Correction

In our article "Chronic Haloperidol Promotes Corticostriatal Long-Term Potentiation by Targeting Dopamine D2L Receptors," by Diego Centonze, Alessandro Usiello, Cinzia Costa, Barbara Picconi, Eric Erbs, Giorgio Bernardi, Emiliana Borrelli, and Paolo Calabresi, which appeared on pages 8214–8222 of the September 22, 2004 issue, mistakes were made in preparing the figures. In several cases, incorrect control data points were used corresponding to pretetanus values of the LTP experiments. We have reanalyzed each experiment using the correct values. The statistical significance and the conclusion of the study were not affected and thus remain valid. However, revised versions of Figures 2–6, showing the corrected values, are printed here. We apologize for the inconvenience to readers of the *Journal*.

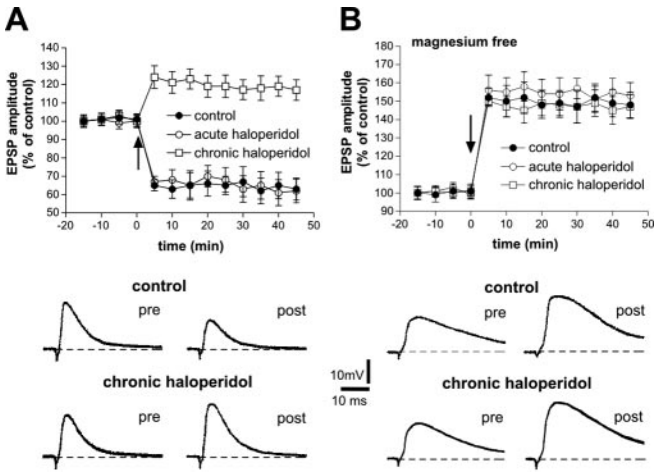

Figure 2. Chronic, but not acute haloperidol treatment alters corticostriatal synaptic plasticity. *A*, The graph shows that in the presence of 1.2 mM external magnesium (LTD protocol), HFS (arrow) of corticostriatal fibers induced LTD of glutamatergic transmission in striatal neurons recorded from sham-treated (control) and 1-d-treated (acute haloperidol) rats. Conversely, the same stimulation protocol induced LTP in rats treated chronically (20 d) with haloperidol. *B*, HFS delivered in the absence of magnesium ions from the bathing solution (LTP protocol) induced LTP in sham-treated, acute, and chronic haloperidol-treated rats. Traces in the bottom part of the figure are intracellular recordings from single experiments showing EPSPs 10 min before (pre) and 30 min after (post) HFS in control and chronic haloperidol-treated rats, in the presence (*A*) or in the absence (*B*) of external magnesium. In this figure and in the following ones, the resting membrane potential and input resistance of the recorded neurons were constant and ranged between -83 ± 5 mV and 48 ± 16 MΩ.

