The Basal Ganglia and Adaptive Motor Control

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The basal ganglia are neural structures within the motor and cognitive control circuits in the mammalian forebrain and are interconnected with the neocortex by multiple loops. Dysfunction in these parallel loops caused by damage to the striatum results in major defects in voluntary movement, exemplified in Parkinson's disease and Huntington's disease. These parallel loops have a distributed modular architecture resembling local expert architectures of computational learning models. During sensorimotor learning, such distributed networks may be coordinated by widely spaced striatal interneurons that acquire response properties on the basis of experienced reward.

During voluntary movement, large numbers of neurons in the forebrain and hindbrain become active. It is a major goal of research on the motor system to understand the particular functions of individual cortical areas and subcortical sites in generating volitional acts. Earlier views of cortical movement control emphasizing the primary motor cortex have now been augmented by models that involve distributed coding across multiple cortical areas, selective activation of cortical areas during the preparatory phases preceding movement, and differences in the degree of activation of cortical areas before internally guided and stimulus-triggered movements (*1*).

Subcortical movement circuits, including the cerebellum and basal ganglia, also show large-scale changes in activation before and during motor activity (*2*). One of the problems posed by such multilevel neural activity is the motor analog to the binding problem in perception (*3*). How are brain circuits coordinated temporally and spatially so as to produce coordinated motor acts?

One interesting possibility suggested by recent work (*4*) is that synchronous or oscillatory responses in motor cortical areas could underlie these functions in a way analogous to that posited for synchronous firing and ~40-Hz oscillations in the visual cortex and thalamus (*5*). Episodes of synchrony involving a broad range of frequencies have also been observed (*6*), suggesting that the distributed systems known by anatomical description might have a functional counterpart in coherent firing patterns related to perceptual binding. Relatively little information is yet available about coordinated neural firing in motor circuits, however, and with the notable exception of work on cerebellar oscillations (*7*), almost nothing is known about coherence in the subcortical structures that provide information to primary and higher order motor cortical areas.

A particularly interesting set of subcortical structures are the basal ganglia, which in the primate brain form a massive collection of neurons with outputs mainly directed toward the motor and prefrontal areas of the frontal lobes, as well as toward some brainstem motor sites (*8–11*). Dysfunction of the basal ganglia and the brain nuclei interconnected with them leads to disturbances of movement and cognition, including disordered timing of movements (*12, 13*). The anatomical circuit arrangement within which the basal ganglia reside is unique. Their largest input station, the striatum, collects inputs from the entire neocortex and sends processed information through other parts of the basal ganglia to areas of frontal cortex that have been implicated in motor planning and execution. These striatal circuits are modulated by the dopamine-containing nigrostriatal tract, which degenerates in Parkinson's disease. The striatum also receives inputs from thalamic sites implicated in rhythmic firing in the forebrain (*14*) and from the amygdala, which functions in reward learning and emotional behaviors, such as the expression of fear (*15*).

This arrangement, and the multiple internal loops of the basal ganglia, have led to speculation that the basal ganglia are not simply related to motor execution per se. Instead, they may participate in motor planning or predictive control, motor sequencing, motor learning, and action repertoires involving motivational and cognitive drive.

Single-unit studies of striatal neurons are in accord with this general view, because many of these neurons exhibit highly context-dependent firing patterns (*11, 16, 17*). They are active in relation to movements triggered by particular cues, including memory-encoded cues as well as environmental ones. Most of the cells recorded in the striatum are its projection neurons. It is these projection neurons, rather than the rarer striatal interneurons, that degenerate in Huntington's disease (*18*). By virtue of their different transmitter phenotypes and connectivity patterns, striatal projection neurons may enhance or suppress movements through basal ganglia pathways with inhibitory and disinhibitory ("release") effects on their thalamic and brainstem targets (*9, 12, 19*). Dysfunction of these circuits is thus thought to produce either hypokinetic or hyperkinetic basal ganglia disorders (*9, 12, 19*).

