SUPPLEMENTARY MATERIAL

1. Detailed description of the equations

This section includes detailed description of the newly added equations of the extended GODIVA model (equations 15 to 25), as well as the equations modified from the original GODIVA model (equations 13 and 14). Detailed descriptions of the equations of the original GODIVA model are given elsewhere (Bohland, Bullock, & Guenther, 2010). The list of the equations of the original GODIVA model can be found in Appendix A, and the main variables they use are given in Appendix B.

Each section below details and discusses the equations that correspond to a specific brain region of the BG-vPMC loop, except for the last section that concerns with sound/syllable repetitions. Most equations have the form of shunting equations (Grossberg, 1973): the A parameter indicates the strength of the cell's spontaneous decay of activity; the B parameter controls how high the excitatory input can bring the cell activity; and the C parameter indicates the strength of the inhibitory input to the cell, if such input exists. Exceptions are the equations for cells representing spontaneously-firing neurons (e.g., pallidal cells). Because the activities of these cells should not decay below the level fixed by B, their equations are missing a spontaneous decay term (and the A parameter). Furthermore, some equations are missing the C parameter despite having an inhibitory term; in these equations, the C parameter is assumed to equal 1. The variables used by the equations of the BG-vPMC loop are given in Appendix C.

1.1. Ventral Premotor Cortex

Each idealized column of the vPMC represents one well-learned syllable. The activity in the plan cells indicates the degree of match between the set of active phonological cells in the IFS choice layer (the forthcoming phonological syllable) and the stored motor programs associated with the SSM columns (see Bohland et al., 2010). The match is computed via an inner product of the IFS choice-layer inputs with synaptic weights, which were pre-set such that the correct well-learned syllable program would have the highest activation in the planning layer of the vPMC. Nevertheless, other syllable programs that have a partial match to the phonological syllable will be active as well, although to a lesser degree. Changes in the activity of the SSM plan cell, r_k, are governed by the differential equation:

$$\dot{r}_k = D_r\left[-A_r r_k + \left(B_r - r_k\right)\left(\sum_i \sum_j Z_k^{ij} y\left(\left[q_{ij} - \theta_q\right]^+\right) + \left[r_k - \theta_r\right]^+\right) - r_k\left(\sum_{n \neq k} r_n\right)\right] \qquad (13)$$

Here the dot symbolizes the time derivative, thus $\dot{r}_k \equiv \frac{dr_k}{dt}$. The equation is identical to equation (13) from Bohland et al. (2010), other than the D_r parameter, which controls the rate of network dynamics. By setting this parameter below 1, we configure the planning layer of the vPMC to reach equilibrium slower than in the original GODIVA model, thus making the model more realistic. For a detailed description of the equation, see Bohland et al. (2010).

The SSM plan cell r_k gives specific excitatory input to the SSM choice cell s_k within the same idealized cortical column. The equation for s_k was given in the original GODIVA model, but is modified for the extended GODIVA model. Its activation is now given by:

$$\dot{s}_k = A_s s_k + \left(B_s - s_k\right)\left(r_k + F_{excite} w\left(d_k^{Thal}, s_k\right)\right) I - s_k\left(\sum_{j \neq k} F_{inhibit} w\left(d_j^{Thal}, s_j\right) + \Omega_{reset}\right) \qquad (14)$$

The dynamics are such that the most active well-learned syllable's program in the planning layer is selected in the choice layer (Bohland et al., 2010). This is due to the input from each SSM plan cell r_k to the SSM choice cell in the same cortical column, and the winner-take-all competition between the choice cells: only one cell "wins" the competition, while all the "loser" cells are quenched. This competition is mediated by a self-excitation term, $F_{excite} w\left(d_k^{Thal}, s_k\right)$, and lateral inhibition term, $F_{inhibit} w\left(d_j^{Thal}, s_j\right)$, with the parameters F_{excite} and $F_{inhibit}$ controlling the balance between the two terms. For competition to resolve under normal speaking conditions, F_{excite} should be at least twice as large as $F_{inhibit}$. Note that the self-excitation also ensures that the "winner cell" will maintain its activation regardless of any changes in its inputs. I is a binary [0 or 1] input arriving from brain regions not modeled by the extended GODIVA model; when set to 1 (in the simulations reported here, at t=600 ms), it initiates the production of the first syllable in the sequence. If necessary, resetting it to 0 will stop the production of the sequence before it is over. Ω_{reset} is a suppression signal which arrives from the DIVA model when a "reset" is initiated (see Civier, Tasko, & Guenther, 2010).

