Supporting Information

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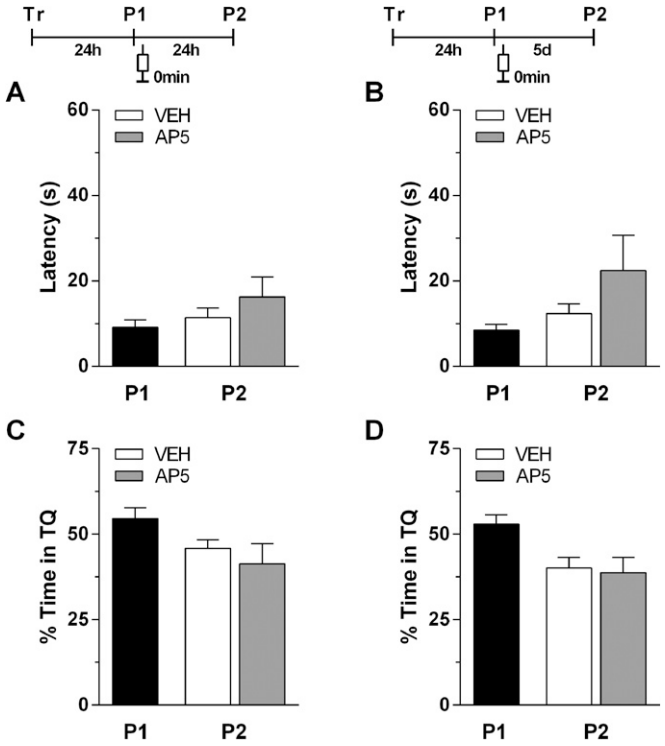

Fig. S1. Intrahippocampal infusion of D(−)-2-amino-5-phosphonopentanoic acid (AP5) immediately after nonreinforced retrieval does not hinder spatial memory retention as measured 24 h or 5 d after reactivation. Animals with infusion cannulae implanted in the CA1 region of the dorsal hippocampus were trained during 5 d in the spatial version of the Morris water maze task (MWM). Twenty-four hours after the last training session, the animals were randomly assigned to one out of four experimental groups and submitted to a 60-s probe test in the absence of the escape platform (P1) (black bar). Immediately after P1, the animals received intrahippocampal infusions of vehicle (VEH) (white bar) or AP5 (25 nmol per side; gray bar). Memory retention was assessed in a second 60-s probe test (P2) carried out 24 h (*A* and *C*) or 5 d after P1 (*B* and *D*). Data are expressed as means (± SEM) of the latency to swim over the previous location of the escape platform (*A* and *B*) or as the percentage of swimming time spent in the target quadrant (TQ) (*C* and *D*). Student *t* test (AP5 vs. vehicle; n = 8–10 per group).