It has puzzled investigators for years that there are millions of projection neurons in the primate striatum, but they project to a very much smaller set of neurons in the basal ganglia output nuclei (*20*). Why would striatal neurons have such very specialized properties if they simply blend their outputs by converging at the next step in processing? Part of this puzzle was resolved when it was discovered that there are multiple, parallel channels from the cortex through the basal ganglia and back to the cortex (*8, 21*). But this finding raised other questions: If information is sent in parallel through the basal ganglia, what do the basal ganglia contribute to the processing? And how are different aspects of the processing coordinated (the binding problem)?

We focus here on two sets of findings that suggest novel approaches to these issues. The first is evidence that the input-output architecture of the sensorimotor striatum has a modular design that remaps cortical inputs onto distributed local modules of striatal projection neurons. The second is evidence that the class of striatal neurons known as tonically active neurons (TANs) may contribute to temporal binding across such modular networks during behavioral learning by undergoing dopamine-sensitive changes in their response properties.

Modular Remapping in the Basal Ganglia

An early hint that input-output remapping might occur in modules in the striatum came from evidence that a dispersed set of neurochemically specialized patchy zones, the striosomes, tend to collect inputs related to the limbic system and to project to the dopamine-containing substantia nigra pars compacta (*9, 22*). It is now clear, however,

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that the entire striatum is modular. There are input-output modules in the large matrix compartment, and these "matrisomes" receive sensorimotor and associative inputs and project to the output nuclei of the basal ganglia (*23*).

Studying the primate somatic sensory and motor cortex, for example, we found that the inputs derived from any small cortical site representing a particular body part (say, the contralateral hand) form a distributed, partly interconnected set of zones in the striatum (*24*). Any given matrisome receives overlapping inputs from the same body-part representation in different subareas of the sensorimotor cortex, so that several sorts of information relevant to that body-part converge. This suggests that the cortical representations of the body are remapped in the striatum by being broken up into local modules with convergent inputs (Fig. 1).

It is biologically expensive to set up such patchy distributed systems. This is not because they necessitate extra axonal length, but because, in addition to implying a general topography to organize the connections, they must also follow local constraints that break the rules of topography in order to focus inputs into special modules (*25*). Why go to the expense?

One possible answer to this question comes from experiments in which anatomical tracers were placed into monkey brains to label simultaneously sensorimotor inputs to the striatum and striatal outputs to the pallidum, a main output structure of the basal ganglia. We found that labeled input fiber clusters can overlap clusters of labeled projection neurons quite precisely (Fig. 1) (*26–28*). This suggests a pattern of remapping in the basal ganglia that involves divergence from the cortex to the striatum followed by reconvergence from the striatum to the pallidum. In effect, the information is dispersed to distributed modules in the striatum, but it can be brought together again at the next stage of processing (Fig. 2).

This divergence-reconvergence pattern suggests one way to have an overall parallel processing scheme for cortico-basal ganglia interactions and at the same time to maximize computational power within channels. The estimated amount of divergence and convergence is impressive. The modules labeled from a roughly 1-mm-wide site in the sensorimotor cortex stretch over as much as 7 mm of the length and width of the putamen and fill a volume three to five times the volume of the cortical site from which they were labeled. The striatal modules labeled from a given small site in the pallidum have about the same dimensions (*26–28*). Such dispersion is great enough so that the individual modules in any one input-output set could have different nearest neighbor relations. This design thus favors local spatiotemporal coherence within individual input-output modules as well as diversity across them, which could allow plasticity and variability in striatal processing. Individual modules might also be subject to different patterns of within-module convergence (for example, different degrees of submodality convergence) and be subject to different sorts of local processing (for example, reflecting different neurochemical environments). This would further increase the possibility of dynamic processing in apparently parallel channels.

