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Cortical and basal ganglia contributions to habit learning and automaticity

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Abstract

The 20th century was dominated by the view that novel behaviors are mediated primarily in cortex and that the development of automaticity is a process of transferring control to subcortical structures. Much evidence suggests however, that subcortical structures, such as the striatum, make significant contributions to initial learning. More recently, evidence has been accumulating that neurons in the associative striatum are selectively activated during early learning, whereas those in the sensorimotor striatum are more active after automaticity has developed. At the same time, other recent reports suggest that automatic behaviors are striatum- and dopamine-independent, and may be mediated entirely within cortex. Resolving this apparent conflict should be a major goal of future research.

The Classical View of Automaticity

Almost any cognitive or motor skill is executed faster and more accurately the more it is practiced. Eventually these improvements become so great that the behavior is executed habitually or automatically (see Box 1 for some behavioral criteria of habits and automatic behaviors). Such dramatic improvements following practice have been documented in motor and cognitive behaviors as diverse as cigar rolling and proving geometry theorems [1].

Interest in the neural basis of automaticity has a long history, dating back at least to Sherrington [2], who argued that long periods of practice gradually make skills reflexive. These ideas led to the theory that dominated the 20th century: Novel behaviors require attention and flexible thinking and therefore are dependent on cortex, whereas automatic behaviors require neither of these and so are not mediated primarily by cortex. Instead, it has long been assumed that automatic behaviors are primarily mediated by subcortical structures (see [3] for an influential example).

Progress on understanding the neural basis of habit learning and automaticity has lagged considerably behind progress on understanding the neural basis of initial learning, partly because studies of automaticity require more time, patience, and resources than studies of initial learning (where meaningful data are available from the first trial). For example, to study the

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effects of automaticity on neuronal responses in motor cortex, Matsuzaka, Picard, and Strick [4] had monkeys practice the same motor sequence almost daily for up to two years. Despite the difficulty of studying automaticity, the past few years have seen some important advances in our understanding of this topic. As we will show, the classical view has been significantly altered, and some dramatically different theoretical notions of automaticity have been proposed.

Automaticity and habit learning are vast areas of research that span many diverse fields of study. A complete review of all this work is beyond the scope of this article. Instead, our focus is on cortical and basal ganglia contributions to habit learning and automaticity in simple motor and cognitive tasks. A number of recent reviews discuss automaticity in more complex cognitive [5], social [6], and athletic [7] domains, or focus on the contributions of other neuroanatomical regions, including the cerebellum [8–10].

The Role of the Associative Striatum in Habit Learning and Automaticity

One of the first revisions to the classical view occurred with the discovery that there are significant subcortical contributions to initial learning. For example, there is solid evidence that the initial learning of many skills depends critically on the striatum (see Box 2; for reviews, see, e.g., [11–13]).

More recent evidence suggests that associative and sensorimotor regions of the striatum may play different roles in learning and automaticity (see the following section for a discussion of research on the role of the sensorimotor striatum). Several studies have reported that the associative striatum is active during initial skill acquisition and that its activity decreases with extended training. For example, Miyachi, Hikosaka, and Lu [14] recorded the activity of single neurons in associative and sensorimotor regions of the striatum as monkeys learned a sequence of button presses. Neuronal activity was recorded either during early learning or after several months of training on a sequence. Most of the striatal neurons that responded more strongly during early learning were in the associative striatum (Figure 1). Several fMRI experiments subsequently reported a similar finding – that is, that activation in the associative striatum decreases with training [15–17].

These data establish that there is often activity in the associative striatum during initial learning, but they do not address the question of whether this activity helps mediate *acquisition* of the behavior. Evidence supporting a role for the associative striatum in behavioral acquisition was reported by Miyachi et al. [18], who found that temporary inactivation of the associative striatum disrupted learning of new motor sequences, but had less effect on the execution of previously-acquired sequences.

As described in Box 1, habit learning is thought to involve a shift from goal-directed action to behavior that is automatically elicited by an environmental stimulus to which it has become strongly tied. The shift from goal-directed actions to stimulus-response (S-R) habits typically occurs slowly as a result of extended training [19], but lesions of the associative striatum can greatly speed this process. In particular, rats with lesions of the associative striatum show S-R-like habit performance even during early stages of training when goal-directed behavior would normally be seen. In this case, the rate of bar-pressing is unaffected by outcome devaluation [20].

