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Learn. Mem. 2000 7: 357-362
Access the most recent version at doi:10.1101/lm.34800

Brief Communication

Role of a Striatal Slowly Inactivating Potassium Current in Short-Term Facilitation of Corticostriatal Inputs: A Computer Simulation Study

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Striatal output neurons (SONs) integrate glutamatergic synaptic inputs originating from the cerebral cortex. In vivo electrophysiological data have shown that a prior depolarization of SONs induced a short-term (≤1 sec) increase in their membrane excitability, which facilitated the ability of corticostriatal synaptic potentials to induce firing. Here we propose, using a computational model of SONs, that the use-dependent, short-term increase in the responsiveness of SONs mainly results from the slow kinetics of a voltage-dependent, slowly inactivating potassium A-current. This mechanism confers on SONs a form of intrinsic short-term memory that optimizes the synaptic input–output relationship as a function of their past activation.

The striatum, the main input stage of the basal ganglia, provides a dynamic neural network that is involved in adaptive control of behavior (for review, see Graybiel 1995). To achieve this function, GABAergic striatal output neurons (SONs) integrate glutamatergic synaptic inputs from many converging cortical neurons (Wilson 1995). However, one of the main electrophysiological features of SONs that has been recorded in vivo is a low level of spontaneous firing (Wilson 1995; Charpier et al. 1999). It is now assumed that this weak excitability of SONs is due to nonlinear electrical membrane properties rather than to a mutual synaptic inhibition (Jaeger et al. 1994; Nisenbaum et al. 1994; Nisenbaum and Wilson 1995; Wilson 1995). The nonlinear properties of SONs result from a set of voltage-gated potassium and sodium currents, including an inwardly rectifying potassium current (I_{Kir}), a fast (I_{Af}), a slowly inactivating A-current (I_{As}), a persistent (I_{Krp}) potassium current, a slowly inactivating (I_{NaS}), and a persistent (I_{NaP}) sodium current (Hoehn et al. 1993; Nisenbaum et al. 1994; Chao and Alzheimer 1995; Nisenbaum and Wilson 1995; Nisenbaum et al. 1996; Gabel and Nisenbaum 1998; Nisenbaum et al. 1998). A distinctive voltage behavior of SONs is conferred by I_{As}, which is responsible for a slowing of the rate of depolarization that is evident from membrane potentials near −60 mV (Bargas et al. 1989; Nisenbaum et al. 1994; Gabel and Nisenbaum 1998). When a depolarizing input is maintained, the slow ramp of depolarization in SONs can lead to a long latency of action potential discharge (Fig. 1A, part 1, crossed arrow; Nisenbaum et al. 1994).

Recently, using in vivo intracellular recordings from anesthetized rats, we have investigated the role of intrinsic electrical properties of SONs in the temporal integration of their cortical synaptic inputs (Mahon et al. 2000). We showed that direct activation of SONs through intracellular injection of a depolarizing current pulse induced a short-term (≤1 sec) increase in their membrane excitability. This change in excitability facilitated the ability of corticostriatal excitatory postsynaptic potentials (EPSPs) to induce firing.

In this study we attempted to specify the mechanisms involved in the use-dependent increase in SON excitability. To this end, we have now performed a computer neuronal modeling that includes the well-described intrinsic voltage-dependent conductance of SONs. The computer simulation shows that the short-term increase in SON responsiveness probably results from the slow kinetics of the striatal I_{As}.

