

FLUCTUATIONS IN SOMATOSENSORY RESPONSIVENESS AND BASELINE FIRING RATES OF NEURONS IN THE LATERAL STRIATUM OF FREELY MOVING RATS: EFFECTS OF INTRANIGRAL APOMORPHINE

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Abstract—Somatosensory responsiveness and baseline firing rates of 102 striatal neurons were studied in freely moving rats. For individual neurons, mean levels of responsiveness and baseline firing fluctuated unpredictably in direction and magnitude and independently of each other throughout an experiment.

Following microinjections of apomorphine into the substantia nigra, which were used as a means of reducing nigral output activity, the magnitude of fluctuations in striatal somatosensory responsiveness significantly increased, while the magnitude of fluctuations in baseline firing was unaltered.

The receptive zones of 54 neurons studied in control experiments remained stable, whereas receptive zones changed in 12 of 25 neurons studied after apomorphine microinjection.

Normal nigrostriatal dopamine transmission appears to selectively restrict the magnitude of fluctuations in responsiveness of striatal neurons to corticostriatal synaptic input and may exert additional control over afferent projections from cutaneous receptive zones to these neurons. © 2004 IBRO. Published by Elsevier Ltd. All rights reserved.

Key words: basal ganglia, corticostriatal, single neuron recording, substantia nigra, dopamine, microinjection.

Nigrostriatal influences regulating the processing of information flow from neocortex, thalamus, and other brain structures to the basal ganglia are mediated monosynaptically by dopaminergic neurons of substantia nigra pars compacta (SNc), and polysynaptically by GABAergic neurons of substantia nigra pars reticulata (SNr). The latter project to nonspecific midline-intralaminar nuclei of the thalamus, which, in turn, send abundantly collateralized axons to the striatum and to striatal afferent sources in cortex (Deschênes et al., 1996; Mengual et al., 1999; Gerfen, 2000; Nakano, 2000; Tsumory et al., 2000).

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Abbreviations: APO, apomorphine; PSTH, peristimulus time histogram; SN, substantia nigra; SNc, substantia nigra pars compacta; SNr, substantia nigra pars reticulata; RZ, receptive zone.

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The somatosensory responsiveness of striatal neurons in behaving animals (Carelli and West, 1991; Brown, 1992; Cho and West, 1997) provides an analytical tool for studying the normal operation of mechanisms governing striatal output activity, given the evidence that phasically responsive neurons are medium spiny, projection neurons (Kimura, 1990).

Our aim was to examine, in freely moving rats, the time course of alterations in somatosensory responses and baseline firing of lateral striatal neurons in the normal state and during the transient lowering of general nigral output activity and nigrostriatal dopamine transmission. This was accomplished by microinjecting small doses of the dopamine receptor agonist, apomorphine (APO), into the substantia nigra (SN) in order to partially model the actions of intrinsic nigral dopamine release (Geffen et al., 1976; Cheramy et al., 1981). It was assumed that intranigral APO acting via D2 receptors (Seeman, 1995) on SNc neurons, which are highly sensitive to direct application of dopamine or APO (Skirboll et al., 1979; Okuyama et al., 1986; Hyland et al., 2002), could depress monosynaptic nigrostriatal dopamine transmission that would be reflected in the evoked activity of striatal neurons (Freund et al., 1984). It was also assumed that intranigral APO would influence GABAergic neurons of SNr via D₁-receptors (Seeman, 1995) on striato-nigral terminals (Martin and Waszczak, 1994; Radnikow and Misgeld, 1998) that could produce, in turn, changes in baseline activity of striatal neurons.

EXPERIMENTAL PROCEDURES

Experiments were conducted in accord with guidelines of the Institutional Animal Care and Use Committee of Rutgers University, the National Institutes of Health, and United States Department of Agriculture, including minimizing the number of animals used, and minimizing pain and suffering.

Data were collected from 26 male Long-Evans rats (275–310 g; Charles River Laboratories, Wilmington, MA, USA). Animals were surgically prepared for recording in the awake, unrestrained state by implantation of microdrive assembly (Josef Biela Engineering, Anaheim, CA, USA) as described previously (Carelli and West, 1991).

During surgery, two bases (the first for attaching a microdrive containing a tungsten microelectrode [10–12 MΩ; Haer, Brunswick, ME, USA] and the second for attaching a removable intracerebral microinjector [Prokopenko et al., 1997]) were cemented to the skull above holes centered: 1) over lateral striatum, either side, perpendicular to level skull and 2) over SN at an angle of 16.5° from vertical, toward midline, ipsilateral to the microdrive base.

