# Expanding the Go/NoGo depiction of the action of Basal Ganglia Pathways

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***Abstract*—We present a neuronal network model of Basal Ganglia that departs from the classical Go/NoGo picture of the function of its key pathways – the direct and indirect pathways (DP & IP). The model is instantiated in a simple action selection task. Striatal dopamine is assumed to switch between DP and IP activation. Simulations reveal that between the Go and NoGo regimes, exhibited at extreme values of dopamine, the system displays, at intermediate values of dopamine, a new Explore regime, which enables it to explore the space of action alternatives. The exploratory dynamics originates from the chaotic dynamics of pallido-subthalamic loop. Following the tradition of applying reinforcement learning (RL) concepts to BG function, we associate this new regime with the Explorer, a key component in Actor-Critic framework.**

## INTRODUCTION

Recent years have seen a heightened activity in the area of computational modeling of basal ganglia (BG). The BG constitute 6 or 7 nuclei, which are distributed along two pathways – the direct pathway (DP) and the indirect pathway (IP). BG circuitry receives inputs from various cortical regions and projects back to the cortex via thalamus. The DP consists of the striatum, the input port to BG, which projects to Globus Pallidus interna (GPi), whereas in the IP, the striatum is connected to GPi via a longer route consisting of Globus Pallidus externa (GPe), and Subthalamic Nucleus (STN) [1]. Classically, activation of the DP is thought to facilitate movement, as though a gate is lifted and movement is released; therefore the DP is known as the Go pathway. Contrarily, activation of IP inhibits movement; the IP is dubbed the NoGo pathway. This switching between Go to NoGo is thought to be controlled by the dopamine projections to striatum arising from midbrain centers like the Substantia Nigra pars compacta [2]. The Go/NoGo dichotomy of the two BG pathways is also adopted in several modeling studies of BG function.

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There have been minor variations of Go/NoGo account of BG function. For example, the observations of [3] in this matter stem from the known anatomical connectivity patterns among the BG nuclei. The Caudate to Substantia Nigra pars reticulata (SNr) projection leads to a selective inhibition of SNr neurons. However, since the IP projections (GPe-SNr and/or STN-SNr) are more divergent, they exert opposing but spatially more distributed influence on SNr neurons. Therefore, s*imultaneous* action of the two pathways creates a greater focusing in the SNr activity. Similarly, it has been suggested [3] that when IP alone is activated, it produces a general inhibition of movement, like the kind, for example, necessary just before a specific movement is initiated.

Questions regarding validity of the simple Go/NoGo picture may be raised when we start applying concepts of Reinforcement Learning (RL) to BG function. The early experiments of Schultz [4] which suggested that mesencephalic dopamine signals are comparable to the temporal difference error of RL, have inspired an enormous body of modeling and experimental literature that developed an Actor-Critic based view of BG [5]-[8]. Interestingly, though Actor and Critic components have been associated with BG components [7][9][10], no association was found to the Explorer, an RL component that provides the perturbations needed to explore the space of actions. We had earlier hypothesized that the STN-GPe system of BG is the subcortical substrate for exploration and worked out some of consequences in normal and pathological conditions like Parkinson's disease[11]. Evidence that is consistent with such an idea was reviewed in [12]. In [11] we proposed that the Go/NoGo dichotomy needs to be expanded to a three-regime formula: Go/Explore/NoGo, where the dopamine signal to various BG targets switches the BG circuit among the three regimes of Go, Explore and NoGo. In [13] we incorporated this idea in a lumped model of BG, and used it to explain features of Parkinsonian reaching movements. We now present a network model of the BG circuit, which incorporates in simplified terms several known features of BG dynamics: 1) dopamine action on neuron dynamics of striatum, GPe and STN [14]-[17]; 2) the STN-GPe loop which produces oscillations on strong coupling [18]. The network is applied to a simple action selection task. When dopamine is high, the network consistently selects ("Go") the more salient action (of the two actions); when dopamine is low, it does not select the more salient action ("NoGo"); when dopamine is at intermediate level, the network

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randomly selects one of the actions ("Explore"). Chaotic dynamics of the STN-GPe is the source of the randomness seen in the Explore regime. The outline of the paper is as follows. In the following section the modeling components are described. Section III describes the numerical results – the three regimes and the effect of the chaotic dynamics of STN-GPe. The work is discussed in Section IV.

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## THE MODEL

Fig. 1 shows the architecture of the system being modeled.

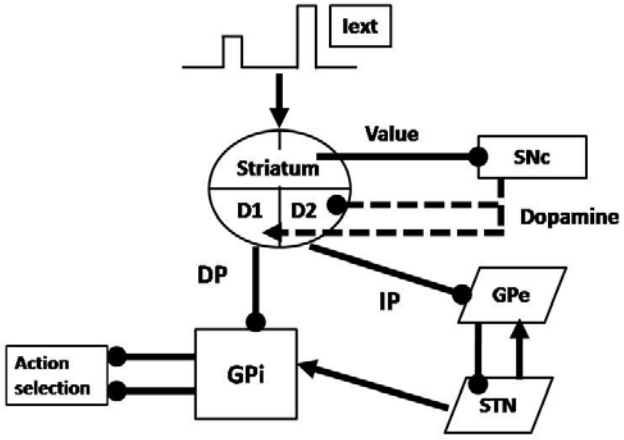


Fig. 1: The architecture of our model, showing excitatory connections (→), inhibitory connections (⊣) and DAergic modulatory connections (---) among the different BG nuclei. The spatially extended input (Iext) with two pulses of different amplitudes (assumed to be from cortex) is presented to the striatum where value computation occurs, resulting in release of different amounts of DA from SNc (not modeled). DP or IP is chosen depending on DA value, resulting in selection of one of the two inputs.