There is a provocative similarity between this biological architecture and the network architecture proposed by Jacobs and colleagues for a supervised learning system (*29*). The input-output modules resemble the "local experts" of their model, which are subsets of elements that perform distinct subtasks of a training routine and which therefore split up the computational problem. The outputs of these local experts can be independently adjusted and gated and then summed at an output stage. This architecture should have advantages for dealing with the degrees-of-freedom problem in motor planning (*30*). In biological systems, we and others find input and output modules throughout the striatum, not just in the sensorimotor striatum (*23*). For example, in the head of the caudate nucleus, which has been implicated in higher order cognitive functions and has abnormal activity in some neuropsychiatric disorders (*31*), this local clustering phenomenon is very pronounced, and the limbic system–related striosomal modules are most conspicuously represented there. Thus, there may be many sets of local expert modules, with some, probably including striosomes, serving nonsensorimotor functions, and there may be different sorts of nearest neighbor relations among the modules as well. These input-output modules in the striatum may be one alternative to the multilayer processing typical of the neocortex (*32*).

These computational considerations suggest that local processing organized with respect to matrisomal and striosomal modules may be a key feature of input-output remapping in the striatum. But how can the activity in different constellations of modules be coordinated? Studying the physiological changes that occur in a particular set of striatal neurons during behavior learning in the monkey, we found evidence for one potential solution to this problem.

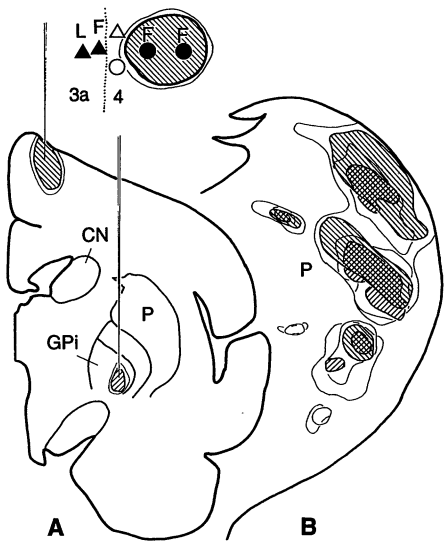

Fig. 1. Modularity of striatal inputs and outputs documented in an axon transport experiment. (**A**) An injection of anterograde tracer was made in a small site in the motor cortex (area 4) representing the foot (\). In the same hemisphere, a small site in the pallidum was injected with retrograde tracer (/). Both the labeled axon projections from cortex to terminal sites in the striatum (\) and the labeled striatopallidal output cells (/) are organized as sets of patches in the putamen (**B**). The input clusters and output clusters overlap extensively [cross-hatching in (B)]. Cortical recording sites (triangles) and stimulation sites (circles) are shown in the inset above (A) (L, leg; F, foot; open symbols, no response) for areas 3a and 4. CN, caudate nucleus; P, putamen; GPi, internal segment of the pallidum.

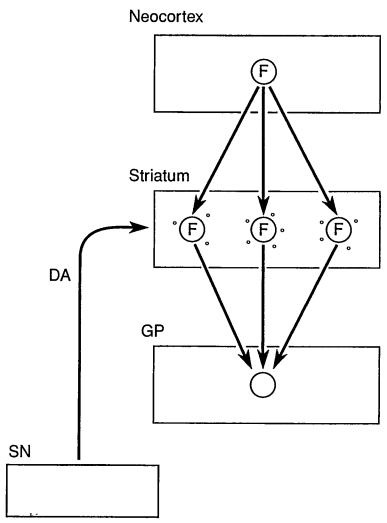

Fig. 2. Model of divergent-reconvergent processing in basal ganglia pathways. Experimental evidence favors the divergence of cortical inputs to modules in the striatum. Any given module can receive somatotopically matched inputs, symbolized by F (foot), from different SI areas (areas 3a, 3b, and 1) and from the motor cortex (area 4). This divergence can be followed by reconvergence onto sets of basal ganglia output cells in the pallidum (GP). Inputs from the midbrain substantia nigra (SN) using the neurotransmitter dopamine (DA) modulate this processing, as do local interneurons (small dots).