In vivo Data

As illustrated in Figure 1A, a direct activation of SONs (n = 13) through intracellular injection of a depolarizing current pulse produced a decrease (mean = 55 ± 21 ms) in the first spike latency (Fig. 1A, parts 1 and 2) and additional spikes (Fig. 1A, part 1) in response to a subsequent current pulse of the same intensity. The increase of SON responsiveness, which was quantified by the decrease of the first spike latency, decayed exponentially (τ = 364 ± 37 ms; n = 5 SONs) as a function of the time interval between the conditioning pulse and the test-current pulse and SONs recovered their control excitability after 1.2 sec. As shown in Figure 1B, the prior direct depolarization of SONs was also able to facilitate the ability of cortically evoked EPSPs to induce firing. In the 11 tested neurons, the probability of inducing a suprathreshold synaptic response (out of 50 cortical stimulations applied every 1.5 s) was 0.2 ± 0.15 in the control and reached a value of 0.56 ± 0.26 during the 200-

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Article and publication are at www.learnmem.org/cgi/doi/10.1101/lm.34800.

LEARNING & MEMORY 7:357–362 © 2000 by Cold Spring Harbor Laboratory Press ISSN1072-0502/00 $5.00

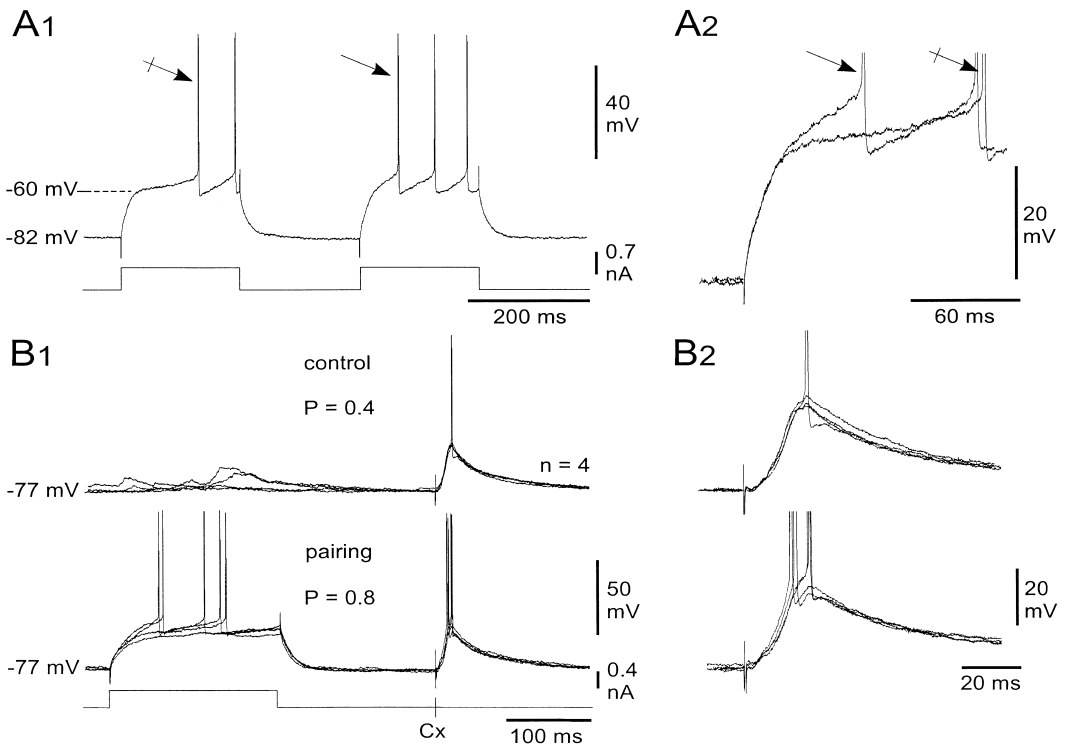

Figure 1 In vivo experimental evidence for use-dependent increase in excitability of striatal output neurons. (*A1, 2*) Increase in intrinsic membrane excitability. (*A1*) Voltage response to intracellular injection of two identical suprathreshold current pulses applied with a time interval of 200 ms. At a membrane potential of −60 mV (dashed line), the striatal cell displayed a slow ramp depolarization that led to a long latency for spike discharge. Note the increase in the number of spikes evoked by the second pulse. (*A2*) Expansion and superimposition of the voltage traces shown in *A1* showing the decrease in the first spike latency (the arrows indicate the spikes shown in *A1*). Spikes are truncated. (*B1, 2*) Facilitation of corticostriatal inputs by a prior depolarizing current pulse. (*B1*) Superimposition of four successive depolarizing synaptic potentials induced in a striatal cell by electrical cortical stimulations (Cx every 1.5 sec), without prior