Author Correction: Endocannabinoids disinhibit the ventral tegmental nucleus of Gudden to dorsal premammillary nucleus pathway to enhance escape behavior following learned threat experience

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Ruikai Chai, Nawen Wang, Jinlu Nie, Zongyi Xu, Shuqian Zhang, Suixin Deng, Rongxin Wang, Mu Li, Xinyi Gao, Ruijie Geng, Haibin Li, Lei Li, Hebi Wu, Zhiming Li, Tian-Lin Cheng, Xiao-Hong Xu, Yousheng Shu, Huilin Hong, Xiao Huang & Weisheng Wang

Following publication of this article, some patch clamp recording raw data were found to be in error, resulting in mistakes in Fig. 3j, Supplementary Fig. 5c–f and associated Source data. In the Results section "eCB system controls plasticity at VTg-PMd synapses and behavioral adaptation", the original sentence reading "We first measured the effect of single or five-time 2MT exposure conditioning on VTg^{PV} neurons' excitability (Supplementary Fig. 5a) and found no difference among groups (Supplementary Fig. 5b–f)" has now been amended and extended as follows: "We first measured the effect of single or five-time 2MT exposure conditioning on VTg^{PV} neurons' excitability (Supplementary Fig. 5a). Since we recorded the membrane potentials while holding the cell at about −45 to −60 mV with no spontaneous firing (already in the method). Although baseline membrane potentials differed among the various groups (Supplementary Fig. 5d), this only reflects variations in fundamental intrinsic and extrinsic electrophysiological properties. Crucially, when we applied simulated current injection to test excitability, the number of action potentials elicited showed no difference among groups (Supplementary Fig. 5c), indicating that the level of cellular excitability remained unchanged. These data suggest that learning-induced suppression of VTg^{PV} neurons does not affect their excitability at the somatic level, but could be due to changes at the $VTg^{PV} \rightarrow PMd^{CCK}$ synapses." The text, figures and source data are now amended in the HTML and PDF versions of the article.