Tonically Active Neurons and Striatal Neuroplasticity

The TANs of the striatum, thought to be interneurons (*17*, *33*), are rare striatal neurons that fire "spontaneously" at low (2 to 10 Hz) frequency. Unlike many projection neurons, the TANs do not fire in relation to overt movement. By recording their firing patterns during the course of classical conditioning, we found that these cells undergo changes in responsiveness during the conditioning (*34*).

In tasks in which conditioning stimuli (clicks or lights) were paired with reward presentations (fruit juice), the monkeys learned to respond behaviorally (by licking) to reward-associated conditioning stimuli. During acquisition of conditioning, the TANs developed a pause in their tonic firing, often flanked by brief excitatory phases, which came shortly after the click or light (Fig. 3). The number of responding TANs increased from about 15% before conditioning to over 50 to 70% after conditioning.

The increase in the population response represents a gradual recruitment of responsive TANs during the course of behavioral conditioning and a lengthening of their responses (*35*). These responses clearly were related to conditioned stimuli predictive of reward, because they disappeared when the animals underwent extinction training, during which the stimuli were presented without reward. The TANs did not respond to the rewards alone, however, nor in relation to the licks made to acquire the reward.

As an estimate of the temporal precision of the TAN responses after learning, we calculated coefficients of variation (CVs) for the pause responses of individual TANs and compared these to the CVs for the base line interspike intervals of the TANs (*35*). This analysis demonstrated that a highly significant coordination of TAN activity occurs after learning. The onsets of the pauses that developed showed considerable temporal alignment, and the offsets of the pauses (and onsets of rebound excitations) were also closely correlated in time (*36*). One speculation suggested by these findings is that the pause in TAN firing that develops after learning may represent a resetting of the spike discharge imposed by a suppression of the TANs followed by a time-locked resumption of their activity (a "latch-on") after the inhibition (*35*).

Modules, TANs and the Binding Problem

A remarkable feature of the change in neural firing of TANs accompanying learning is that it occurred in TANs very widely dispersed through the striatum (Fig. 4). Moreover, the TAN response offsets were temporally coordinated independent of whether they lay in striatal districts receiving inputs predominantly from the sensorimotor cortex (much of the putamen) or in regions with inputs predominantly from prefrontal and other association cortex (much of the caudate nucleus).

This broad distribution and temporal coordination of the TAN response suggested that TANs might have a special function in coordinating the distributed modular circuitry of cortico-basal ganglia channels. To test this idea, we explicitly mapped the locations of TANs with respect to the chemically defined striosomes, which we could detect with immunohistochemical stains (*35*). There was a pronounced tendency for TANs to lie at striosome-matrix borders. Moreover, in some of the rare instances in which boundaries could be neurochemically detected within the matrix, TANs could be found by them as well. These results suggest that the distribution of the TANs, although very broad, is not random and may be related to the modular organization of the striatum (Fig. 5). Approximately half the TANs identified in acute recording experiments in the squirrel monkey striatum lay at striosome-matrix borders. These TANs could link limbic information with information of other types (for example, that derived from frontal association cortex) in a coordinated, distributed way (*35*, *37*).

The fact that TANs distributed over broad regions of the striatum come to respond in a temporally coordinated way after behavioral conditioning suggests that these striatal neurons could facilitate coordinated changes in the activity of other striatal neurons, including the projection neurons, during learning. Even modules stretching across millimeters of striatum and forming parts of different "parallel" circuits could be affected. In the striatum, then, the binding problem imposed by the modular organization of the striatum, by its divergent input architecture, and by the presence of parallel basal ganglia channels running through the striatum could be solved by having distributed sets of neurons (almost certainly interneurons) that coordinate the modules, inputs, and loop activities.