Both the self-excitation and the lateral-inhibition terms in (14) include the function w, which by itself is a multiplication of two faster-than-linear activation functions (cf. Grossberg,

1973) whose neural correlates may be spike rates that vary non-linearly with membrane potential. The function *w* is given by:

$$w(d^{Thal}, s) = u\left(\left[d^{Thal} - T_d\right]^+\right) z\left([s - \theta_s]^+\right) \qquad (15)$$

where the thalamic input d^{Thal} is thresholded by the thalamic activation threshold T_d (here $[\ ]^+$ indicates half-wave rectification: it yields the value of the argument or zero, whichever is larger) and strongly amplified via the function $u(x)=x^4$, and the SSM plan layer input *s* is thresholded by the low threshold θ_s (to prevent very weak candidates from affecting the competition between the major candidates) and weakly amplified via the function $z(x)=x^{1.5}$. The modulation by the thalamus biases competition in the vPMC choice layer in favor of likely winners, as discussed in Section 2.2.1 of the main text. Cortical cells whose corresponding thalamic cells' activation levels are below T_d are totally deprived of self-excitation, and will quickly be eliminated from competition. Cortical cells whose thalamic cell activation levels are above T_d will be excited in proportion to the activation level of the thalamic cell.

When the competition yields a "winner" SSM cell in the choice layer, the cell excites its corresponding thalamic cell, which then maintains the SSM choice cell's self-excitation. In the absence of a reset signal Ω_{reset}, the deletion of this SSM choice cell activity is possible only when the thalamic cell is pushed below the threshold T_d by inhibition from the indirect pathway. Because the natural decay of SSM choice cells is not strong, it might be that stopping self-excitation is not sufficient to quickly terminate choice cell activation. However, the baseline lateral inhibition in the choice layer ensures prompt termination of activity.

1.2. Putamen

The activity of the direct pathway striatal projection neurons (putamen D1R cells) in BG channel *j* is given by:

$$\dot{b}_j^{D1} = -A_{D1} b_j^{D1} + \left(\beta_{D1} B_{D1} - b_j^{D1}\right) r_j - C_{D1} b_j^{D1} \left(\sum_{k \neq j} b_k^{IN}\right) \qquad (16)$$

where r_j is the SSM plan cell activation that provides input to the BG loop, and the product β_{D1} B_{D1} controls the maximum strength of the excitatory term. Whereas B_{D1} is a constant, β_{D1} is determined by the amount of dopamine that binds to the D1Rs of the putamen (cf. Frank, 2005), which has the value of 1 when dopamine levels are normal. Because SSM plan cells are the main

inputs to the putamen D1R cells, the activations of the latter cells reflect the pattern of activity in the former ones. This pattern of activity depends on the next syllable in the sequence: The SSM plan cells, and thus the putamen D1R cells, that are active at each phase of the sequence represent the set of similar syllables from which the next syllable is selected. This property of the model is consistent with the findings that cells in the putamen are selective to the phase of an executed motor sequence (Filatova, Orlov, Tolkunov, & Afanas'ev, 2005; Ueda & Kimura, 2003).

The cell b_j^{D1} also receives feedforward lateral inhibition from the GABAergic striatal interneurons (putamen IN cells) b_k^{IN} in the other BG channels ($k \neq j$). Since the short-term dynamics of the putamen IN cells are fast (Koos & Tepper, 1999), their dynamics are not explicitly modeled (see Brown, Bullock, & Grossberg, 2004, p. 489). Instead, the activity of each putamen IN cell b_k^{IN} is simply defined in terms of the input it receives from its corresponding SSM plan cell:

$$b_k^{IN} = G_{IN} r_k \qquad (17)$$

where G_{IN} is a parameter that controls the gain of cortical input to the putamen IN cells. Notice that in contrast with the striatal projection neurons, striatal interneurons are not inhibited by other striatal neurons (Koos & Tepper, 1999).

The activity of the indirect pathway striatal projection neuron (putamen D2R cell) in BG channel j is given by:

$$\begin{aligned} \dot{b}_j^{D2} = & -A_{D2} b_j^{D2} \\ & + \left((1/\beta_{D2}) B_{D2} - b_j^{D2} \right) \left((\lambda_{WMF})^2 m_j(\dot{M}) p\left(\left[s_j - T_s \right]^+ \right) + G_{D2} p\left(\left[s_j - \theta_s \right]^+ \right) \right) \\ & - C_{D2} b_j^{D2} \left(\sum_{k \neq j} b_k^{IN} \right) \end{aligned} \qquad (18)$$

where β_{D2} and B_{D2} control the maximum strength of the excitatory term. B_{D2} is constant, whereas β_{D2} represents the amount of dopamine that binds to the D2Rs of the putamen. As with β_{D1}, β_{D2} equals 1 at normal dopamine levels. In contrast to the direct pathway, increased binding in the indirect pathway weakens the excitatory term (cf. Frank, 2005).