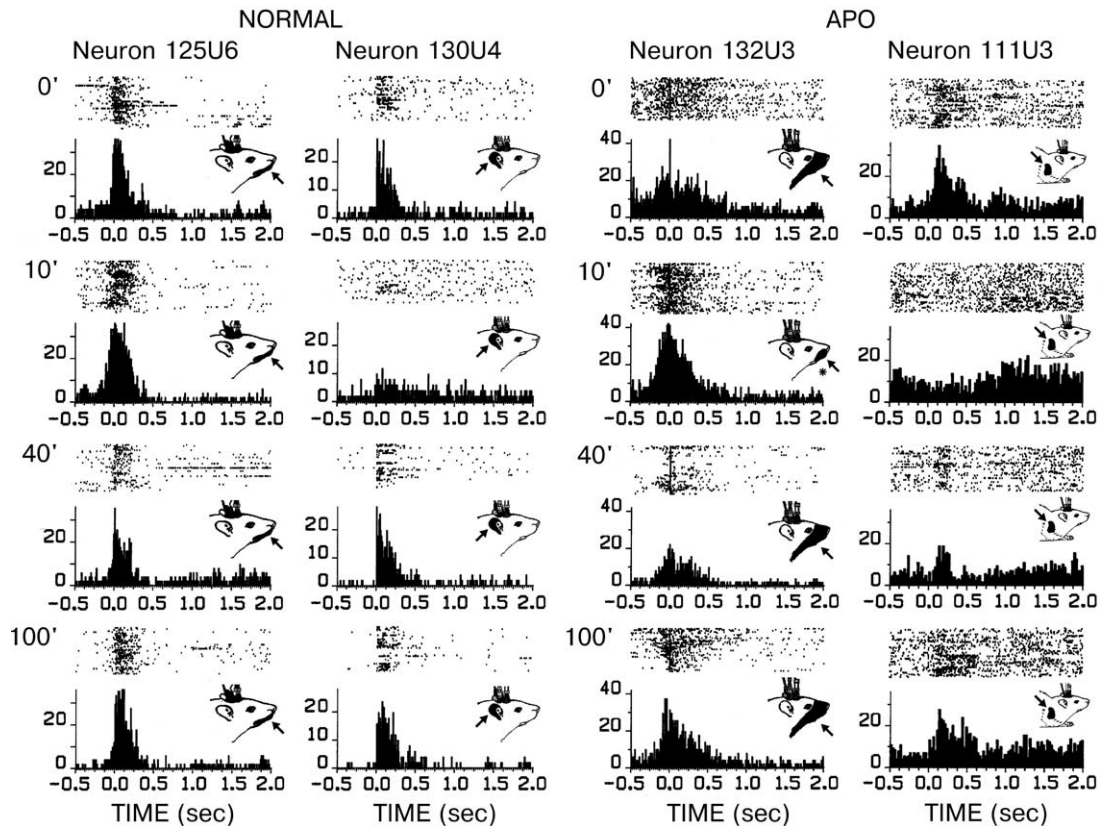


Fig. 1. Changes in baseline firing and somatosensory responsiveness. Each column represents the data of a single striatal neuron. Left two columns: NORMAL group; right two columns: APO group. Each row (raster display and PSTH) depicts the neuron's activity recorded at one of the following time points: 0 min (onset of the experiment), 10 min, 40 min or 100 min. For a given row, 50 stimulus events contributed to the displayed firing patterns. Insets indicate position of cutaneous RZ (arrow) for that neuron. Asterisk in third column depicts shift in the neuron's RZ following APO injection.

Recording sessions began 1 week after surgery. Extracellularly recorded action potentials of single neurons were amplified, filtered (bandpass 500–8000 Hz) and two-stage discriminated (West, 1998). For each striatal neuron that exhibited spontaneous activity, impulses were delivered in parallel to a computer and to the experimenter's headphones during visually monitored testing of responsiveness to mechanical cutaneous stimulation. Stimulation was delivered via a handheld probe (2 mm in diameter), calibrated to deliver 2–4 g of force. The probe traveled a distance up to 3 cm in 0.15 s. Monotonous, unidirectional, stable stimuli were presented 5–10 s apart. Instantaneously on contact of the bipolar probe with the animal, a DC pulse was delivered to the computer to generate a time-stamp, or node, of the approximate onset of each stimulus.