In summary, the associative striatum is especially active early in skill learning. With extended training, activation in the associative striatum is reduced. Disruptions of this region impair initial learning and goal-directed actions, and lead to premature S-R-like habit behavior. Although more data are needed, the current evidence suggests that the associative striatum is particularly important for task *acquisition* and/or performance during early stages of learning.

The Role of the Sensorimotor Striatum in Habit Learning and Automaticity

Whereas the associative striatum seems more critical to early than late stages of learning, the sensorimotor striatum shows the opposite pattern. For example, Miyachi et al. [14] found that most striatal neurons that responded more strongly after over-learning a motor sequence were in the sensorimotor striatum (see Figure 1). Furthermore, temporary inactivation of the sensorimotor striatum does not interfere with the learning of new motor sequences, but it does disrupt the execution of previously-acquired sequences [18]. In rats with lesions of the sensorimotor striatum, extended training of a behavior does not lead to a shift from goal-directed action to S-R habit. In these rats, reductions in the expected value of food reward reduce rates of lever-pressing even after extended training [21]. These results suggest that the sensorimotor striatum may be needed a) for the performance of habit-like behaviors and/or b) for the transition from goal-directed to habit-like performance.

On the other hand, there is evidence that the response of the sensorimotor striatum to extended training may depend on the type of task that is used. In tasks that require subjects to learn a sequence of motor responses (e.g., button presses), a variety of fMRI studies have reported that sensorimotor striatal activation increases with extended training [15–17]. In contrast, different results have been reported for tasks with a single motor response, such as a reinforced lever-press, head-movement, or directed locomotion in a T-maze [22–24]. These studies have reported that extended training leads to a *decrease* in the number of neurons within the sensorimotor striatum showing task-related activation. For example, Carelli et al. [22] trained rats to lever-press to a tone and, over an extended training period, recorded from single units in the sensorimotor striatum that were known to activate phasically during forelimb movements. Figure 2 shows spike histograms from this experiment from five separate experimental sessions. Early in learning, many cells showed burst activity just before the lever press. Several days later, similar striatal units still fired bursts, but now the bursts came after the response had been made, and therefore they could not have played a role in response selection. In later sessions, presumably after automaticity was well established, the cells ceased responding altogether; that is, neither the tone nor the response elicited any activity from striatal units that responded vigorously when the same movement was made under control conditions.

Other studies also reported a decrease in the number of sensorimotor striatal neurons showing task-relevant phasic activations over the course of training, but in addition, reported that a small group of neurons within the sensorimotor striatum continued to show task-related activations and that the magnitude of the phasic neuronal responses in this restricted population of neurons increased over the course of extended training [23–24].

In sequence-learning tasks, performance is often facilitated by the learning of motor-motor associations, whereas this is less likely the case in tasks with a single motor response. All of premotor and motor cortex projects directly into the sensorimotor striatum [Box 2; 25–26], so one possibility is that the greater activation seen in the sensorimotor striatum with extended training in sequence learning tasks is driven by input from premotor/motor areas that is mediating the learning of motor-motor associations.

These results clearly suggest a role for the sensorimotor striatum in automatic responding, and perhaps also a role in the transition from goal-directed to habit-like performance. Not surprisingly, there have been several proposals that the development of automaticity involves a gradual transfer of control from the associative to the sensorimotor striatum [27–29]. The exact details of how such a transfer is mediated have not been worked out.

It is also important to note that some results seem inconsistent with the hypothesis that the sensorimotor striatum mediates automatic responding. For example, temporary inactivations of sensorimotor regions of the internal segment of the globus pallidus (via injections of the

GABA agonist muscimol) should prevent the sensorimotor striatum from influencing cortex. Thus, if the striatum mediates the expression of over-learned behaviors, then such inactivations should disrupt highly practiced responses. In contrast to this prediction, however, Turner et al. [30] reported that the great majority of such inactivations had no effect on the ability of monkeys to produce a highly practiced motor sequence. Other evidence that the striatum is important for initial learning but not for the expression of well-learned behaviors comes from the study of song behaviors in birds. Several studies have shown that disconnecting the bird homologue of the basal ganglia completely blocks new song learning, but has little effect on the expression of well-learned songs (see [31] for a review).