Term m_j in (19) is a function that detects when articulation of the syllable coded by SSM choice cell j is about to terminate. In particular, it calculates the match between the motor commands $\dot{M}$ currently executed by vMC (see Guenther, Ghosh, & Tourville, 2006), and a long term memory for the motor commands that predict imminent termination of this specific syllable. If the match is high, such that the articulation of the syllable is about to terminate, then $m_j(\dot{M}) = 1$, and the putamen D2R cell becomes strongly active. This strong activation ultimately inhibits the thalamus and undercuts SSM cell activation. While the match remains low, i.e., $m_j(\dot{M}) = 0$, the syllable is still far from terminating, and the putamen D2R cell activation remains low. Because the putamen receives copies of motor commands via collaterals of vMC's efferents, the strength of the striatal activation generated during a match depends on the corticostriatal projection's integrity, which is quantified by λ_{WMF} in the above formula. These fibers are presumed to be impaired in stuttering.

The model does not attempt to explicate the neural site or biophysical bases of the match computation or the learning of long term memories for motor commands that predict imminent terminations (See Section 2.2.2 of the main text). Instead, m_j is calculated algorithmically. In particular, (each motor command can be described by sixteen values, corresponding to the commands sent to the various articulators, see Guenther et al., 2006). Comparing the current motor command with a region in the 16 dimensional articulatory command space, instead of a single point in that space, makes the detection of syllable completion robust. The following formula achieves this:

$$m_j(\dot{M}) = \max\left(\left((H_j^M - L_j^M) - abs(\dot{M} - L_j^M) - abs(\dot{M} - H_j^M) + 1\right), 0\right) \qquad (19)$$

where L_j^M and H_j^M code for the low and high bounds, respectively, of the 16 dimensional region associated with the syllable coded by the idealized vPMC column *j*. Any motor command that falls inside this region signals this syllable's imminent termination

To prevent putamen D2R cells of other syllables from mistakenly detecting completion based on the motor commands of the currently executed syllable, the function m_j in (18) is multiplied by the term $p\left(\left[s_j - T_s\right]^+\right)$ that ensures that only the putamen D2R cell that corresponds to the current syllable is primed. The term for a specific channel *j* equals 1 only if

the current syllable corresponds to that channel, because the SSM choice cell for the current syllable is the only one with activation above T_s, and p is a binary function defined by:

$$p(x) = \begin{cases} 1 & if\ x > 0 \\ 0 & otherwise \end{cases} \qquad (20)$$

The term $G_{D2} p\left(\left[s_j - \theta_s\right]^+\right)$ in (18) enables weak (due to the parameter G_{D2} being set below 1) activation of the indirect pathway during syllable selection. It represents excitation of all putamen D2R cells whose corresponding SSM choice cells have supra-threshold activity (θ_s is a low threshold on SSM choice cell activity, s_j). This scales the inhibition that the indirect pathway exerts on the thalamus, such that the total inhibition is proportional to the number of SSM cells competing in the vPMC choice layer. The described mechanism is important in ensuring that the thalamus properly expresses the gradient in the activation levels of the SSM plan cells, i.e., the contrasts between the well-learned syllables competing for execution. If the gradient in the thalamus is lost, the thalamus excites several SSM choice cells to the same degree, and selection of the motor program for the next syllable cannot be expedited (similarly to the simulations of the DA GODIVA version).

Like the putamen D1R cells, also the putamen D2R cells, b_j^{D2}, receive feedforward inhibition from the putamen IN cells b_k^{IN} in the other BG channels ($k \neq j$). Since the putamen IN cells inhibit the putamen D2R cell to a lesser extent than they inhibit the putamen D1R cells (see Section 2.2.1 of the main text), the parameter C_{D2} in equation (18) is set to be smaller than the parameter C_{D1} in equation (16).

1.3. Globus pallidus

The activation of pallidal cells, like that of the striatal cells, is dependent on the phase of the sequence (Mushiake & Strick, 1995). The putamen D1R cells b_j^{D1} are those informed by the cortex regarding the next element in the sequence; information which they then transmit to the GPi. The putamen D1 projection cells connect to GPi cells within the same BG channel via an inhibitory synapse. The activity of the GPi cell c_j, which is itself inhibitory to a corresponding thalamic cell d_j, is given by:

$$\dot{c}_j^{GPi} = \left(B_{GPi} - c_j^{GPi}\right) - c_j^{GPi}\left(L_{direct} b_j^{D1} + L_{indirect} c_j^{GPe}\right) \qquad (21)$$

where B_{GPi} controls the level of spontaneous tonic activation of the GPi cell, and L_{direct} and $L_{indirect}$ are parameters that control the relative influence of the direct and indirect pathways on the GPi (in the current implementation, the influence of the direct pathway is stronger). The tonic activation of the GPi (Delong, 1971) is essential to the proper functioning of the model. It permits changes of GPi cell activation to mediate both net excitation and inhibition of the thalamic cell. When the putamen is silent, the tonic activation of the GPi cell moderately inhibits the thalamus. When the GPi cell is inhibited by the putamen D1R cell, its activation drops below tonic levels, and the thalamic cell it targets is excited due to disinhibition. When the tonic inhibition of the GPi cell by the GPe is removed, the GPi cell activation rises above tonic levels, and the thalamic cell it targets is inhibited.