Neurons were selected if they exhibited cutaneous responsiveness and little or no correlation with active movements (e.g. neurons responsive to cutaneous stimulation on the head; Cho and West, 1997), in order to avoid difficulties in interpreting neural data in the APO group, in which abnormal movement might produce altered somatosensory feedback. For each neuron, the approximate borders of the receptive zone (RZ) in which cutaneous stimulation produced stable, repetitive responses, and any changes of this zone during the session, were recorded. Ten to 20 stimuli were applied outside the RZ to verify unresponsiveness.

During each experiment, the activity of one neuron was tested at four periods. The first was termed the "time 0" period. The three remaining periods occurred at 10, 40 and 100 min after 1) the end of the time 0 period, or 2) the start of microinjection. Peristimulus time histograms (PSTHs) and raster displays were constructed around stimulus onset (50 stimuli were delivered over approxi-

mately 6 min at each time period). Evoked firing rates (impulses/s) were calculated during the "evoked response epoch" defined for each neuron by visual inspection of the time 0 PSTH. Baseline firing rates were calculated from each PSTH during the interval from –0.5 to –0.15 s before stimulus onset.

A removable microinjector was used to deliver fluids (1 μ l, 0.15–0.20 μ l per minute) to the SN. The interval between two experiments for each animal was at least 4 days. Each rat received nine or fewer microinjections (one injection per experiment), and histological examination revealed no damage to SN.

Single intracerebral microinjections of APO (R(-)-Apomorphine HCl; RBI (Nantick, MA, USA); 2 μ g/ μ l; pH=7.0) solution were given in each APO experiment (APO group; $N=48$ neurons). The drug was dissolved in saline buffer (Dulbecco et al., 1979) with a supplement of 0.1% weight/volume ascorbic acid. Microinjections (same volume) of clear Dulbecco's saline buffer were administered in the second group of experiments ($N=30$ neurons). In the third group, injection manipulations with the empty device on the rat's head were simulated ($N=24$ neurons). The latter two groups did not statistically differ from each other in any parameter, which made it possible to combine them into one NORMAL group. Each animal participated in APO and NORMAL experiments.

After the last experiment, each animal was anesthetized with sodium pentobarbital (150 mg/kg). A low-impedance insulated wire (200 μ m diameter) was placed in the following: 1) in the same location as that at which a particular neuronal recording had been obtained and 2) through the microinjector base at the depth of the lowered cannula. Electrolytic lesions (0.03 mA; 10 s) were made

at the wire tips. Procedures for perfusion, histology, and identification of locations of electrolytic lesions were described previously (Carelli and West, 1991).

Multiple *t*-tests, ANOVAs, and χ^2 were used. Differences were judged significant at the 0.05 level.

RESULTS

Neuronal activity

The firing of 102 neurons responsive to somatosensory cutaneous stimulation was studied in 102 experiments. According to histological verification, all neurons were recorded from the dorsolateral striatum (Bregma AP 0.5–1.5 mm, ML 3.0–4.0 mm, and DV 3.0–4.6 mm from cortex surface) and the microinjector cannula tips were positioned in the SN (Bregma AP –4.8 to –5.8 mm, ML 1.8–2.5, DV 6.8–7.5).

At the beginning of experiments, NORMAL and APO groups showed no significant differences in baseline firing or responsiveness ($P > 0.05$). The distribution of mean baseline and response firing rates at time 0 is shown in Fig. 2A.

Baseline firing rate

Spontaneous, non-periodic changes (fluctuations) in baseline firing (mean impulses/s) of both polarities (increases or decreases) were observed (Fig. 1). The magnitudes of these fluctuations over time are shown in Fig. 2B. Mean baseline activity tended to decrease non-significantly in both groups at 10 min and 40 min and to be slightly decreased or unchanged at 100 min. Separation of these fluctuations into increases versus decreases revealed no differences between mean baseline firing in NORMAL and APO groups at any time point (Fig. 2C). The proportions of neurons exhibiting changes in baseline firing and responsiveness are shown in Table 1.