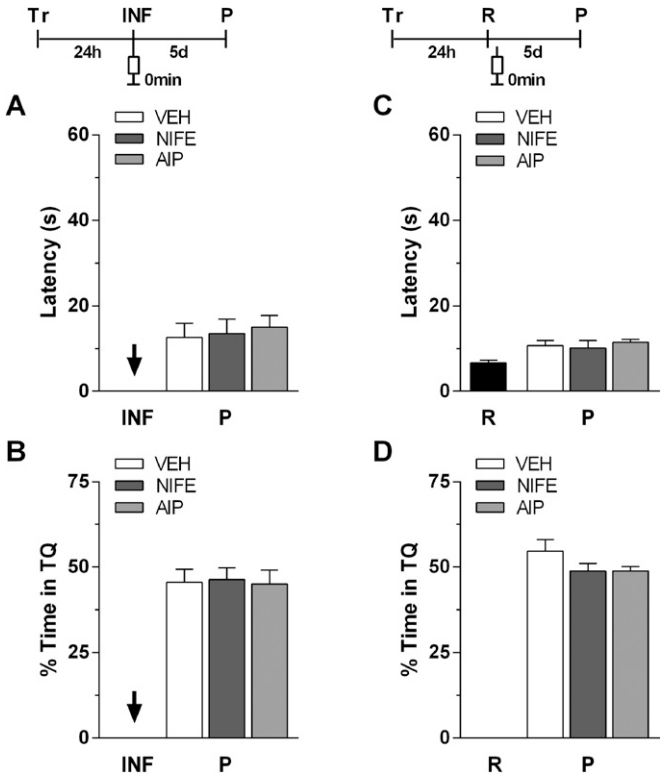

Fig. S2. Late posttraining inhibition of L-voltage–dependent calcium channel (L-VDCC) or calcium/calmodulin-dependent protein kinase (CaMK) II in the absence of relevant behavioral stimuli, as well as immediately after a reinforced retrieval test, does not affect retention of long-term spatial memory. Animals with infusion cannulae implanted in the CA1 region of the dorsal hippocampus were trained during 5 d in the spatial version of the MWM. In the first control experiment, 24 h after the last training session the animals received bilateral intrahippocampal infusions (INF) (black arrow down) of vehicle (VEH) (white bar), nifedipine (NIFE) (10 nmol per side; dark gray bar), or autocamtide-2–related inhibitory peptide (AIP) (1.0 nmol per side; light gray bar). Memory retention was assessed in a 60-s probe test (P) carried out 5 d after INF. The arrowheads indicate the moment of infusion (*A* and *B*). In the second control experiment, 24 h after the last training session, the animals were submitted to a retraining test (R) (black or absent bar) in the presence of the escape platform and immediately after that received bilateral intrahippocampal infusions of vehicle (VEH) (white bar), NIFE (10 nmol per side; dark gray bar), or AIP (1.0 nmol per side; light gray bar). Memory retention was assessed in a probe test in the absence of the escape platform (P) carried out at 5 d after a retraining test (R) (*C* and *D*). Data are expressed as means (± SEM) of the latency to swim over the previous location of the escape platform (*A* and *C*) or as the mean percentage time swimming in the target quadrant (TQ) (*B* and *D*). Dunnett's test after one-way ANOVA (P2 groups vs. vehicle; $n = 8–10$ per group).

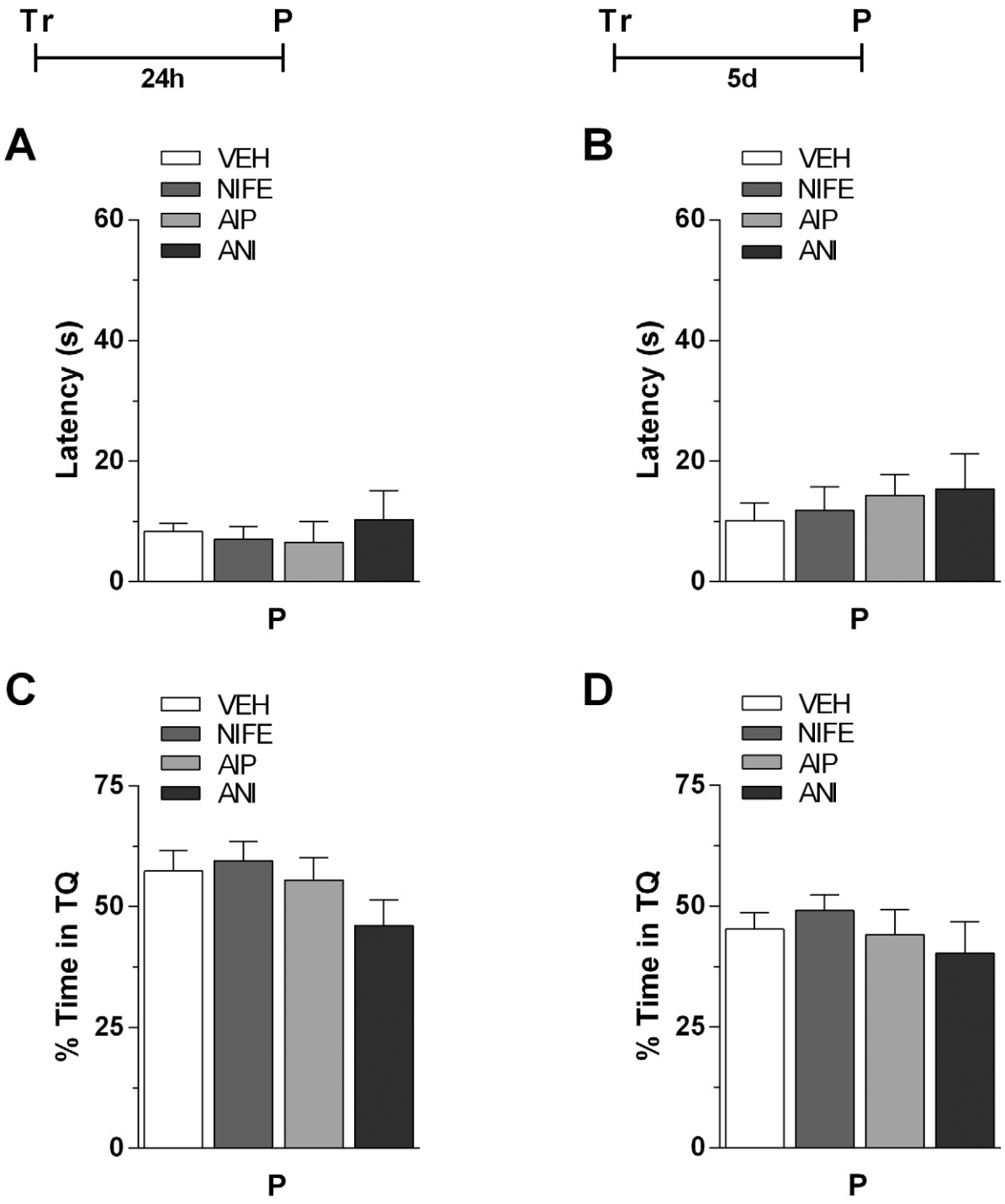

Fig. S3. The amnesic effect of NIFE, AIP, or anisomycin (ANI) was entirely reversible. Animals from experiments depicted in Figs. 1–4 that had received vehicle (VEH) (white bar), NIFE (gray bar), AIP (light gray bar), or ANI (dark gray bar) were retrained during 5 d in the spatial version of the MWM, with the platform localized in the opposite quadrant. Twenty-four hours (*A* and *C*) or 5 d (*B* and *D*) after the last training session, the animals were submitted to a 60-s probe test in the absence of the escape platform (P). Data are expressed as means (± SEM) of the latency to swim over the previous location of the escape platform (*A* and *B*) or as the percentage of swimming time spent in the target quadrant (TQ) (*C* and *D*). Dunnett's test after one-way ANOVA (P2 groups vs. vehicle; $n = 8–9$ per group).