This proposal suggests a novel solution for part of the binding problem in the sensorimotor system. Rather than having all systems synchronous with each other, the coordination is a contingent coordination, built up through learning and implemented, in the case of the striatum, by distributed sets of local neurons sensitive to motivation-related signals. Simultaneous multiple TAN recordings will be necessary to judge how synchronous the TAN responses are. However, our results already suggest that their effects could be within the summation times of their potential target neurons (*38*, *39*) and that the pause overlaps could be sufficiently aligned to create a time window in which TAN activity is coherently diminished (*40*).

Binding, Reward, and Striatal Dopamine

The reward association of the TAN response acquired through conditioning suggested a further experiment based on evidence obtained by Schultz and co-workers (*41*). These investigators have shown that the dopamine-containing neurons of the substantia nigra, which project to the striatum, fire in response to primary rewards and, after conditioning, to reward-related stimuli. To determine whether these dopamine-containing fibers influence TAN neuroplasticity, we destroyed these fibers

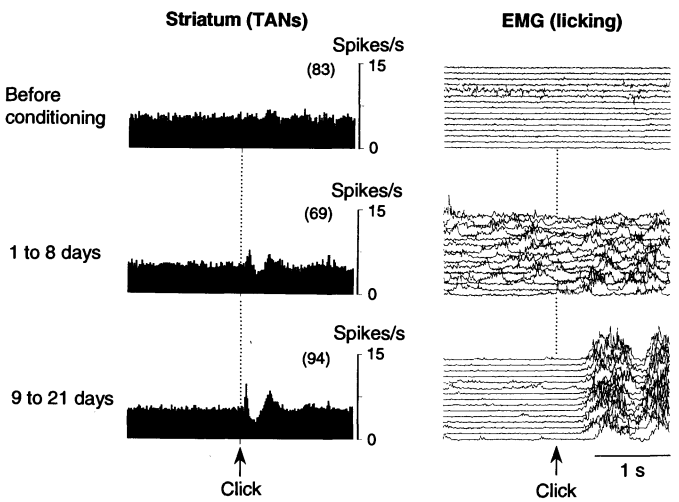

Fig. 3. Emergence of population responses of TANs in the striatum during a classical conditioning task in which the monkey learns to associate the presentation of a click with a juice reward that the animal can obtain by licking. (**Left**) Population histograms of TANs before, during, and after behavioral learning. The neurons show an initial activation, then a reduction in firing, and then a rebound excitation. (**Right**) The corresponding acquisition of the conditional behavioral response (licking). As the monkey learns the conditioned response, the TANs acquire a response to the conditioning stimulus. The numbers in parentheses indicate the number of neurons sampled. EMG, electromyogram.

unilaterally by local infusion of the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) into the striata of monkeys that had learned the conditioning task. When the dopamine-containing fibers were destroyed in one hemisphere, the monkeys could still perform (albeit clumsily) the licking task, which required muscle activity on both sides. However, most TANs on the side of the infusion lost their pause response, even though their tonic activity seemed normal. The TANs in the control hemisphere, by contrast, continued to respond. These experiments strongly suggest that the expression of the acquired responses of the TANs requires tonic dopaminergic input. Moreover, systemic injections of apomorphine could reinstate the responses lost on the side of the infusion, which suggests that the dopamine signal needed for expression of the response may be spatiotemporally permissive (*42*).

Striatal Activity and Sensorimotor Learning

The experiments described above suggest that during conditioning, striatal TANs develop a response that is spatially distributed, temporally coordinated, predictive of reward, and dependent on dopamine. What could such a neural signal accomplish? An impediment to answering this question is that the cellular phenotype of the TANs is not known. However, their electrophysiological properties point to their being striatal interneurons, and in particular their broad action potentials, prolonged after-hyperpolarizations, and tonic firing closely resemble properties of cholinergic interneurons and do not resemble those of projection neurons or noncholinergic interneurons, as identified in studies in the rat (*38*, *43*). Our finding that TANs are differentially distributed at striosomal borders and are strongly represented in the matrix is also compatible with the view that TANs could be cholinergic interneurons (*35*).