In the real BG, the indirect pathway has two branches: {putamen D2R cells to GPe to GPi}, and {putamen D2R cells to GPe to **STN** to GPi}. For simplicity, the STN (subthalamic nucleus) stage was omitted. However, greater divergence has been reported in the STN to GPi projection than in the GPe to GPi projection. This greater divergence implies reduced specificity in the indirect pathway, relative to the direct pathway. To accommodate this, and to show that the model operation does not require high specificity in the indirect pathway, the GPe stage was implemented as a kind of hybrid GPe/STN stage. To achieve this, and reduced signaling specificity, each putamen D2R cell b_k^{D2} sends inhibitory inputs to the GPe cells of *all* channels. The activity of the GPe cell c_j^{GPe}, which is itself inhibitory to the GPi cell within the same channel, is given by:

$$\dot{c}_j^{GPe} = \left(B_{GPe} - c_j^{GPe}\right) - c_j^{GPe}\left(\sum_k b_k^{D2}\right) \qquad (22)$$

where B_{GPe} controls the level of spontaneous tonic activation of the GPe cell, and the inhibitory inputs in the last term arrive from all the putamen D2R cells (see Section 2.2.2 of the main text).

At rest the GPe is tonically active, moderately inhibiting the GPi. However, when one of the putamen D2R cells is activated, the GPe is inhibited, and the degree of inhibition it exerts on the GPi will be reduced. Thus, the putamen D2R cells can excite (by disihibition) the GPi via the GPe. This excitation, however, is transient: new feedforward commands are constantly being sent to the articulators, and they fall within the "match" region for only a short time. Such

transient GPi excitation can explain the spike in pallidal activity which precedes all but the first in a sequence of hand movements (Brotchie, Iansek, & Horne, 1991). Because the GPi inhibits the thalamus, this spike will be translated to transient inhibition of the thalamus.

To summarize, in the extended GODIVA model the BG are capable of both exciting (disinhibiting) and inhibiting the thalamus (Mink & Thach, 1993; Wichmann & Delong, 1996). The putamen D1R cells excite thalamic cells via the GPi (the direct pathway), while the D2R putamen cells inhibit thalamic cells via the GPe-GPi route (the indirect pathway).

1.4. Thalamus

The activity of a thalamic cell d_k is given by:

$$\dot{d}_k^{Thal} = \left(B_d - d_k^{Thal}\right)\left(r_k + \left[d_k^{Thal} - \theta_d\right]^+ + z\left(s_k\right)\right) - C_d d_k^{Thal}\left(c_k\right) \qquad (23)$$

where r_k is the activation of the SSM plan cell in idealized column k, s_k is the activation of the SSM choice cell in idealized column k, and c_k is the activation of the GPi cell in the same BG channel. Because each thalamic cell excites itself (d_k^{Thal} is included in the excitatory term), all thalamic cells initially excited by the cortex will ultimately converge, to a constant activation level. During speech, however, the putamen is not silent. The putamen cells are excited by the cortex, and modulate thalamic cells by using inhibition and disinhibition via the GPi. The last excitatory input to the thalamus, $z(s_k)$, arrives from the SSM choice cells in the vPMC. Being much faster-than-linear, it ensures that activity in the thalamic cells of winning cortical cells persists until inhibited by the indirect pathway. This inhibition must push the thalamic cell activation below θ_d in order to ensure its deactivation. Our proposal that projections to the thalamus from superficial (planning) and deep (choice) layers differ in nature is based on recent evidence from Zikopoulos and Barbas (2007).

1.5. Sound/syllable repetitions

Computationally, sound/syllable repetitions due to impaired readout of motor commands are simulated by lowering α_{ff} (default value 0.85), the DIVA model's parameter representing the strength of feedforward motor commands, and raising α_{fb} (default value 0.15), the parameter representing the extent to which sensory-feedback is utilized for motor control (see Civier et al., 2010). The modified parameters are given by:

$$\alpha_{ff} = 0.85\frac{s(t)}{T_s} \qquad (24)$$

$$\alpha_{fb} = 1 - \alpha_{ff} \qquad (25)$$

with $s(t)$ being the activation of the SSM choice cell for the next syllable at time t, and T_s being the cortical selection threshold.