depolarization (control) and 200 ms after a positive current pulse (pairing, paired stimuli every 1.5 sec). The probability to produce a suprathreshold synaptic potential (50 successive trials) in both conditions is indicated in the figure. (*B2*) Expansion and superimposition of the corresponding synaptic responses are shown at the left. Spikes are truncated. In *A1* and *B1*, the resting potential is indicated to the left of the traces. Here and in the following figures, the injected current and the timing of stimulations are shown below the voltage traces.

ms pairing procedure. This facilitation in the corticostriatal synaptic transmission showed the same time course as the change of intrinsic excitability obtained by successive direct stimulations.

Computer Simulation

The increase in SON intrinsic excitability observed in vivo in response to direct stimulation was expressed by a faster rise of membrane depolarization that was associated with a decrease in the first spike latency and an increase in the number of evoked spikes (see Fig. 1A). Because I_{As} is responsible for delayed excitation of SONs, we first examined the putative role of this current in the SON model.

In the whole SON model, application of two successive current pulses with an interstimulus interval (ISI) of 200 ms revealed, on the second stimulus, a decrease of the first spike latency compared to the first voltage response (Δt = 36 ms; Fig. 2A). This facilitation, similar to that observed experimentally (Fig. 1A), was associated with substantial inactivation of I_{As} (Fig. 2A, lower traces). By increasing the time intervals between the conditioning pulse and the test pulse, Δt decreased exponentially (τ = 487 ms) and recovered to its control value for an ISI of 1.4 s (Fig. 2B, triangles). This time course of recovery, which is close to that calculated in vivo (see above), matched the kinetics of recovery from inactivation of I_{As} (Fig. 2B). Without I_{As}, the current-induced increase in excitability (with an ISI of 200 ms) was reduced by 83% (Δt = 6 ms; Fig. 2,C and D). The small remaining time-dependent increase in excitability had a slow time course (τ = 980 ms; Fig. 2B, circles) that paralleled the recovery from inactivation of I_{Krp} (τ = 920 ms).