Dopamine and Cortico-Striatal Plasticity

Cortico-striatal synapses can undergo both strengthening (long-term potentiation, or LTP), and weakening (long-term depression, or LTD; [32–33]). Cortical high-frequency stimulation has most frequently been observed to produce LTD at cortico-striatal synapses. However, when dopamine is applied in brief pulses coinciding with the time of pre-synaptic stimulation and post-synaptic depolarization of the striatal cell, cortico-striatal synapses show potentiation rather than depression [33].

When learning a new skill, some instances of the behavior are more successful than others. Improvement requires increasing the probability of the successful instances and decreasing the probability of unsuccessful instances. The striatum is ideally suited for such learning because the conditions required for LTP at cortico-striatal synapses closely match the conditions for reinforcement learning [34–36]. An extensive literature shows that dopamine cells exhibit phasic increases in discharge rate following unexpected primary or conditioned rewards and depress their discharge below baseline following an unexpected failure to receive a reward [34,36]. It has been proposed that cortico-striatal synapses active at the time of phasic dopamine activation are strengthened, and that cortico-striatal synapses active in the absence of phasic dopamine undergo LTD [37–38].

As a more concrete example, consider the network shown in Figure 3. Glutamate inputs to the striatum from some area of sensory association cortex converge on striatal output neurons (R_A and R_B) that eventually lead to particular behavioral responses [37]. During reinforcement (S-R) learning, dopamine-mediated promotion of LTP when an animal encounters a less-than-fully-predicted primary or conditioned reward may strengthen the currently-active striatal synapses. In this model, the S-R striatal synapse that is active at the time the dopamine-mediated reward signal arrives (S_B to R_A) continues to strengthen with repeated reinforcement trials, until an asymptotic level of synaptic strength is reached. S-R synapses that become activated in the absence of the phasic dopamine-reward signal (S_B to R_B) undergo LTD, reducing the likelihood that the same sensory input will depolarize the post-synaptic output cell enough to produce neuronal activation in the future. As this model predicts, sensory responses are acquired in striatal cells over the course of reinforcement learning, and dopamine depletion prevents the acquisition of these responses [37,39].

It has also been proposed that response selection in the striatum may be enhanced by lateral inhibition (not depicted in Figure 3), which is mediated by the inhibitory synaptic contacts that striatal GABAergic output neurons make with one another via local axon collaterals [38,40–41]. Through such a mechanism, strongly activated striatal neurons could inhibit potential competitors from becoming activated. With each successive reinforced trial, dopamine-mediated LTP should progressively increase the ability of active striatal output cells to exert lateral inhibition on other striatal cells, eventually producing a highly selective activation pattern that includes only those striatal cells whose output closely corresponds to the successful behavioral response under current stimulus conditions. As noted above, the number of striatal

cells showing task-related activations decreases dramatically over the course of operant S-R learning, as focused activity time-locked to task-related events emerges in a select group of striatal cells [23–24]. These data may reflect LTD in non-reinforced striatal input-output synapses and/or the increasing lateral inhibitory strength exerted by those cells that have undergone learning-related LTP.

Dopamine and Habit Expression

Dopamine plays a role not only in reinforced learning, but also in the expression of previously-learned behaviors [42–44]. Of particular interest, dopamine appears to play a diminishing role in behavioral expression over the course of extended training [38,42]. For example, some Parkinson's patients are able to emit an automatic motor response when presented with a familiar visual cue (e.g., kicking a ball), despite difficulties in initiating novel voluntary movements [45]. As another example, blockade of D1 receptors strongly disrupts rats' performance of a simple Pavlovian approach response to a sensory cue during early stages of training, but has little or no disruptive effect in rats that receive extended training prior to dopamine antagonist challenge [46–47].

Reduced dopamine mediation of a sensory-motor behavior with extended training can be explained in at least two ways. One possibility is that with extended training, the behavioral response shifts to mediation by non-dopamine target areas, and therefore becomes less subject to dopamine modulation [48; see next section for more detail]. Another possibility, however, is that over-learned responses are controlled by the same neurons that mediate initial learning, but that dopamine plays a declining role in modulating that expression. For example, cortical glutamate input to striatal cells may depend on activation of dopamine D1 receptors to amplify the strength of task-relevant input signals (S_B to R_A in Figure 3) during early stages of learning [49–50]. During later stages of learning, these same cortico-striatal synapses may become so efficient that dopamine facilitation of glutamate transmission is no longer necessary for normal striatal discharge and behavioral responding. This hypothesis nicely accounts for the enhanced activation of a reduced set of neurons in the sensorimotor striatum that has been observed following extended training [23–24], but it does not account for results showing that disconnection of the sensorimotor striatum has little effect on the expression of highly practiced skills [30–31].