2. Evaluation of the hypotheses based on a literature review

This section presents a literature review-based summary of each hypothesis's strengths and weaknesses. A major flaw in the WMF hypothesis, as implemented here, is that it does not explain the subtle non-oral motor-control deficits observed in PWS (for review, see Bloodstein & Ratner, 2008; Max, 2004; Max et al., 2004). If the white-matter impairment is indeed restricted to those corticostriatal projections that carry copies of motor commands for syllable production, the only BG loop affected should be the one that handles the selection and initiation of syllables; dysfunction in this loop would not be expected to disrupt non-oral movements. Nevertheless, the fact that the white matter abnormality was detected at different locations in the left precentral gyrus (see Cykowski et al., 2010) raises the possibility that the impairment affects several neighboring BG loops after all. Such non-specific white matter impairment could account for the non-oral deficiencies of PWS, but raises the question of why the motor behavior most affected in the stuttering disorder is speech. In summary, the WMF hypothesis does not account for the full range of behaviors associated with stuttering.

The DA hypothesis, in contrast, does not lead to similar caveats, as the non-specificity of dopamine implies that excessive levels of the neurotransmitter affect all BG loops. This effect, however, is not uniform: dopamine seems to have the greatest effect on those BG loops involved in oral motor control (Nevet et al., 2004). This is compatible with the extended GODIVA model, and it draws attention to a subtle, oft-overlooked fact: dopamine can boost signal in the direct pathway both at dendrites, in striatum, and at axon terminals, in GPi and SNr (substantia nigra pars reticulata, which by virtue of circuit embedding and histology may be considered a separated portion of the "internal pallidum", GPi). In the exposition above, we emphasized the role of DA release in striatum. However, synchronous DA release in the GPi and SNr acts synergistically (with striatal release) to boost GABA release from the axon terminals of D1R putamen cells, via presynaptic D1Rs located on such terminals. This can be especially potent in the SNr, through which the oral-motor-control BG loops pass (Delong, Crutcher, & Georgopoulos, 1983; Gracco & Abbs, 1987), because the putamen cells' D1 axon terminals that reach many SNr cells receive DA that is released directly from the dendrites of DA cells whose somas reside in the nearby SNc (substantia nigra pars compacta Abercrombie & Deboer, 1997;

Cheramy, Nieoullon, & Glowinski, 1979; Conde, 1992). Indeed, the SNr has one of the densest expressions of D1Rs in the brain (Abercrombie & Deboer, 1997).

An additional strength of the DA hypothesis is that it implies greater normal variability across episodes of producing a given syllable (than the WMF hypothesis). Because dopamine levels fluctuate over time, the DA hypothesis can account for day-to-day variability in stuttering (Van Riper, 1982). Furthermore, because dopamine release is modulated by emotional state and the novelty of tasks and situations (psychophysiological adaptation), the DA hypothesis can in principle account for the role of emotions in stuttering (Alm, 2004, pp. 332, 344, 359; Smith, 1999; Zimmermann, 1980), as well as for the fluency enhancing effect of repeating the same utterance several times, i.e., the adaptation effect (Wingate, 1966, 1972). A caveat regarding this hypothesis is the current scarcity of direct experimental evidence. We know of only one study, using FDOPA-PET imaging, which showed direct evidence for elevated dopamine levels in PWS (Wu et al., 1997). Although the findings were highly significant, despite the small sample size, definitive conclusions must await replications or convergent data based on further studies (e.g., Rastatter & Harr, 1988).

A promising research methodology that has already provided some convergent data is gene association analysis. Lan et al. (2009) found a mutant gene that is associated with increased susceptibility to stuttering (but see Kang et al., 2011), and is likely to be associated with elevated DA levels as well. Whereas direct evidence for the later association is lacking, indirect evidence is provided by the finding that the same gene mutation predicts lower D2R binding in the striatum (Hirvonen et al., 2004, 2005). A possible dopamine excess due to the mutant gene[1] would lead to greater occupancy of D2Rs by DA, and according to the occupancy model (Laruelle, 2000), to the observed reduction in the availability of D2Rs for binding by other ligands. This indirect evidence should be taken with caution, though, because dopamine tone is only one of the factors that modulate the binding potential of D2Rs (Hirvonen et al., 2009).

[1] It is still unclear how the mutant gene can lead to DA excess, but one hypothesis is that the D2 autoreceptors on DA terminals (and probably also on putamen cells) may not be built normally (but see Hirvonen et al., 2009). With the auto-regulatory function of these inhibitory receptors being impaired, too much DA release is expected.