Fluctuations in neuronal responsiveness

Fluctuations were also observed in neuronal responsiveness evoked by somatosensory cutaneous stimulation over time in all neurons studied. These fluctuations exhibited considerable variability among neurons in direction and magnitude (Fig. 2B, C). Averaged together, the mean magnitude of responsiveness showed insignificant decreases throughout the experiment (Fig. 2B). In comparison with the NORMAL group, the most marked effect of intranigral APO microinjection was the significant increase in magnitude of both positive and negative fluctuations in responsiveness to cutaneous stimulation (Figs. 2 and 3).

Each neuron recorded at all four time points in both groups ($N=81$) was rated as to whether its baseline and evoked firing changed in the same direction at each time point relative to the preceding time point. Thus, a neuron could receive a rating of *same* at none, one, two, or all three time points. Only 26% of neurons were rated as *same* at all three time points in the NORMAL group (i.e. chance level), compared with 19% in the APO group, which was not significantly different between groups. Thus, fluctuations in baseline and evoked firing of individual neu-

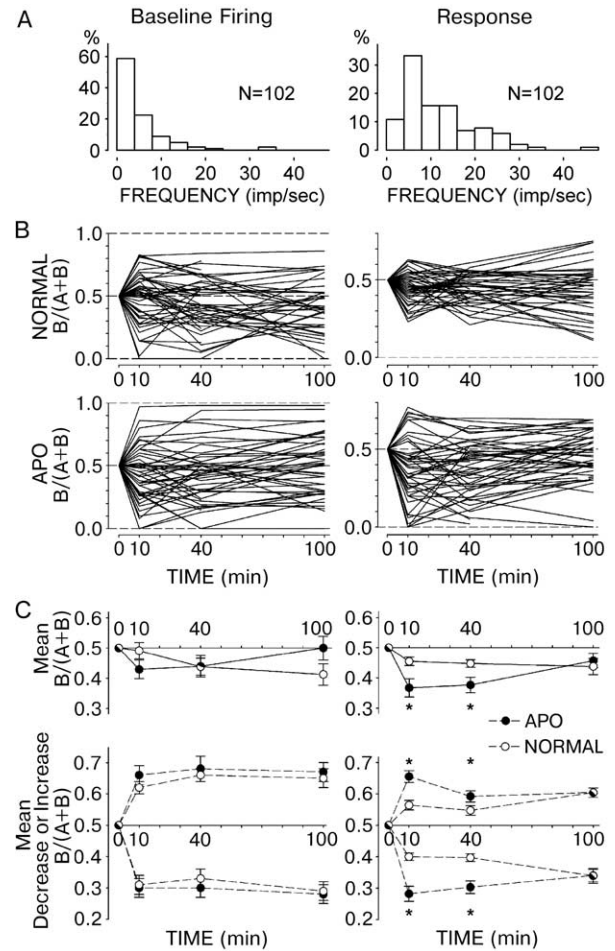


Fig. 2. Baseline firing (left column) and somatosensory responsiveness (right column). (A) Distribution of mean baseline and response firing rates of all neurons registered at time 0. (B) Time course of fluctuations in baseline firing and somatosensory responsiveness of individual neurons. Calculated changes in firing $[B/(A+B)]$ are displayed as a function of time. Each solid line represents a single neuron. Values >0.5 indicate increases; values <0.5 indicate decreases, relative to time zero. Time course of changes in baseline firing and somatosensory responsiveness of individual neurons in NORMAL group (top row) and APO group (bottom row). The formula used was $[B/(A+B)]$, in which A was the firing rate of the neuron at time 0 min and B was the firing rate of that neuron at subsequent times: 10 min, 40 min or 100 min. A value equal to 0.5 is indicative of no change between the "B" and "A" periods. (C) Time course of mean values of baseline and evoked neuronal activity. Mean calculated change in firing $[B/(A+B)]$ is displayed as a function of time (empty circles, NORMAL group; filled circles, APO group). Top row: mean values of neuronal activity averaged across all neurons. Bottom row: mean values of increases (>0.5) and decreases (<0.5), averaged separately at each time point. Asterisks indicate significant differences between NORMAL and APO groups.

rons varied independently in their directions and magnitudes (e.g. Figs. 1 and 3).

In addition, three striatal neurons in the APO group responded to stimulation of two spatially separate RZs. The time course and polarity of changes in the same neuron's responsiveness to stimulation of each RZ were independent.