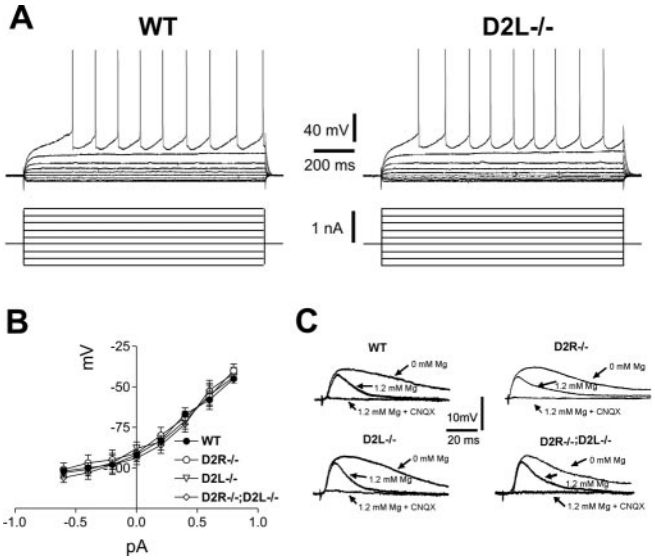

Figure 3. Intrinsic and synaptic properties of striatal spiny neurons of WT and D2 receptor mutants. *A*, Electrophysiological traces showing that in WT and D2L−/− neurons, the membrane responses to hyperpolarizing and depolarizing current steps were similar. *B*, Graph showing that the current–voltage relationships of striatal neurons recorded from WT, D2R−/−, D2L−/−, and D2R−/−;D2L−/− mice were virtually coincident ($n = 15$ for each genotype). *C*, The electrophysiological traces are cortically evoked EPSPs recorded from WT, D2R−/−, D2L−/−, and D2R−/−;D2L−/− striatal neurons in the presence and in the absence of magnesium ions and in the presence of magnesium plus 10 μM CNQX, an antagonist of non-NMDA glutamate receptors. Note that in the absence of external magnesium, EPSP amplitude and duration increased in the four genotypes, as an expression of the contribution of glutamate NMDA receptors in corticostriatal transmission.

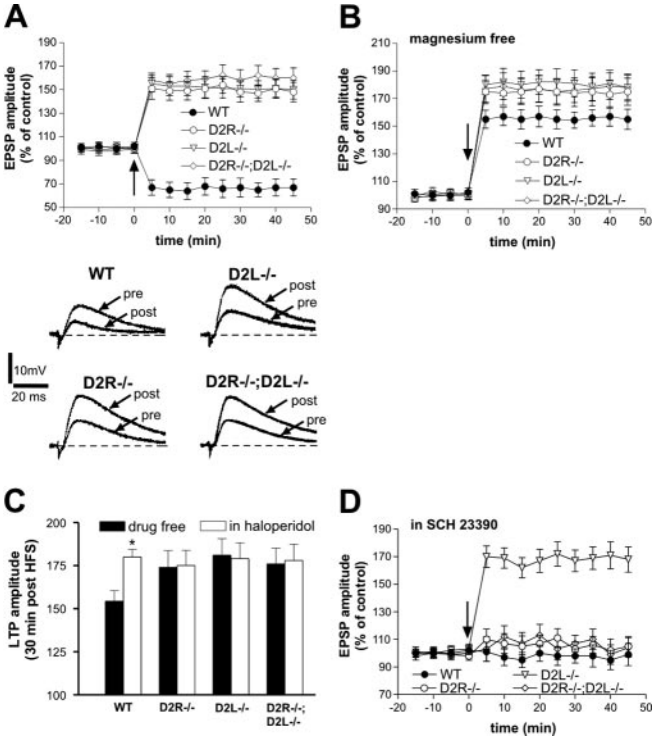

Figure 4. Dopamine-regulated synaptic plasticity in WT and mutant mice. *A*, The graph shows that HFS (arrow) of corticostriatal fibers induced LTD of glutamatergic transmission in WT neurons recorded in the presence of 1.2 mM external magnesium (LTD protocol). In contrast, the same stimulation protocol induces LTP in D2R−/−, D2L−/−, and D2R−/−;D2L−/− striatal neurons. Superimposed traces in the bottom part of the figure are intracelluar recordings from single experiments showing EPSPs 10 min before (pre) and 30 min after (post) HFS in WT, D2R−/−, D2L−/−, and D2R−/−;D2L−/− striatal neurons. *B*, HFS delivered in the absence of magnesium ions from the bathing solution (LTP protocol) induced LTP in WT, D2R−/−, D2L−/−, and D2R−/−;D2L−/− striatal cells. Note that LTPs recorded from the three mutants had higher amplitude than the one recorded from WTs. *C*, The histogram shows that chronic haloperidol treatment enhanced the amplitude of corticostriatal LTP in WT mice but not in the three D2 receptor mutants. All of these experiments were performed in the absence of magnesium ions. *$p < 0.01$ at 30 min after HFS. *D*, This graph shows that preincubation with the D1-like receptor antagonist SCH 23390 (10 μM) prevented LTP in WT, D2R−/−, and D2R−/−;D2L−/− neurons but not in D2L−/− cells. In this graph, the LTP-inducing protocol in WT cells consisted of the application of HFS in the absence of magnesium ions. In contrast, in the three mutants, HFS was delivered either in the absence (at least 3 cells per group) or in the presence (at least 3 cells per group) of magnesium ions. Because the experiments gave similar results, the data were pooled together for each genotype.

