



**Supplementary Figure 1: Recall of aversive memories activates the endocannabinoid system within the dorsal hippocampus of C57BL/6JOlaHsd mice.**

(a) Experimental procedure: Naive C57BL/6JOlaHsd mice were randomly assigned to three groups (n = 20, each). Mice of the first and the third group were conditioned with a single tone-shock pairing. Mice of the second group received the same tone and shock but in explicitly unpaired manner. Mice of the first and the second groups were exposed to a 180-s tone in the test context at day 1 (d1) after the conditioning procedure. Mice of the third group were just placed into the test context without tone presentation. All mice were killed immediately in the end of the test exposure and the brains were removed within 5 min. Because of the limited detectability of endocannabinoids in tissue samples from the dorsal hippocampus, we pooled the samples of 5 animals in order to obtain a single data point (N), resulting in a sample size of N = 4 per protocol. Measurements of endocannabinoids were performed essentially as described before (Marsicano et al., 2002) (b) Histological section stained with cresyl violet depicting the dissected area of the dorsal hippocampus (circle indicates the inner diameter of the brain puncher). (c) Recall of auditory-cued fear memory caused an increase in anandamide (AEA;  $F_{2,11} = 12.4$ ,  $P = 0.003$ ) and 2-arachidonoylglycerol (2-AG;  $F_{2,11} = 4.8$ ,  $P = 0.038$ ) levels. \*  $P < 0.05$ , \*\*  $P < 0.01$  (1-way ANOVA followed by Newman-Keuls post-hoc test).