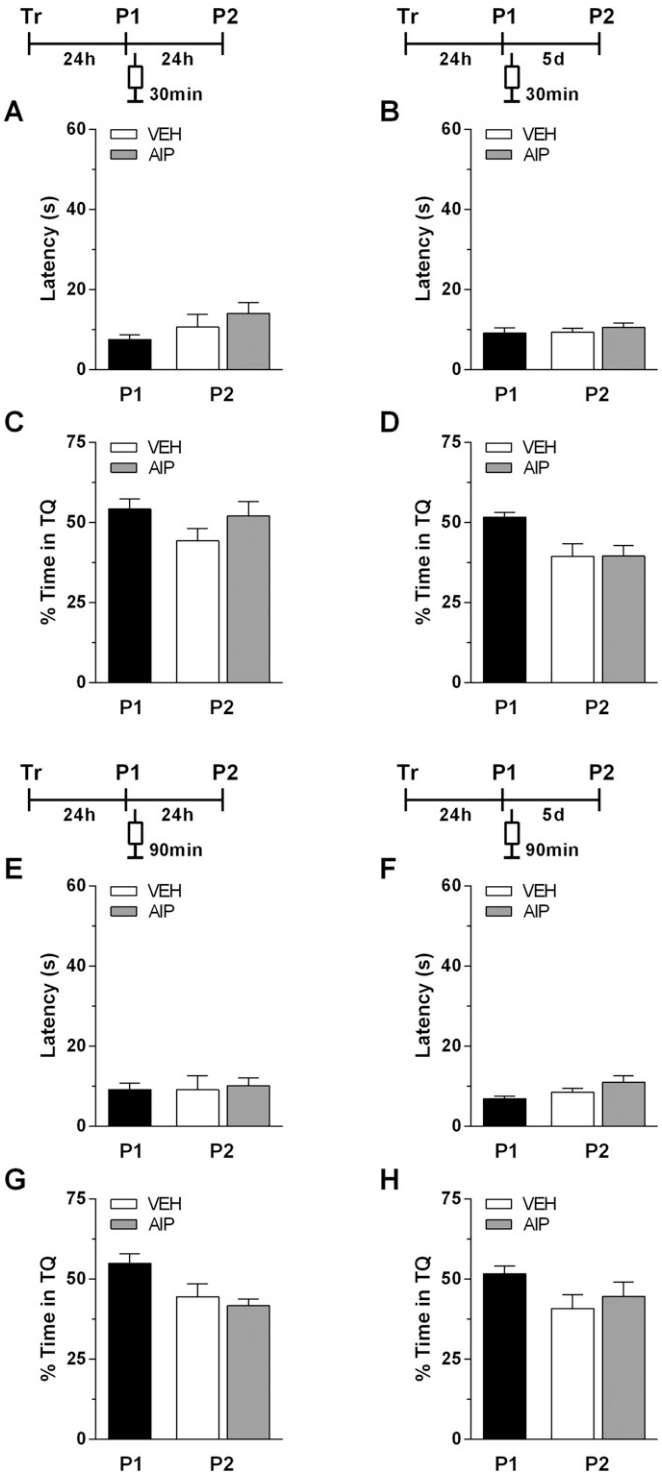

Fig. S4. Intrahippocampal infusion of AIP 30 or 90 min after nonreinforced retrieval does not hinder spatial memory retention as measured 24 h or 5 d after reactivation. Animals with infusion cannulae implanted in the CA1 region of the dorsal hippocampus were trained during 5 d in the spatial version of the MWM. Twenty-four hours after the last training session, the animals were randomly assigned to one of eight experimental groups and submitted to a 60-s probe test in the absence of the escape platform (P1) (black bar). Thirty minutes (*A*–*D*) or 90 min (*E*–*H*) after P1, the animals received intrahippocampal infusions of vehicle (VEH) (white bar) or AIP (1.0 nmol per side; gray bar). Memory retention was assessed in a second 60-s probe test (P2) carried out 24 h (*A*, *C*, *E*, and *G*) or 5 d after P1 (*B*, *D*, *F*, and *H*). Data are expressed as mean (± SEM) of the latency to swim over the previous location of the escape platform (*A*, *B*, *E*, and *F*) or as the percentage of swimming time spent in the target quadrant (TQ) (*C*, *D*, *G*, and *H*). Student *t* test (AIP vs. vehicle; $n = 8$–9 per group).