We simulated alternative SON models by separately re-

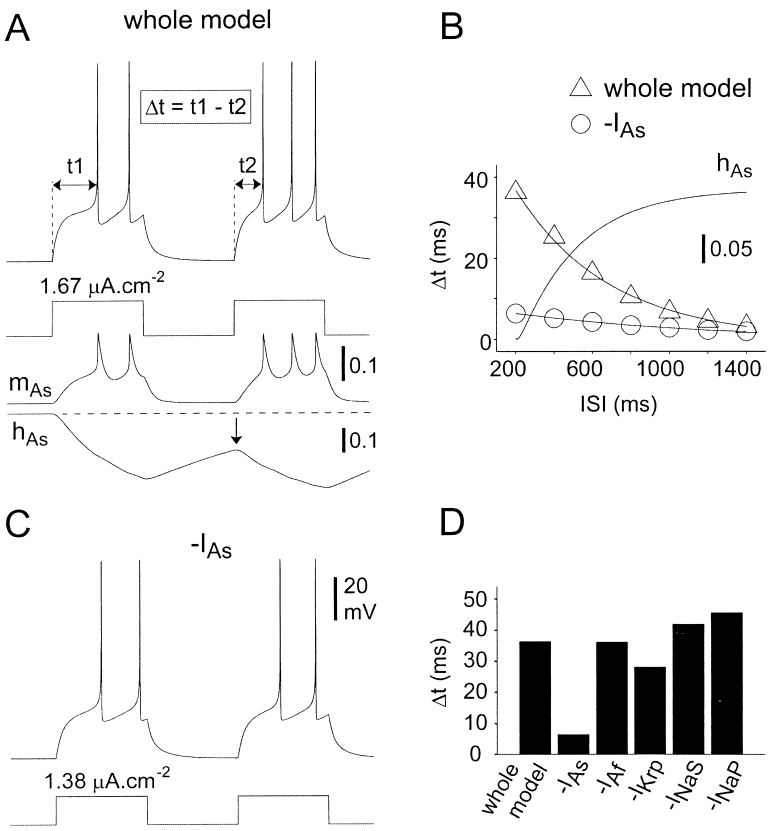

Figure 2 Role of I_{As} in short-term increase in intrinsic excitability of striatal output neuron (SON) model. (*A*, upper traces) Responses to two successive suprathreshold current pulses (200-ms duration) applied with a time interval of 200 ms. The increase in neuron excitability was evident as a decrease (Δt) in the first spike latency between the first (t1) and the second response (t2) and by an increase in the number of evoked spikes. (*A*, lower traces) Activation (m_{As}) and inactivation (h_{As}) dynamics of I_{As} during the pairing protocol. The vertical arrow indicates the level of I_{As} inactivation at the onset of the second pulse. (*B*) Δt as a function of the time interval (ISI) between current pulses, in the whole model (triangles) or in the absence of I_{As} (circles). In the whole model, the recovery of the facilitation was best fitted by a single exponential (τ = 487 ms) and was in accordance with the dynamics of h_{As}. In the absence of I_{As}, a residual facilitation with a slower time course was still present (see text for details). (*C*) Response of the model neuron to a protocol similar to *A* but without I_{As}. Note the lack of significant change in the cell responsiveness. (*D*) Histogram showing the value of Δt, obtained from 200-ms protocols as in *A*, in the whole SON-model neuron and without the indicated individual currents. In *A* and *C*, the intensity of the injected current is indicated above the corresponding traces. The voltage calibration in *C* applies to *A*. The calibration bar in *B* applies to h_{As}. Resting potential = –77.4 mV.

moving I_{Krp}, I_{Af}, I_{NaS}, or I_{NaP}. Using a 200-ms pairing protocol, the marked facilitation observed in the full model remained unchanged ($-I_{Af}$) or was not affected by more than 26% (Fig. 2D).

In a second set of simulations, we tested the effect of a direct depolarization of the SON model on the efficacy of excitatory synaptic inputs. In the control situations, simulated EPSPs (n = 50 trials) triggered an action potential with a probability (P) of 0.34 (Fig. 3A, upper trace). The conditioning procedure consisted of pairing protocols in which simulated EPSPs were induced 200 ms after the offset of a suprathreshold current pulse (200-ms duration). In the whole model, the prior depolarization increased the ability to induce suprathreshold EPSPs, with a P = 0.74, corresponding to 218% of the control value (Fig. 3A, lower trace). This facilitation of the corticostriatal synaptic transmission decayed exponentially (τ = 354 ms; Fig. 3B, triangles) as a function of the time interval between the current pulses and the simulated EPSPs. When I_{As} was omitted from the SON model, the use-dependent facilitation, using a 200-ms paradigm, was almost cancelled, leading to a P-value (0.38) close to that obtained without prior depolarization (Fig. 3,B and C). As expected from this result, the time-dependent increase in synaptic transmission was dramatically weakened (Fig. 3B, circles). As observed with the pairing protocols using successive direct stimulations, the absence of I_{Krp}, I_{Af}, I_{NaS}, or I_{NaP} (Fig. 3C) was without significant effect (see Fig. 3 legend) on the facilitation observed in the whole model.

