Correction

In the article "G-Protein-Coupled Receptor Modulation of Striatal $Ca_V1.3$ L-Type Ca^{2+} Channels Is Dependent on a Shank-Binding Domain," by Patricia A. Olson, Tatiana Tkatch, Salvador Hernandez-Lopez, Sasha Ulrich, Ema Ilijic, Enrico Mugnaini, Hua Zhang, Ilya Bezprozvanny, and D. James Surmeier, which appeared on pages 1050–1062 of the February 2, 2005 issue, the right panels of Figures 1*C* and 4*C* are not correct and have been replaced. The errors in the printed images were introduced when the figures were composed and do not affect any of the summaries or figure legends. However, revised versions of Figures 1 and 4 are printed here.

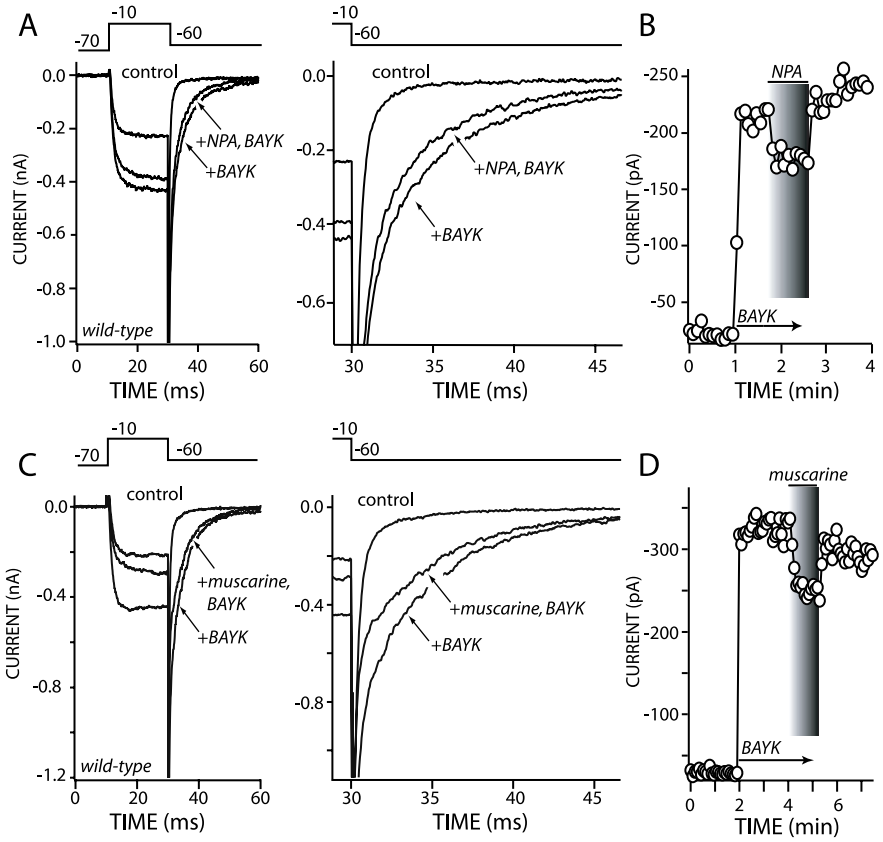

Figure 1. D_2 dopaminergic and M_1 muscarinic receptors inhibit L-type calcium channels in medium spiny neurons. *A*, Control current traces from a dorsal striatal medium spiny neuron from a wild-type mouse: current elicited by S(−)-BayK 8644 (BAYK; 1 μM) alone or with NPA (10 μM). *B*, Measuring the tail current 5 ms after the step isolated L-type current, providing the time course shown. NPA inhibited L-type tail current by 18%. *C*, Control current traces from a dorsal striatal medium spiny neuron from a wild-type mouse: current elicited by BayK 8644 (1 μM) alone or with (+)-muscarine chloride (muscarine; 2 μM). *D*, Measuring the tail current 5 ms after the step isolated L-current, providing the time course shown. Muscarine application inhibited L-type tail currents by 25%.