REFERENCES FOR SUPPLEMENTARY MATERIAL

Abercrombie, E. D., & Deboer, P. (1997). Substantia nigra d1 receptors and stimulation of striatal cholinergic interneurons by dopamine: A proposed circuit mechanism. *Journal of Neuroscience, 17*(21), 8498-8505.

Alm, P. A. (2004). Stuttering and the basal ganglia circuits: A critical review of possible relations. *Journal of Communication Disorders, 37*(4), 325-369.

Bloodstein, O., & Ratner, N. B. (2008). *A handbook on stuttering* (6th ed.). Clifton Park, NY: Thomson/Delmar Learning.

Bohland, J. W., Bullock, D., & Guenther, F. H. (2010). Neural representations and mechanisms for the performance of simple speech sequences. *Journal of Cognitive Neuroscience, 22*, 1504-1529.

Brotchie, P., Iansek, R., & Horne, M. K. (1991). Motor function of the monkey globus pallidus. 2. Cognitive aspects of movement and phasic neuronal activity. *Brain, 114*, 1685-1702.

Brown, J. W., Bullock, D., & Grossberg, S. (2004). How laminar frontal cortex and basal ganglia circuits interact to control planned and reactive saccades. *Neural Networks, 17*(4), 471-510.

Cheramy, A., Nieoullon, A., & Glowinski, J. (1979). In vivo evidence for a dendritic release of dopamine in the cat substantia nigra. *Applied Neurophysiology, 42*(1-2), 57-59.

Civier, O., Tasko, S. M., & Guenther, F. H. (2010). Overreliance on auditory feedback may lead to sound/syllable repetitions: Simulations of stuttering and fluency-inducing conditions with a neural model of speech production. *Journal of Fluency Disorders, 35*(3), 246-279.

Conde, H. (1992). Organization and physiology of the substantia nigra. *Experimental Brain Research, 88*(2), 233-248.

Cykowski, M. D., Fox, P. T., Ingham, R. J., Ingham, J. C., & Robin, D. A. (2010). A study of the reproducibility and etiology of diffusion anisotropy differences in developmental stuttering: A potential role for impaired myelination. *Neuroimage, 52*(4), 1495-1504.

Delong, M. R. (1971). Activity of pallidal neurons during movement. *Journal of Neurophysiology, 34*(3), 414-427.

Delong, M. R., Crutcher, M. D., & Georgopoulos, A. P. (1983). Relations between movement and single cell discharge in the substantia nigra of the behaving monkey. *Journal of Neuroscience, 3*(8), 1599-1606.

Filatova, E. V., Orlov, A. A., Tolkunov, B. F., & Afanas'ev, S. V. (2005). Neuron activity in the monkey striatum of identifies integration sequential actions into functional blocks. *Neuroscience and Behavioral Physiology, 35*(9), 943-949.

Frank, M. J. (2005). Dynamic dopamine modulation in the basal ganglia: A neurocomputational account of cognitive deficits in medicated and nonmedicated parkinsonism. *Journal of Cognitive Neuroscience, 17*, 51-72.

Gracco, V. L., & Abbs, J. H. (1987). Programming and execution processes of speech movement control: Potential neural correlates. In E. Keller, & M. Gopnik (Eds.), *Motor and sensory processes of language* (pp. 163-201). Hillsdale, NJ: L. Erlbaum Associates.

Grossberg, S. (1973). Contour enhancement, short term memory, and constancies in reverberating neural networks. *Studies in Applied Mathematics, 52*(3), 213–257.

Guenther, F. H., Ghosh, S. S., & Tourville, J. A. (2006). Neural modeling and imaging of the cortical interactions underlying syllable production. *Brain and Language, 96*(3), 280-301.

Hirvonen, M. M., Laakso, A., Nagren, K., Rinne, J. O., Pohjalainen, T., & Hietala, J. (2004). C957t polymorphism of the dopamine d2 receptor (drd2) gene affects striatal drd2 availability in vivo. *Molecular Psychiatry, 9*, 1060-1061.

Hirvonen, M. M., Laakso, A., Nagren, K., Rinne, J. O., Pohjalainen, T., & Hietala, J. (2005). C957t polymorphism of the dopamine d2 receptor (drd2) gene affects striatal drd2 availability in vivo. *Molecular Psychiatry, 10*, 889 (corrigendum).

Hirvonen, M. M., Laakso, A., Nagren, K., Rinne, J. O., Pohjalainen, T., & Hietala, J. (2009). C957t polymorphism of dopamine d2 receptor gene affects striatal drd2 in vivo availability by changing the receptor affinity. *Synapse, 63*(10), 907-912.