In many central neurons, such as hippocampal CA1 pyramidal neurons (Storm 1988), thalamic lateral geniculate nucleus relay neurons (McCormick 1991), and prefrontal cortical neurons (Hammond and Crepel 1992), which exhibit slowly inactivating potassium currents similar to the striatal I_{As}, an increase in intrinsic membrane excitability has been described in vitro during repetitive membrane depolarization. However, the role of the dynamic properties of these potassium A-currents in the temporal integration of synaptic signals was suggested rather than shown at synaptic connections (Turrigiano et al. 1996). Our experimental data have shown that a prior depolarization of SONs causes a short-term increase in membrane intrinsic excitability (≤1.2 s) that facilitates the ability to induce suprathreshold EPSPs. This computational study strongly suggests that this time-dependent increase in SON responsiveness mainly results from the slow kinetics of recovery from inactivation of the striatal potassium current, I_{As}. This conclusion is supported by the fact that I_{As} is required in the SON computer

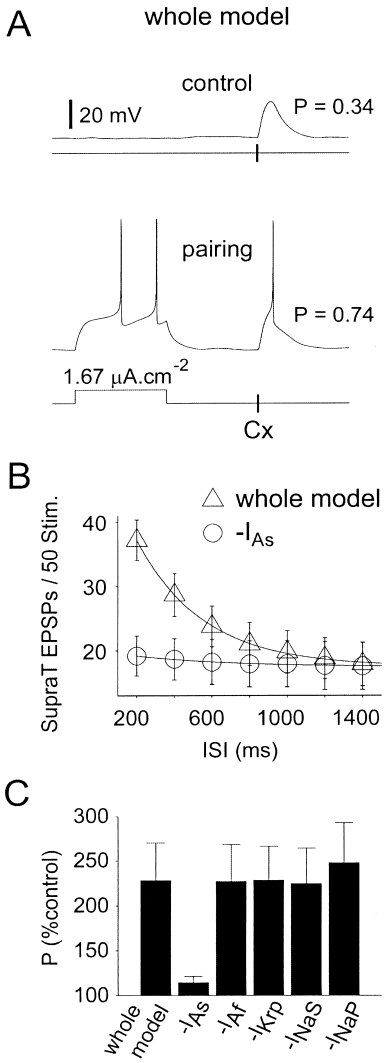

Figure 3 The striatal output neuron (SON) model shows that I_{As} is required for the short-term facilitation in the corticostriatal synaptic transmission. (*A*) Responses of the whole SON model to isolated simulated cortically evoked excitatory postsynaptic potentials (EPSPs; control) and following a depolarizing current pulse (200-ms duration; pairing). The simulated synaptic responses were applied 200 ms after the offset of the current pulse. The control and the pairing protocols ($n = 50$ stimulations in both protocols) were repeated 20 times. The mean probability (P) to elicit a suprathreshold EPSP was increased by the prior depolarization, the P-values in control and during the pairing procedure are indicated in the figure. (*B*) Time course of the synaptic facilitation as a function of the time interval (ISI) between the current pulse and the simulated synaptic potential, in the whole model (triangles; $\tau = 354$ ms) and without I_{As} (circles). (*C*) Histogram showing the increase of P during the 200-ms pairing protocols in the whole SON model and when the different currents were separately withdrawn. P is expressed as a percentage of the corresponding control value (i.e., without prior depolarization). Changes in P-values compared to the whole model were not significant ($P > 0.1$) except when I_{As} was removed ($P < 0.0001$). In *B* and *C*, the error bar represents the standard deviation.

model to reproduce reliably the experimental data. Indeed, a significant facilitation could not be obtained when I_{As} was removed from the SON model and, conversely, the facilitation was still observed in absence of other currents. Moreover, when I_{As} was present in the computer simulation, the strength and the time course of the facilitation were similar to those observed from in vivo experiments. This causal link between I_{As} and the change in excitability is also supported by the similarity between kinetics of recovery from inactivation of I_{As} and the time course of the increase in SON responsiveness. This consistency between our experimental results, obtained at identified synaptic connections, and this computational analysis strongly supports the hypothesis that dynamics of postsynaptic potassium A-currents are crucial for shaping the integrative properties of neurons.