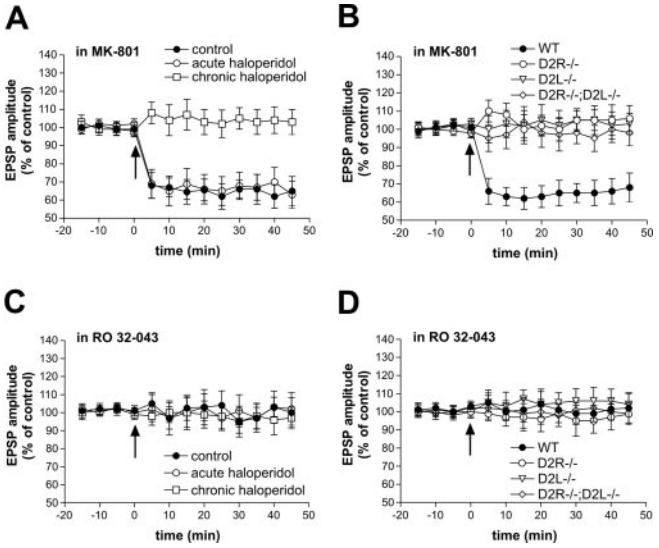

Figure 5. LTPs from haloperidol-treated rats and from D2 receptor mutant mice are sensitive to NMDA receptor and PKC blockade. *A*, *B*, Graphs showing that preincubation with the glutamate NMDA receptor antagonist MK-801 (30 μM) blocked LTP induction in control and chronic haloperidol-treated rats (*A*) as well as in WT and D2R−/−, D2L−/−, and D2R−/−;D2L−/− striatal neurons (*B*). Note that in control rats and in WT mice, blockade of NMDA receptors not only prevented LTP but also favored the emergence of LTD, *C*, *D*, Intracellular application of the PKC inhibitor RO 32-043 prevented LTP in control and chronic haloperidol-treated rats (*C*) as well as in WT and mutant mice (*D*). In the four graphs, the LTP-inducing protocol in WT cells consisted of the application of HFS in the absence of magnesium ions. In contrast, in the three mutants, HFS was delivered either in the absence (at least 4 cells per group and experimental condition) or in the presence (at least 4 cells per group and experimental condition) of magnesium ions. Because the experiments gave similar results, the data were pooled together for each genotype.

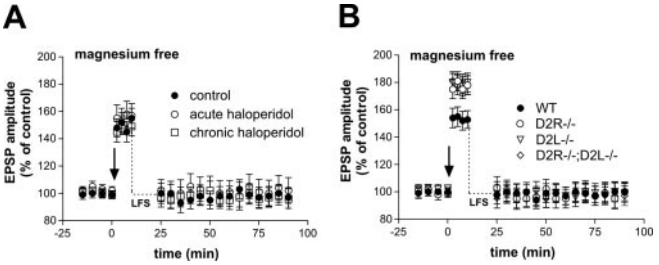

Figure 6. Synaptic depotentiation is unaffected by chronic haloperidol treatment or genetic manipulations of D2 receptors. *A*, *B*, After the induction of LTP by HFS, a 10 min application of LFS (2 Hz) of corticostriatal terminals reversed this form of synaptic plasticity in control and chronic haloperidol-treated animals (*A*) as well as in WT and in the three genotypes of D2 receptor mutant mice (*B*). These experiments were performed in the absence of magnesium ions from the external solution, because synaptic depotentiation is regulated by NMDA receptors.