Kang, C., Domingues, B. S., Sainz, E., Domingues, C. E., Drayna, D., & Moretti-Ferreira, D. (2011). Evaluation of the association between polymorphisms at the drd2 locus and stuttering. *Journal of Human Genetics, 56*(6), 472-473.

Koos, T., & Tepper, J. M. (1999). Inhibitory control of neostriatal projection neurons by gabaergic interneurons. *Nature Neuroscience, 2*(5), 467-472.

Lan, J., Song, M., Pan, C., Zhuang, G., Wang, Y., Ma, W., et al. (2009). Association between dopaminergic genes (slc6a3 and drd2) and stuttering among han chinese. *Journal of Human Genetics, 54*(8), 457-460.

Laruelle, M. (2000). Imaging synaptic neurotransmission with in vivo binding competition techniques: A critical review. *Journal of Cerebral Blood Flow and Metabolism, 20*(3), 423-451.

Max, L. (2004). Stuttering and internal models for sensorimotor control: A theoretical perspective to generate testable hypotheses. In B. Maassen, R. D. Kent, H. F. M. Peters, P. H. H. M. Van Lieshout, & W. Hulstijn (Eds.), *Speech motor control in normal and disordered speech* (pp. 357-387). Oxford, UK: Oxford University Press.

Max, L., Guenther, F. H., Gracco, V. L., Ghosh, S. S., & Wallace, M. E. (2004). Unstable or insufficiently activated internal models and feedback-biased motor control as sources of dysfluency: A theoretical model of stuttering. *Contemporary Issues in Communication Science and Disorders, 31*, 105-122.

Mink, J. W., & Thach, W. T. (1993). Basal ganglia intrinsic circuits and their role in behavior. *Current Opinion in Neurobiology, 3*(6), 950-957.

Mushiake, H., & Strick, P. L. (1995). Pallidal neuron activity during sequential arm movements. *Journal of Neurophysiology, 74*(6), 2754-2758.

Nevet, A., Morris, G., Saban, G., Fainstein, N., & Bergman, H. (2004). Discharge rate of substantia nigra pars reticulata neurons is reduced in non-parkinsonian monkeys with apomorphine-induced orofacial dyskinesia. *Journal of Neurophysiology, 92*(4), 1973-1981.

Rastatter, M. P., & Harr, R. (1988). Measurements of plasma levels of adrenergic neurotransmitters and primary amino acids in five stuttering subjects: A preliminary report (biochemical aspects of stuttering). *Journal of Fluency Disorders, 13*, 127-139.

Smith, A. (1999). Stuttering: A unified approach to a multifactorial, dynamic disorder. In N. B. Ratner, & E. C. Healey (Eds.), *Stuttering research and practice: Bridging the gap* (pp. 27-43). Mahwah, NJ: Erlbaum.

Ueda, Y., & Kimura, M. (2003). Encoding of direction and combination of movements by primate putamen neurons. *European Journal of Neuroscience, 18*(4), 980-994.
Van Riper, C. (1982). *The nature of stuttering*. Englewood Cliffs, NJ: Prentice-Hall.
Wichmann, T., & Delong, M. R. (1996). Functional and pathophysiological models of the basal ganglia. *Current Opinion in Neurobiology, 6*(6), 751-758.
Wingate, M. E. (1966). Prosody in stuttering adaptation. *Journal of Speech and Hearing Research, 9*, 550–556.
Wingate, M. E. (1972). Deferring the adaptation effect. *Journal of Speech and Hearing Research, 15*(3), 547-550.
Wu, J. C., Maguire, G., Riley, G., Lee, A., Keator, D., Tang, C., et al. (1997). Increased dopamine activity associated with stuttering. *Neuroreport, 8*(3), 767-770.
Zikopoulos, B., & Barbas, H. (2007). Parallel driving and modulatory pathways link the prefrontal cortex and thalamus. *PLoS ONE, 2*(9), e848.
Zimmermann, G. (1980). Stuttering: A disorder of movement. *Journal of Speech and Hearing Research, 23*(1), 122-136.