In sharp contrast to the TANs, striatal projection neurons are peculiar in being almost completely silent in the absence of movement or equivalent activation. But once they are brought to firing threshold, they exhibit bursts of firing (*38*, *39*). They tend to be in "up" or "down" states and to flip between them. It is likely that the burstlike projection neuron firing, rather than the small modulations of TAN firing, contributes the immediate transfer of neural information to striatal output targets in the pallidum and substantia nigra. However, the TANs could have a considerable influence on the subthreshold potentials of projection neurons, potentials that are of great importance in determining whether and when the burstlike activity of these normally nearly silent neurons will occur (*44*).

Interestingly, muscarinic cholinergic receptor agonists tend to stabilize the up or down states of projection neurons by modulation of A-currents (*45*). If the TANs are cholinergic interneurons, a coordinated pause plus a rebound in TAN firing could serve as a temporally coordinated signal to reset the activity states of projection neurons over widespread regions of the striatum (*35*). Over time, through the gradual acquisition of time-locked conditioned responses

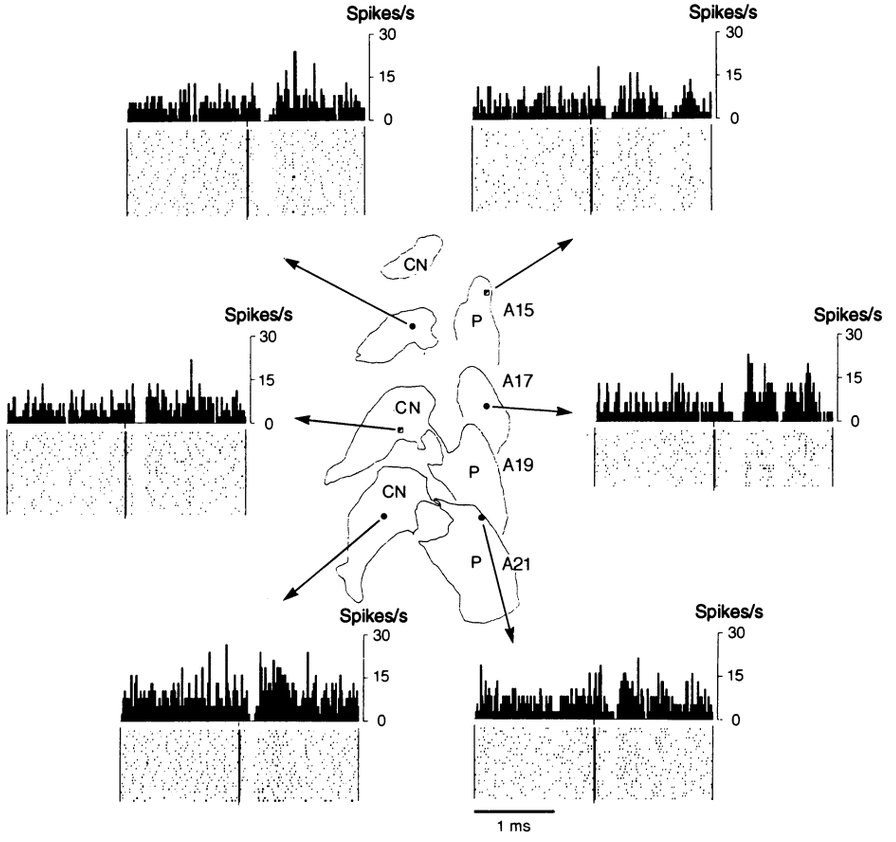

Fig. 4. Similarity of pause responses acquired by TANs in widely spaced regions of the striatum as a result of conditioning with clicks and light-emitting diode (LED) lights as conditioned stimuli. The responses of six representative TANs recorded at particular sites (indicated by dots or squares) are shown in individual raster plots and spike histograms. Despite their widespread distribution, all of the cells show a pause response after the presentation of one or both of the stimuli used as conditioning stimuli (●, cell that responded to both; ◤, responsive cell that was tested only with a click).

