SUPPLEMENTAL MATERIAL

Supplemental Figure 1. Anesthesia does not compromise the actions of dopamine receptor antagonists. *In vivo* recordings of dopamine neurons in the substantia nigra *pars compacta* (n = 3 neurons; 1 per animal) were used to test whether the dopamine receptor antagonists, SCH-23390 and raclopride, acted predictably in animals under general anesthesia. Dopamine neurons (2 of 3 were verified as such after juxtacellular labeling with Neurobiotin [Nb], and co-localization of Nb with tyrosine hydroxylase [TH]; see inset and Ungless et al., 2004) exhibited stereotypical slow and regular single-spike firing (ranges: 2.3-3.0 Hz, CVs of 0.08-0.30; see inset, x) and long-duration action potentials (1.19-1.31 ms from start to first trough; Ungless et al., 2004) during baseline recordings. Systemic administration of the mixed D1/D2 receptor agonist apomorphine (0.05 mg/kg, s.c.) led to a prolonged reduction in the activity of dopamine neurons (0-21.5% of baseline firing rates), in agreement with the expected actions of apomorphine at somatodendritic D2 'autoreceptors' (Bunney et al., 1973; Sanghera et al., 1984; Lacey et al., 1987), as well as more modest actions at D1 receptors on striatonigral neurons and elsewhere (Scarnati et al., 1980; Napier et al., 1986; Carlson et al., 1987; Shi et al., 1997). This decreased activity was rapidly reversed by administration of the receptor antagonists SCH-23390 (0.5 mg/kg, s.c.) and raclopride (2 mg/kg, i.p.). The antagonists also led to a 'rebound' increase in spontaneous activity (149-190% of baselines), as described previously (Sanghera al., 1984; Pucak and Grace, 1994). A subsequent dose of apomorphine had no effect on firing (Pucak and Grace, 1994). These data indicate that the receptor antagonists blocked central dopamine receptors, and thus acted as predicated, in our anesthetized animals.

Supplemental References

(To accompany Supplemental Figure 1)

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