It has been proposed that the nonlinear electrical properties of SONs (see above) enable the filtering out of small and temporally uncorrelated depolarizing synaptic inputs. Therefore, numerous synchronized excitatory inputs are required to produce a substantial membrane depolarization in SONs (Wilson 1995; Charpier et al. 1999). The short-term facilitation described here could prolong the window for detection of temporally distributed cortical synaptic inputs. In behaving animals, this could provide a cellular substrate for the necessary temporal links within the striatum between sensory- and motor-cortical information during behavioral learning (Flaherty and Graybiel 1991).

MATERIALS AND METHODS

Physiology

Experiments were conducted on adult Sprague-Dawley male rats anesthetized with pentobarbital. The methods are described in detail elsewhere (Mahon et al. 2000).

Briefly, intracellular recordings of SONs were obtained with micropipettes filled with a 2 M solution of potassium acetate (DC resistance 50–70 MΩ). Signals were recorded using the conventional current-clamp method with a high-impedance amplifier. SONs were located in the striatal projection field of the orofacial motor cortex and monosynaptic EPSPs were evoked in the recorded neurons by electrical stimulation of this cortical region (see Charpier et al. 1999).

Computational Modeling

Standard Hodgkin-Huxley modeling techniques were used to simulate an isopotential model of SONs. Membrane potential obeyed $CdV/dt = -(I_{Na} + I_K + I_{lcak} + I_{Kir} + I_{Af} + I_{As} + I_{Krp} + I_{NaP} + I_{NaS} + I_{syn} + I_{noise}) + I_{inj}$, with $C = 1\mu F \cdot cm.^{-2} \cdot I_{inj}$ was an injected current ($\mu A \cdot cm^{-2}$) applied to the SON model. Action potential currents (I_{Na}, I_K) were taken from a computer modeling of GABAergic neurons (Wang and Buzsáki 1996) because their spike features were similar to those observed in SONs. However, kinetics were shifted 7 mV rightward in order to match precisely the spike properties of SONs recorded in vivo (see Charpier et al. 1999; Mahon et al. 2000). The voltage-dependent currents responsible for subthreshold membrane potential behavior were simulated using electrophysiological data obtained from SONs recorded in vitro, including I_{Kir} (Nisen-

Table 1. *Model Parameters of Striatal Output Neuron (SON) Model Membrane Currents Including Current Denomination, Gating Structure, Maximal Conductance, Steady State, Time-Constant Functions Parameters, and Reversal Potentials*

			s_∞ (V)		τ (V)			
Current	m^k, h	$\bar{g}$(mS · cm^{-2})	V_S (mV)	k_S (mV)	τ_0 (ms)	V_τ (mV)	k_τ (mV)	E (mV)
Na	m_{Na}^3, h_{Na}	35			see text			55
K	n_K^4	6			see text			−90
leak	—	0.075	—	—	—	—	—	−75
Kir	m_{Kir}	0.15	−100.0	−10.0	—	—	—	−90
Af	m_{Af}	0.09	−33.1	7.5	1.0	—	—	−73
	h_{Af}		−70.4	−7.6	25.0	—	—	
As	m_{As}	0.32	−25.6	13.3	131.4	−37.4	27.3	−85
	h_{As}		−78.8	−10.4		see text		
Krp	m_{Krp}	0.42	−13.4	12.1	206.2	−53.9	26.5	−77.5
	h_{Krp}		−55.0	−19.0		see text		
NaP	m_{NaP}	0.02	−47.8	3.1	1.0	—	—	45
NaS	m_{NaS}	0.11	−16.0	9.4	637.8	−33.5	26.3	40

These parameters values were obtained in vitro at room temperature (see Materials and Methods).