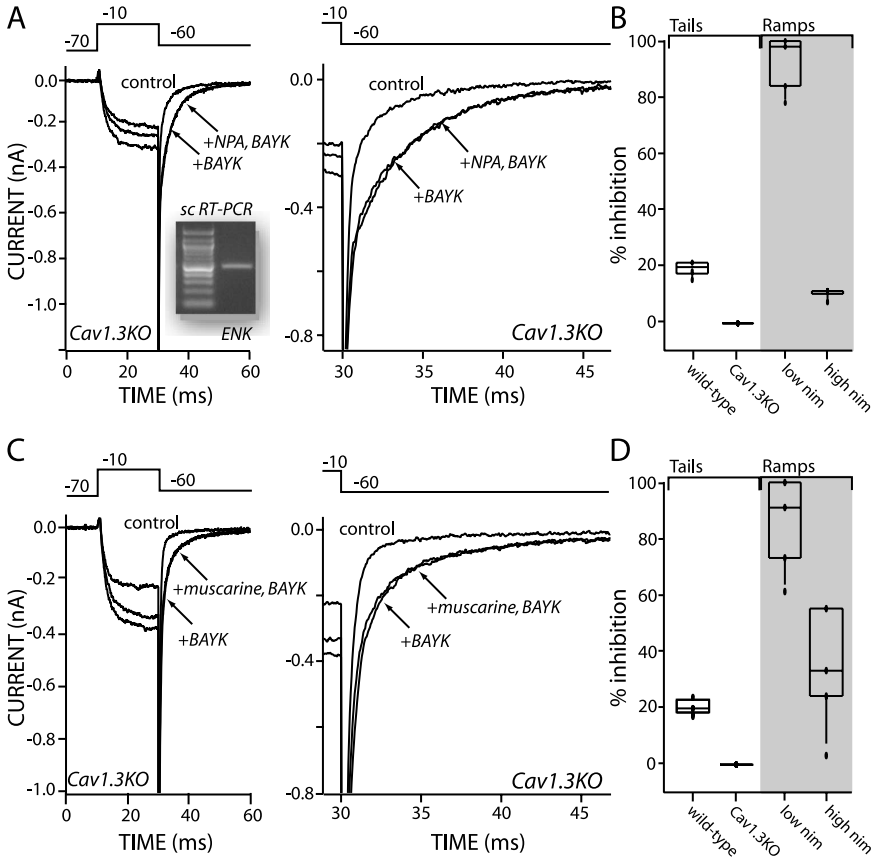

Figure 4. D_2 dopaminergic and M_1 muscarinic receptor modulations of L-type calcium channels are lost in neurons from a $Ca_V1.3$ knock-out. *A*, Current traces from a medium spiny neuron from a $Ca_V1.3$ knock-out mouse. Experimental conditions were the same as in Figure 1. NPA had no effect on the L-type tail current. The inset shows scRT-PCR confirmation that the neuron expressed enkephalin, a marker for D_2 receptor expression. *B*, Box plot illustrates the difference in percentage inhibition of L-type tail currents by D_2 receptor activation in medium spiny neurons from wild-type ($n = 3$; median, 18%) and $Ca_V1.3$ knock-out ($n = 6$; median, 0%) mice ($p < 0.05$; Mann–Whitney). Also shown is the box plot summary of experiments examining the reduction in peak ramp current by NPA (10 μM) after the addition of low (low nim; 1 μM; $n = 4$) and high (high nim; 10 μM; $n = 4$) nimodipine. The amplitude of the modulation in low and high nimodipine divided by the control modulation is plotted; values close to 100 indicate no effect on the modulation, whereas values close to 0 indicate occlusion. *C*, Current traces from a dorsal striatal medium spiny neuron from a $Ca_V1.3$ knock-out mouse. Experimental conditions were the same as Figure 1. Muscarine had no effect on the L-type tail current. *D*, Box plot illustrates the difference in percentage inhibition of L-type tail currents by M_1 receptor activation in medium spiny neurons from wild-type ($n = 5$; median, 20%) and $Ca_V1.3$ knock-out ($n = 5$; median, 0%) mice ($p < 0.05$; Mann–Whitney). Also shown is the box plot summary of experiments examining the reduction in peak ramp current by muscarine (10 μM) after the addition of low (low nim; 1 μM; $n = 5$) and high (high nim; 10 μM; $n = 5$) nimodipine. The amplitude of the modulation in low and high nimodipine divided by the control modulation is plotted; values close to 100 indicate no effect on the modulation, whereas values close to 0 indicate occlusion.