The Role of Cortex in Habit Expression and Automaticity

Many neuroimaging studies have examined cortical activity during practice on a cognitive or motor task (for a review, see [51]). Depending on the task, some studies have reported a general decrease in cortical activity [52–55], a few have reported increases [56–59], and some have reported a more complex redistribution where activity increases in some areas and decreases in others [60–62]. Kelly and Garavan [51] noted that decreases are often observed in prefrontal and parietal areas associated with attentional control, increases have generally been seen in premotor and motor areas during motor tasks, and a redistribution is often found in tasks where different cognitive processes operate during early and late training [61,63]. The decreases support the classical view [3] that cortical activity diminishes as a behavior becomes 'automatic', but clearly the many increases and redistributions that have been reported do not. For instance, extended training of a motor skill leads to *increased* rather than decreased task-related activation of the sensorimotor cortex [56–58,64–65], and extended training (e.g., for two years) induces learning-related changes in task-related activity of neurons in primary motor cortex [4]. Furthermore, well-acquired S-R habits become "goal-directed" following inactivation of the infralimbic cortex, suggesting that this region of the medial prefrontal cortex plays a critical role in habit expression [66].

A modern reformulation of the classical view, however, might be that if the development of automaticity involves a transition from goal-directed to S-R mediated responding, then cortical regions that contribute to representations of expected outcome value should play a diminishing role in behavioral performance. In fact, considerable evidence supports this prediction. For example, lateral and ventromedial prefrontal cortices, which both encode the value of expected outcomes [67–68,37] do show reduced activation with extended training [69–70].

Given that there is a prominent dopamine projection into frontal cortex, one might expect that plasticity at cortical-cortical synapses should follow reinforcement learning rules similar to those described above for cortico-striatal synapses. A necessary feature of any reinforcement training signal, however, is high temporal resolution. If the first response is correct then dopamine must be released into the relevant synapses quickly, before the critical traces disappear. But after the correct synapses have been strengthened, it is also essential that excess dopamine be quickly cleared from the synapse. If it is not, and the next response is an error, then the residual dopamine will strengthen inappropriate synapses – namely, those responsible for producing the incorrect response. This would undo the beneficial learning that occurred following correct responses, and thereby prevent skill learning.

Within the striatum, dopamine re-uptake is exceptionally fast (e.g., [71]). In contrast, in frontal cortex, because of low concentrations of the dopamine reuptake molecule DAT, it takes much longer to clear dopamine from synapses (for reviews, see, e.g., [72–74]). For example, the delivery of a single food pellet to a hungry rat elevates dopamine levels in prefrontal cortex for approximately 30 minutes [75]. For this reason, it has been proposed that this poor temporal resolution effectively rules out dopamine as a trial-by-trial reinforcement training signal in cortex [48]. Instead, although dopamine may facilitate cortical LTP, there is much evidence that synaptic plasticity at cortical-cortical synapses follows classical two-factor Hebbian learning rules (for a review, see [76]).

Many sensory association areas of cortex project directly into premotor cortex. Ashby et al. [48; see also 77–78] proposed that these cortical networks, by themselves, are incapable of skill learning because of the absence of reinforcement learning at cortical-cortical synapses. Even so, they proposed that via reinforcement learning, a subcortical path through the striatum learns to activate the correct post-synaptic target in premotor cortex, which allows the appropriate cortical-cortical synapses in the premotor cortex to then be strengthened via Hebbian learning (because the product of pre- and post-synaptic activations will be greatest at the correct synapse). In this way, control is gradually passed from the slower path through the basal ganglia to the faster cortical-cortical path. Thus, according to this model, the development of motor skill automaticity is a gradual process via which control is passed from the subcortical procedural-learning systems to purely cortical networks that connect sensory association areas of cortex with premotor cortex. In other words, rather than to serve as a long-term store of procedural knowledge, a primary function of the basal ganglia may be to train cortical-cortical representations that mediate automaticity. Note that this theory accounts for results showing that automatic behaviors are striatum- and dopamine-independent, but it does not account for the results reviewed earlier showing activation of sensorimotor striatum after automaticity has developed.

