown in Fig. 9b. Please note that the left-to-right order of the bands was re-arranged in Fig. 9b to match the order in which the behavioral data are presented in Fig. 9a.

Supplementary References

1. Hutton, M.L., Harvey, R.J., Barnard, E.A. & Darlison, M.G. Cloning of a cDNA that encodes an invertebrate glutamate receptor subunit. FEBS Lett. 292, 111-114. (1991).

2. Stuhmer, T., Amar, M., Harvey, R.J., Bermudez, I., Minnen, J.V. & Darlison, M.G. Structure and pharmacological properties of a molluscan glutamate-gated cation channel and its likely role in feeding behavior. J. Neurosci. 16, 2869-2880 (1996).