APPENDIX A. SPECIFICATION OF THE ORIGINAL GODIVA MODEL

The extended GODIVA model uses the following equations of the original GODIVA model as-is (adapted from Bohland et al., 2010):

The activities of the IFS phonological content plan cells are given by:

(1) $\dot{p}_{ij} = -A_p p_{ij} + \left(B_p - p_{ij}\right)\left(\alpha u_{ij}^{p} + \left[p_{ij} - \theta_p\right]^+\right) - p_{ij}\left(\sum_{k \neq i} W_{ik} p_{kj} + 10y\left(\left[q_{ij} - \theta_q\right]^+\right)\right) + N\left(0, \sigma_p\right)$

The activities of the IFS phonological content choice cells are given by:

(2) $\dot{q}_{ij} = -A_q q_{ij} + \left(B_q - q_{ij}\right)\left(d_j\left[p_{ij} - \theta_p\right]^+ + y\left(q_{ij}\right)\right) - q_{ij}\left(\sum_{kj, k \neq i} W_{ik} y\left(q_{kj}\right) + \Gamma_{ij}\right)$

The response suppression signal from vPMC choice layer to IFS choice layer is given by:

(3) $\Gamma_{ij}\left(t\right) = 10 Z_k^{ij} s_k\left(t\right)$

The activities of the pre-SMA frame plan cells are given by:

(4) $\dot{f}_i = -A_f f_i + \left(B_f - f_i\right)\left(\alpha u_i^{f} + \left[f_i - \theta_f\right]^+\right) - f_i\left(\sum_{k \neq i} f_k + 10y\left(\left[g_i - \theta_g\right]^+\right)\right)$

The activities of the pre-SMA frame choice cells are given by:

(5) $\dot{g}_i = -A_g g_i + \left(B_g - g_i\right)\left(\Omega\left[f_i - \theta_f\right]^+ + y\left(g_i\right)\right) - g_i\left(\sum_{k \neq i} y\left(g_k\right)\right)$

The activities of the pre-SMA positional chain cells are given by:

(6) $h_j^k\left(t\right) = \begin{cases} 1 & \text{if } \left(\mathrm{t}_0 + \left(j-1\right)\tau\right) \leq t \leq \left(t_0 + j\tau\right) \\ 0 & \text{otherwise} \end{cases}$

The activities of the planning loop striatal projection neurons (direct pathway) are given by:

(7) $\dot{b}_j = -A_b b_j + \left(B_b - b_j\right)\left(h_j \wedge \left[\sum_k p_{kj} - \delta\right]^+\right) - b_j\left(\sum_{k \neq j} y\left(\underline{b}_k\right)\right)$

The activities of the planning loop striatal interneurons are given by:

(8) $\dot{\underline{b}}_j = -A_{\underline{b}} \underline{b}_j + \left(B_{\underline{b}} - \underline{b}_j\right)\left(h_j \wedge \left[\sum_k p_{kj} - \delta\right]^+\right) - \underline{b}\left(\sum_{k \neq j} y\left(\underline{b}_k\right)\right)$

The activities of the planning loop GPi cells are given by:

(9) $\dot{c}_j = -A_c c_j + \beta_c\left(B_c - c_j\right) - c_j\left(b_j\right)$

The activities of the planning loop anterior thalamic cells are given by:

(10) $\dot{d}_j = -A_d d_j + \beta_d \left(B_d - d_j \right) - d_j \left(c_j \right)$

The synapse from IFS choice cells to SSM plan cells that code multi-phoneme targets are given by:

(11) $Z_k^{ij} = \begin{cases} \frac{1}{N_k} & \text{if } r_k \text{ includes phoneme } i \text{ at position } j \\ 0 & \text{otherwise} \end{cases}$

APPENDIX B. VARIABLES OF THE ORIGINAL GODIVA MODEL

The main symbols used to refer to cell populations in the specification of the original GODIVA model are given in the following table (reproduced from Bohland et al., 2010):

Cell Group	Symbol
External Input to IFS	u^p
External Input to preSMA	u^f
IFS Phonological Content Plan Cells	p
IFS Phonological Content Choice Cells	q
Pre-SMA Frame Plan Cells	f
Pre-SMA Frame Choice Cells	g
Pre-SMA Positional Chain Cells	h
Planning Loop Striatal Projection Cells	b
Planning Loop Striatal Interneurons	$\underline{b}$
Planning Loop GPi Cells	c
Planning Loop Anterior Thalamic Cells	d

APPENDIX C. VARIABLES OF THE EXTENDED GODIVA MODEL

The symbols used to refer to cell populations in the specification of the BG-vPMC loop of the extended GODIVA model are given in the following table:

Cell Group	Symbol
SSM Plan Cells (vPMC planning layer)	r
SSM Choice Cells (vPMC choice layer)	s
BG-vPMC Loop Direct Pathway Striatal Projection Neurons (Putman D1R cells)	b^{D1}
BG-vPMC Loop Indirect Pathway Striatal Projection Neurons (Putman D2R cells)	b^{D2}
BG-vPMC Loop GABAergic Striatal Interneurons (Putamen IN cells)	b^{IN}
BG-vPMC Loop GPi Cells	c^{GPi}
BG-vPMC Loop GPe Cells	c^{GPe}
BG-vPMC Loop Thalamic Cells	d^{Thal}