Concluding remarks

Understanding the neural basis of automaticity is tremendously important, not only from a theoretical perspective, but also because many societal problems are due to maladaptive automatic behaviors. For example, a complicating factor in treating drug addiction is that many aspects of drug-seeking behaviors become automatized [79–82]. This is also true of many other harmful habits. Thus, an accurate model of how automaticity develops could lead to new

behavioral and pharmacological treatments that might potentially benefit a great many individuals.

Automaticity is a challenging construct to study. The experiments are expensive in both time and resources. Theoretically, the problem is difficult because the development of automaticity is characterized by changes in widely distributed neural networks, rather than in some single brain region. Despite these obstacles, the past few years have seen some dramatic breakthroughs in our understanding of this important phenomenon. The classical view has been gradually replaced by other, more subtle accounts. The most pressing open questions (see Box 3) may be to resolve the apparent conflict between results showing an enduring role for the sensorimotor striatum with the results suggesting that automatic behaviors become striatum- and dopamine-independent.

Box 1 – Behavioral Criteria of Automaticity

How do we know that a behavior is automatic? In many cases, researchers have assumed that a behavior is automatic if the subject has received significantly more training than is required for accuracy and speed to asymptote. Although this criterion is commonly used, other more formal tests have been proposed. In cognitive science, the most widely used criteria come from Schneider and Shiffrin [83], who suggested that a behavior should be considered automatic if 1) the triggering sensory events almost always elicit the behavior, and 2) the behavior can be executed successfully while the subject is simultaneously engaged in some other secondary task.

In the animal learning literature, the term “automatic” is rarely used. Instead, the focus is on a similar concept, called a “habit”, which is a behavior that is under the control of stimulus-response (S-R) mechanisms. In other words, a habit is a behavior that is elicited by environmental stimuli to which it has become strongly tied. This is contrasted with ‘goal-directed’ actions observed during earlier stages of learning. A behavior is goal-directed if the rate or likelihood of the behavior is decreased by a) reductions in the expected value of the outcome, and b) reductions in the contingency between the action and the outcome. In contrast, habits are behaviors that have become insensitive to reductions in both outcome value and response-outcome contingency [19,84]. For example, if the bar pressing of a hungry rat is goal directed, then allowing the animal to eat to satiety will greatly decrease its subsequent bar pressing. However, if the bar-pressing is a habit, then feeding the animal should have little effect on its subsequent bar pressing.

Use of these different behavioral criteria is strongly segregated across the cognitive science and animal-learning fields. We know of no systematic attempts to compare the different criteria, or even any examples where both sets of criteria were used in the same study. Thus, it is unknown whether the two sets of criteria would identify the same or different behaviors. Finally, it is also important to note that even after these behavioral criteria are met, it is possible that further practice on the task could continue to induce changes in the underlying neural representations. For example, almost daily practice of a task over the course of many years may cause continual changes in the mediating neural circuitry [4]. If so, then the shift from “goal-directed” to “habit” behavior may be continuous rather than discrete.

Box 2 – The Striatum

The basal ganglia are a large collection of subcortical nuclei. The interconnections among the most important basal ganglia structures are shown in Figure I. The striatum is a major input structure within the basal ganglia that includes two parallel structures known as the caudate nucleus and the putamen. The striatum receives massive and highly convergent input from almost all of cortex. It can be subdivided into associative and sensorimotor

regions depending on the origins of this input. Roughly speaking, the associative striatum, which includes all of the caudate nucleus and the anterior putamen, receives input from sensory association areas in the temporal lobes and from prefrontal cortex. In contrast, the sensorimotor striatum, which includes all of the putamen except its most anterior portion, receives input from the parietal lobes and from motor and premotor cortex. In rodents, the caudate and putamen are undifferentiated, and the associative striatum includes the dorsomedial striatum, whereas the sensorimotor striatum includes the dorsolateral striatum.

