Correction

Correction: Trantham-Davidson et al., Chronic Alcohol Disrupts Dopamine Receptor Activity and the Cognitive Function of the Medial Prefrontal Cortex

In the article "Chronic Alcohol Disrupts Dopamine Receptor Activity and the Cognitive Function of the Medial Prefrontal Cortex" by Heather Trantham-Davidson, Elizabeth J. Burnett, Justin T. Gass, Marcelo F. Lopez, Patrick J. Mulholland, Samuel W. Centanni, Stan B. Floresco, and L. Judson Chandler, which appeared on pages 3706–3718 of the March 5, 2014 issue, the sign of one connection in the summary in Figure 8 was incorrect. A corrected version of the figure and corresponding figure legend is presented here. This correction does not affect any conclusions from the study.

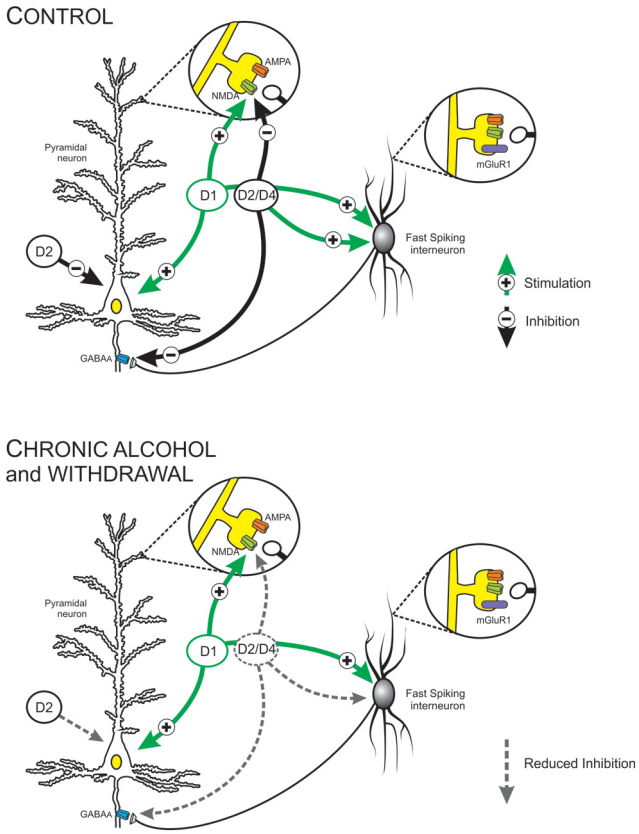

Figure 8. A schematic summary depicting the effects of CIE exposure on the intrinsic and synaptic actions of DA receptors on pyramidal neurons and fast-spiking interneurons in Layer 5 of the medial PFC. Top, Under control conditions, activation of D1 receptors enhanced the intrinsic firing of both cell types and facilitated glutamatergic neurotransmission by enhancing the activity of NMDA receptors but had no effect on AMPA currents. In contrast, D2 receptors inhibited the intrinsic firing of pyramidal neurons and enhanced firing of fast-spiking interneurons. D4 receptor stimulation had no effect on the intrinsic activity of pyramidal neurons but increased evoked firing of fast-spiking interneurons. Both D2 and D4 receptor stimulation inhibited synaptic NMDA and $GABA_A$ currents on pyramidal neurons. Bottom, Following CIE exposure and withdrawal, D1 receptor-mediated responses were not affected, whereas the modulatory actions of D2 and D4 receptors on intrinsic and synaptic activity were significantly attenuated. Also depicted is activation of mGluR1 receptors enhances evoked firing of fast-spiking interneurons similar to D4 receptors, but this effect is not altered by CIE exposure. In light of the important role of fast-spiking interneurons on controlling and organizing prefrontal network activity, mGluR1 receptors may thus represent a novel pharmacological target to reverse the effects of CIE on the prefrontal cortex.

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