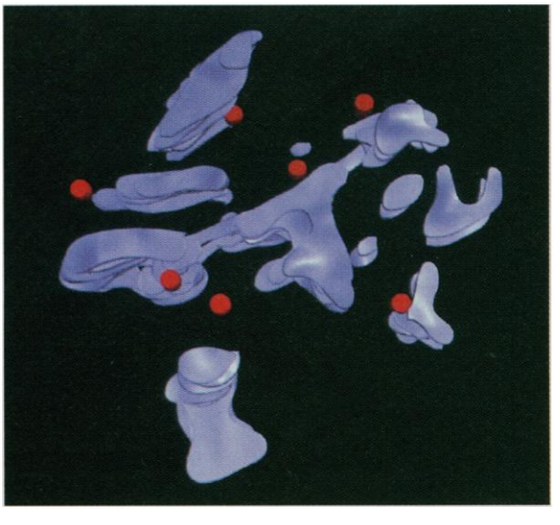

Fig. 5. Preferential distribution of TANs in the matrix and at the edges of striosomes. Shown are striosomes (irregular shaded forms) in nine serial sections from a squirrel monkey striatum, reconstructed in three dimensions and plotted together with the sites of TANs (red dots) marked in acute recording experiments in this monkey. A disproportionate number of TAN sites lie at striosome-matrix borders; others lie in the matrix and may be at matrisome-matrisome borders.

during repeated exposure to particular behavioral contexts, TANs could come to influence the probability of the firing of sets of surrounding neurons when these neurons contemporaneously receive inputs related to the stimulus-response context. The short-term modifiability introduced by the latch-on function could thus have long-term effects.

The scheme of striatal processing that we propose has elements of both unsupervised (Hebbian) and supervised learning models (*46*). Within local striatal modules, the degree of temporal coherence of inputs to the projection neurons could determine input-output functions with Hebbian mechanisms acting in adjustments of synaptic strengths (*47*). In addition, however, the learning would be supervised: The modifiability of any given module at any particular time point would be influenced by distributed interneurons active in a conditional spatiotemporal gating of the activity in the distributed modules.

The response of the TANs to conditioned stimuli fits well with the idea that the striatum is involved in predictive control: The cells modulate their spike activity in relation to stimuli that are predictive of reward. Their change in responsiveness is also compatible with the idea that the striatum is involved in some aspects of sensorimotor learning (*48*), because their responses undergo changes with conditioning as the monkey learns the behavioral sensorimotor task. Our evidence that TAN plasticity is modulated by dopamine is in turn compatible with a "teaching" role for reward-related nigrostriatal dopaminergic input (*49*), and it may help in understanding the remarkable success of dopamine replacement therapies in facilitating the movements of patients with Parkinson's disease (*50*). More generally, it is of interest that numerous pharmacologic therapies for basal ganglia disorders target cholinergic and dopaminergic systems (*51*). Both systems have also been implicated in striatal synaptic plasticity in studies of long-term depression and long-term potentiation (*52*). The prediction from our analysis would be that not only difficulties in motor learning or predictive control, as in sequential movement planning, but also difficulties in timing complex movements and even cognitive and psychomotor behaviors could result from reduced temporal coordination of activity in the modular distributed circuits discussed here.

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28. This pattern has been observed for both striatal projections to the external pallidum and projections to the internal pallidum. For technical reasons, however, it is not known how common this pattern is nor whether the convergence from striatum to pallidum is at the regional or at the cellular level (*27*).
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RESEARCH ARTICLE

The "Ozone Deficit" Problem: $O_2(X, v \geq 26) + O(^3P)$ from 226-nm Ozone Photodissociation