The major output structures of the basal ganglia are the internal segment of the globus pallidus (GPi; called the entopeduncular nucleus in non-primates) and the substantia nigra pars reticulata (SNpr). These structures receive inhibitory projections from the striatum and send inhibitory projections to the thalamus (see Figure I). Thus, striatal activation tends to disinhibit the thalamus, which tends to increase activation in targeted regions of cortex. The basal ganglia also include several prominent dopamine producing areas, including the substantia nigra pars compacta, which projects predominantly back to the rest of the basal ganglia, and the ventral tegmental area, which projects to all of frontal cortex and limbic areas (e.g., amygdala, hippocampus, nucleus accumbens). In Parkinson's disease, these dopamine producing cells die and as a result, all parts of the striatum function abnormally.

A widely-held view is that the basal ganglia are topographically segregated into a set of parallel loops from cortex, through the basal ganglia, and back to the originating area of cortex via the thalamus [25,85]. Thus, for example, sensorimotor regions of GPi receive inputs from sensorimotor regions of striatum, and project eventually to motor/sensorimotor areas of cortex. For more details on the striatum and basal ganglia see, e.g., [26].

Box 3 – Outstanding Questions

- What is the relationship between the criteria commonly used to assess whether a behavior is automatic versus a habit? Is it possible to meet one set of criteria but not the other?
- Are there any functional differences in how automaticity develops in motor versus cognitive tasks? Only a few studies have addressed this important question [86].
- Mechanisms of cortico-striatal LTP and LTD (as described here) may explain the gradual reduction in the number of striatal neurons that show task-related activation over the course of training, but they do not seem to predict a shift in control from the associative to the sensorimotor striatum. What type of mechanism (s) could account for such a shift?
- What role does the prefrontal cortex play in automaticity? Lesions to the rat homologue of ventral medial prefrontal cortex (vmPFC; i.e., infralimbic cortex) have been reported to disrupt automatic behaviors [66]. This cortical region sends excitatory projections to the patch compartments of the striatum, which inhibits dopamine producing cells, so one untested hypothesis is that the primary effect on automatic behaviors of lesions to vmPFC is to increase striatal dopamine levels.

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Figure 1.

Responses of one neuron in the associative striatum and one neuron in the sensorimotor striatum of a monkey during the performance of a new and an old motor sequence. The monkey's task was to depress a sequence of ten buttons in a predetermined order. In each case, the recordings shown here were to a movement from the key with the white dot to the black key. The vertical lines indicate the time at which the key with the white dot was depressed. In the associative striatum, more than twice as many neurons were found that responded to new sequences more strongly than to old sequences (i.e., 40% versus 18%), whereas in the sensorimotor striatum almost twice as many neurons were found that responded more strongly to old sequences than to new sequences (i.e., 30% versus 17%) (adapted with permission from [14]).

Figure 2.

Spike histograms from single cells in the sensorimotor striatum of a rat in a task in which it is trained to lever press to a tone (reprinted with permission from [22]). The vertical line denotes the time of the lever press. Note that the time axis is defined relative to the lever press, rather than the tone onset.

Figure 3.

Cortico-striatal inputs (S_A , S_B) coding for sensory events synapse upon striatal output neurons that eventually lead to behavioral responses (R_A , R_B). A behavioral response (R_A) leading to delivery of a reward or reward-predicting stimulus causes a phasic increase in midbrain dopamine activity and a consequent increase in striatal dopamine release. Dopamine-mediated LTP strengthens the currently-active synapses (S_B to R_A ; bottom diagram, bold box in striatum) and increases the likelihood that the reward-procuring behavioral response (R_A) will occur in response to the same sensory stimulus (S_B) in the future. (Reprinted with permission from [37]).

Figure 1.

(Box 2) Schematic diagram of the basal ganglia and its afferents and efferents. Black lines terminating in arrowheads are excitatory, those terminating in filled circles are inhibitory, and gray lines terminating in squares are dopaminergic. All structures are part of the basal ganglia except cortex and thalamus. PFC = prefrontal cortex; SMA = supplementary motor area; GP_e = external segment of the globus pallidus; GP_i = internal segment of the globus pallidus; MD = medial dorsal nucleus; VA = ventral anterior nucleus; VL = ventral lateral nucleus; STN = subthalamic nucleus; SN_{pc} = substantia nigra pars compacta; SN_{pr} = substantia nigra pars reticulata.