Table 1. Proportions of neurons exhibiting changes in baseline firing and responsiveness^a

Activity	Baseline firing						Responsiveness					
	NORMAL		APO		NORMAL		APO					
Post time 0 interval (min)	10	40	100	10	40	100	10	40	100	10	40	100
Increased firing (%)	31	20	12	16	16	24	17	16	13	11	12	18
	(57.4)	(40.8)	(31.6)	(33.3)	(34.0)	(54.5)	(31.5)	(32.7)	(34.2)	(22.9)	(25.5)	(40.9)
No changes (%)	1	2	2	2	3	1	2	1	2	0	0	2
	(1.9)	(4.1)	(5.3)	(4.2)	(6.4)	(2.3)	(3.7)	(2.0)	(5.3)	(0.0)	(0.0)	(4.55)
Decreased firing (%)	22	27	24	30	28	19	35	32	23	37	29	24
	(40.7)	(55.1)	(63.1)	(62.5)	(59.6)	(43.2)	(64.8)	(65.3)	(60.5)	(77.1)	(74.5)	(54.55)
Number of tested cells	54	49	38	48	47	44	54	49	38	48	47	44

^a There were no significant differences between NORMAL and APO groups (χ^2 -test of equality; $P > 0.0083$, corrected for six comparisons).

Receptive zones

RZs of 79 neurons were located on the contralateral surface of the animal's head, mainly on the animal's muzzle (71 neurons). The remaining 23 neurons exhibited RZs on

other, contralateral, body parts. Ninety-nine neurons were activated by stimulation of only one RZ, while three neurons exhibited responses to stimulation of two non-neighboring RZs within the same body part.

No changes were observed in the parameters of RZs for neurons in the NORMAL group throughout the time course of the experiment. In contrast, after intra-nigral microinjection of APO, changes in RZ position and/or dimensions were found in 12 of 25 cases tested. In five experiments, the RZ increased in dimensions; in five others it decreased, and in two experiments the RZ changed both its dimensions and location on the body. In these 12 cases, there was no relationship between the polarity of changes in RZ dimensions and the polarity of simultaneous changes in evoked neuronal activity (Fig. 1).

Behavioral changes

Muscular impairment after APO microinjection was observed in five experiments: contralateral akinesia in four cases and an observable decline in muscle tone in the other case. Nonetheless, changes in neuronal baseline firing or evoked activity in these experiments did not differ in any observable way from others in the APO group.

DISCUSSION

Normal baseline and evoked firing

For individual somatosensory-responsive striatal neurons, the fluctuations in baseline firing and responsiveness, reported for the first time here, appear to be normal and natural. They were independent of each other and unpredictable in direction and magnitude during the time course of experiments. This independence suggests that the mechanisms underlying general excitability versus responsiveness to somatosensory cortical input of striatal neurons are largely separate, and remain to be investigated.

Effects of intranigral APO microinjection

The slight, insignificant increase in magnitudes of positive and negative fluctuations in baseline firing between NORMAL and APO groups corroborates the minimal influence of altered intrastriatal dopamine release observed on baseline striatal firing (Abercrombie and Jacobs, 1985). Our finding suggests a minor indirect influence on somato-