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Highly vibrationally excited $O_2(X\,^3\Sigma_g^-, v \geq 26)$ has been observed from the photodissociation of ozone (O_3), and the quantum yield for this reaction has been determined for excitation at 226 nanometers. This observation may help to address the "ozone deficit" problem, or why the previously predicted stratospheric O_3 concentration is less than that observed. Recent kinetic studies have suggested that $O_2(X\,^3\Sigma_g^-, v \geq 26)$ can react rapidly with O_2 to form $O_3 + O$ and have led to speculation that, if produced in the photodissociation of O_3, this species might be involved in resolving the discrepancy. The sequence $O_3 + h\nu \rightarrow O_2(X\,^3\Sigma_g^-, v \geq 26) + O$; $O_2(X\,^3\Sigma_g^-, v \geq 26) + O_2 \rightarrow O_3 + O$ (where $h\nu$ is a photon) would be an autocatalytic mechanism for production of odd oxygen. A two-dimensional atmospheric model has been used to evaluate the importance of this new mechanism. The new mechanism can completely account for the tropical O_3 deficit at an altitude of 43 kilometers, but it does not completely account for the deficit at higher altitudes. The mechanism also provides for isotopic fractionation and may contribute to an explanation for the anomalously high concentration of heavy O_3 in the stratosphere.

Attempts to predict the stratospheric O_3 concentration date historically to Chapman's pioneering work in 1930 (*1*). Despite years of effort, there is still a significant discrepancy between the predicted and measured O_3 concentration in the upper stratosphere (*2*). It has recently been proposed that if the O_2 produced in O_3 photolysis were sufficiently vibrationally excited to react with ground-state O_2 to form $O_3 + O$, this sequence of reactions might provide a means for formation of stratospheric O_3 (*3*–*5*). The work described here demonstrates that highly vibrationally excited O_2 is indeed formed in the photodissociation of O_3. The quantum yield of this reaction has been determined so that the importance of the mechanism proposed in (*3*–*5*) can be evaluated. Calculations show that the inclusion of highly vibrationally excited O_2 in atmospheric models may help resolve the long-standing O_3 deficit problem. In addition, the new dissociation mechanism involving vibrationally excited O_2 may also be involved in resolving the long-standing mystery of why heavy isotopes of O_3 are more abundant in the stratosphere (*6*).

Two new experiments were performed to explore the photodissociation of O_3 at wavelengths near 226 nm. The newly developed technique of photofragment imaging has been used to measure the velocity distribution of the $O(^3P)$ product; conservation of energy and momentum provide the internal energy of the O_2 sibling fragment. In addition, laser-induced fluorescence (LIF) on the Schumann-Runge bands has been performed to determine the distribution of O_2 $(X^3\Sigma_g^-, v)$ population in the vibrational levels between $v = 19$ and $v = 26$. The experiments taken together indicate that the vibrational distribution of the $O_2(X^3\Sigma_g^-, v)$ produced at this dissociation wavelength is markedly bimodal, with one peak near $v = 14$ and another at $v = 27$.

The chemistry of O_3 has come under increasing scrutiny largely because of concern about stratospheric O_3 loss due to human activity. Production of stratospheric O_3 is believed to be due solely to reaction 1:

$$O_2 + h\nu \rightarrow 2O \qquad (1)$$

(where h is Planck's constant), which then rapidly generates O_3 in excess O_2 through three-body recombination: $O + O_2 + M \rightarrow O_3 + M$ ($M = O_2$ or N_2). Decomposition of O_3 is due to two photochemical pathways:

$$O_3 + h\nu \rightarrow O(^1D) + O_2(a^1\Delta_g) \qquad (2)$$

$$\rightarrow O(^3P) + O_2(X^3\Sigma_g^-) \qquad (3)$$

as well as to three catalytic processes commonly referred to as the HO_x, NO_x, and ClO_x cycles (*7*). Notwithstanding the large number of previous attempts (*8*–*23*), models of stratospheric chemistry underestimate the concentration of O_3 when compared to observations at altitudes between 35 and 80 km (*2*, *24*, *25*). This discrepancy is particularly puzzling because O_3 is in photochemical steady-state during daylight hours at these altitudes and its abundance is controlled by such a small number of well-studied chemical and photochemical reactions.

There have been at least two notable attempts to explain the so-called "ozone deficit" problem, each hypothesizing a sec-

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