baum and Wilson 1995), I_{Af} (Surmeier et al. 1989), I_{As} (Gabel and Nisenbaum 1998; Nisenbaum et al. 1998), I_{Krp} (Nisenbaum et al. 1996), I_{NaS} (Hoehn et al. 1993), and I_{NaP} (Chao and Alzheimer 1995). Currents followed $I = \bar{g}\, m^k h(V - E)$, in which E(mV) is the reversal potential. Activation (m) and optional-inactivation (h) gating particles were first order except for I_{Kir}, for which activation was instantaneous. Steady-state functions obeyed the Boltzmann equation $s_\infty(V) = 1/(1 + \exp[-\{V - V_S\}/k_S])$. On the basis of available data, time constants were either voltage independent (τ_0) or defined as $\tau(V) = \tau_0/(\exp[-\{V - V_\tau\}/k_\tau] + \exp[\{V - V_\tau\}/k_\tau])$, except for inactivation of I_{As} for which the kinetics (Gabel and Nisenbaum 1998) were best fitted by $\tau_{hAs}(V) = 1790 + 2930 \cdot \exp(-[\{V + 38.2\}/28]^2) \cdot ([V + 38.2]/28)$ and for the I_{Krp} inactivation time constant that was three times that of I_{As} (Nisenbaum et al. 1996). The function used here for $\tau_{hAs}(V)$ was extracted from the multimodal voltage-dependent kinetics of inactivation and recovery from inactivation of I_{As} described from in vitro experiments (Gabel and Nisenbaum 1998).

Current parameters listed in Table 1 are from experimental data provided in the related references cited above. Because these values were obtained at room temperature (20–23°C), we adjusted current kinetics, assuming a conservative Q10 value of 2.5, to reproduce the physiological situation at 37°C.

The conditioning used to induce changes of excitability in the SON model reproduced that applied in vivo (see Fig. 1). This consisted of an injection of a positive, suprathreshold current pulse that lasted for 200 ms. The effect of this conditioning pulse was tested either on the voltage response to a test-current pulse (of the same intensity and duration as the conditioning pulse) or on simulated, cortically evoked EPSPs. These excitatory synaptic potentials were simulated using $I_{syn} = \bar{g}_{syn}\alpha(t)(V - E_{syn})$ in which $E_{syn} = 0$ mV and $\alpha(t)$ is the α function ($\tau_\alpha = 15$ ms). In addition, a Gaussian noise current (I_{noise}; mean = 0 ± 0.04 μA·cm^{-2}) filtered with the same time constant was added in simulations with synaptic inputs. The increase in SON excitability described in vivo was seen within a voltage window close to the firing threshold (Mahon et al. 2000). Therefore, we tested in the present computational study the putative role of the striatal currents available in this range of membrane potentials, that is, I_{Af}, I_{As}, I_{Krp}, I_{NaS} and I_{NaP}.

The mechanisms of the use-dependent changes in the excitability of SON model were examined by separately removing each of these currents from the whole model. Because control responsiveness of the SON model could be modified under these different situations, we normalized the control level of excitation by adjusting the intensity of the conditioning current pulse to produce a first spike discharge with a latency of 100 ms. In addition, we used a $\bar{g}_{syn}$ ranged between 0.0345 and 0.0465 mS·cm^{-2} to normalize, under the different control situations, the number of suprathreshold cortically evoked EPSPs (17 out of 50 trials).

Ordinary differential equations were numerically integrated using adaptive time step Runge-Kutta-Fehlberg methods (Press et al. 1992) with order of accuracy 2–3 and 4–5 (with 10^{-3} relative error tolerance and 10^{-6} absolute error tolerances) using standard library routines from the MATLAB language. Data are expressed in mean ± SD. The unpaired Student's *t*-test (two tailed) or the Mann-Whitney rank sum test was used to assess statistical significance.

ACKNOWLEDGMENTS

We thank Drs. Y. Burnod and S. Slagth for thoughtful comments and critical reading of the manuscript. This work was supported by the BIOMED 2 program, PL 962215.

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Received June 19, 2000; accepted in revised form August 10, 2000.