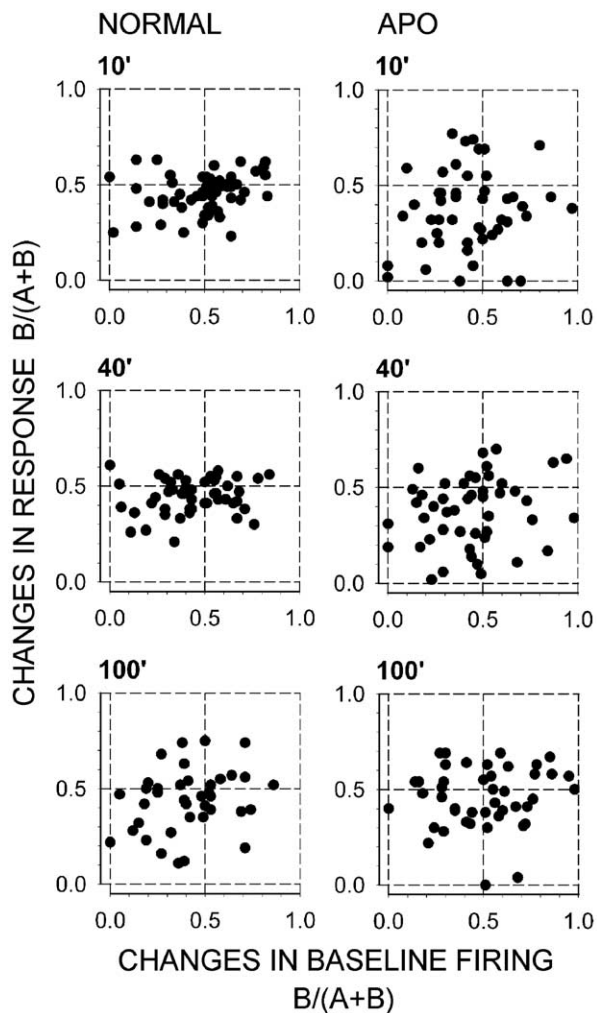


Fig. 3. Comparison of changes in baseline firing and somatosensory responsiveness of each neuron. At each time point (row), each dot represents a single neuron's calculated change $[B/(A+B)]$ in responsiveness plotted against its change in baseline firing. Left column: NORMAL group; right column: APO group.

sensory striatal neurons of SNr-intralaminar thalamic-striatal projections that may have been altered by intranigral APO injection. At the same time, an APO influence on SNr neurons could partially explain some motoric disturbances observed, i.e. via altered SNr transmission to motor thalamo-cortical neurons.

The main effect of decreasing striatal dopamine transmission was to selectively derestrict the magnitudes of fluctuations in striatal somatosensory responsiveness. Fluctuations in both directions were greater in the APO group, compared with the NORMAL group, followed by a return to a lack of difference between groups at 100 min. Thus, dopamine appears to be responsible for the normal restriction on natural fluctuations in striatal responsiveness to synaptic input from somatosensory cortex. Moreover, the changes of RZs in the APO group suggest that some striatal neurons may have undergone a reorganization of their effective afferent somatosensory inputs in the absence of dopamine transmission.

After APO microinjection, the increased magnitude of fluctuations in somatosensory responsiveness, coupled with the minimal changes in baseline firing, suggests that nigrostriatal dopamine transmission mainly influences the somatosensory inputs to striatal spiny type II projection neurons (Kimura, 1990). The present data demonstrate dopamine's ability to modulate corticostriatal transmission, consistent with the suggestion of Freund et al. (1984), based on the pattern by which nigrostriatal and corticostriatal terminals innervate spines of medium spiny neurons. The actions reported here reaffirm the complexity of dopamine's modulatory actions in striatum.

It has been suggested that the nigrostriatal dopaminergic system exerts tonic effects (Chase and Oh, 2000), such as augmented baseline firing due to decreased striatal dopamine levels (Kish et al., 1999). During anesthesia, influences of dopamine-like agents on striatal neurons were mainly inhibitory in nature, but some excitatory actions have been shown. Stimulation of SNc attenuated the excitatory responses to cortical stimulation in the majority of striatal neurons but enhanced it in a minority of these neurons. Both effects were reduced or abolished by systemic administration of dopamine antagonists (Hirata et al., 1984; Vives and Mogenson, 1986).

Alternatively, dopaminergic input may modulate the phasic responses of striatal neurons (Horvitz, 2002). Both excitatory and inhibitory responses of striatal neurons to repetitive sciatic nerve stimulation were decreased by systemically injected amphetamine, while baseline activity showed little change (Abercrombie and Jacobs, 1985). This suggested that dopamine action upon target neurons might be more complex than simple inhibition or excitation, possibly modulating the intensity of neuronal responses to incoming signals. Iontophoretically applied dopamine inhibited glutamate-induced and acetylcholine-induced excitation of striatal neurons (Brown and Arbuthnott, 1983). However, dopamine ejected with lower current could potentiate glutamate-evoked neuronal activity, an effect that required simultaneous stimulation of both D₁ and D₂ receptors (Hu and White, 1997). Under behaviorally relevant

conditions, dopamine acted mainly via D₁-receptors to provide a restraining effect on responsiveness of striatal neurons to glutamate-mediated excitatory input (Kiyatkin and Rebec, 1999).

These data and our results are consistent with the interpretation that dopamine's modulatory actions include limiting the range of responsiveness of striatal neurons to cortical input. Dopamine may exert some dynamic control over the cutaneous RZ representation for each striatal neuron. Our findings regarding the influence of SNc dopamine neurons on striatal somatosensory function encourage further investigation of these complex mechanisms in freely moving animals using intranigral and intrastriatal microinjections of highly specific agonists and antagonists of dopamine receptors.

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