A spatially extended input, $I_{ext}$, consisting of two "pulses" of different amplitudes is presented to the striatum as shown in fig (1). The striatum consists of two 1D layers of neurons. The first layer represents D1-expressing neurons that project to GPi over the DP. The second layer represents D2-expressing neurons that project to GPe. The two layers (D1 and D2) have otherwise identical activation dynamics, differing only in the effect of dopamine signal:

$$u_i^{D1} = -u_i^{D1} + V_i^{D1} + I_i^{ext} \quad (1)$$

$$V_i^{D1} = \tanh(\lambda^{D1}.*u_i^{D1}) \quad (2)$$

$$u_i^{D2} = -u_i^{D2} + V_i^{D2} + I_i^{ext} \quad (3)$$

$$V_i^{D2} = \tanh(\lambda^{D2}.*u_i^{D2}) \quad (4)$$

where $u_i^{D1}$ represent the internal variables of D1 neurons of striatum, and $V_i^{D1}$ are the outputs of those neurons; $u_i^{D2}$ represent the internal variables of D2 neurons of striatum, and $V_i^{D2}$ are the outputs of those neurons; $\lambda_i^{D1}$ and $\lambda_i^{D2}$ represent the slopes of the sigmoid nonlinearities of D1 and D2 neurons respectively. Increased striatal dopamine levels are thought to activate D1 neurons while reducing the firing rates of D2 neurons. Therefore, we model $\lambda_i^{D1}$ and $\lambda_i^{D2}$ as functions of dopamine (δ) as follows:

$$\lambda^{D1} = 5*(\frac{1}{1+e^{-6*(\delta-\theta_{D1})}}) \quad (5)$$

$$\lambda^{D2} = 5*(\frac{1}{1+e^{-6*(\theta_{D2}-\delta)}}) \quad (6)$$

Unlike the striatal D1 and D2 layers, STN and GPe are 2D layers. Therefore, input to GPe from the striatal D2 layer is computed as:

$$I^{D2} = W_{Str}^{GPe}.*V^{D2} \quad (7)$$

where $V^{D2}$ is an nx1 array, $I^{D2}$ is an n x n array, and $W_{Str}^{GPe}$ is a 1xn array whose entries are all ones.

Where $W_X^Y$ refer to the weights from X to Y.

The connections between STN and GPe are assumed to be one-to-one with inclusion of lateral connections in GPe and STN. Each of these layers is implemented in a 2D grid fashion and the dynamics of the layers are given by:

$$\tau_g \frac{dx_{ij}^{GPe}}{dt} = -x_{ij}^{GPe} + \sum_{q=1}^{n}\sum_{p=1}^{n} W_{ij,pq}^{lat} U_{pq}^{GPe} + w_{sg} U_{ij}^{STN} + I_{ij}^{D2} \quad (8)$$

$$U_{ij}^{GPe} = \tanh(\lambda^{GPe}.*x_{ij}^{GPe}) \quad (9)$$

$$\tau_s \frac{dx_{ij}^{STN}}{dt} = -x_{ij}^{STN} + \sum_{q=1}^{n}\sum_{p=1}^{n} W_{ij,pq}^{glat} U_{pq}^{STN} - w_{gs} U_{pq}^{GPe} \quad (10)$$

$$U_{ij}^{STN} = \tanh(\lambda^{STN}.*x_{ij}^{STN}) \quad (11)$$

where (i, j) and (p, q) denote two neuron positions on the 2D grid, 'n' is the size of the 2D grid, $U_{ij}^{GPe}$ and $U_{ij}^{GPe}$ are the internal state and the output, respectively, of the $(i,j)^{th}$ neuron on the GPe grid, $x_{ij}^{STN}$ is the state of $(i,j)^{th}$ neuron on the STN grid, $w_{sg}$ and $w_{gs}$ are the strengths of the connection between STN-GPe and GPe to STN respectively. The translation-invariant lateral connections, $W_{ij,pq}^{slat}$ (>0) and $W_{ij,pq}^{glat}$ (<0), within STN and GPe respectively are:

$$W_{ij,pq}^{slat} = \sigma_{\max}^{s} \exp(-r_{ij,pq}^2 / \sigma_{lat}^2) \quad \text{for } r < R$$
$$= 1, \quad \text{for } r=0$$
$$= 0, \quad \text{otherwise.} \qquad (12)$$

$$W_{ij,pq}^{glat} = \sigma_{\max}^{g} \exp(-r_{ij,pq}^2 / \sigma_{lat}^2) \quad \text{for } r < R$$
$$= -1, \quad \text{for } r=0$$
$$= 0, \quad \text{otherwise.} \qquad (13)$$

Note that STN and GPe are modeled as an excitatory-inhibitory pair of neural layers. Such systems exhibit oscillations under specific parameter settings. Similar models of STN-GPe have been proposed by others [19][20]. Dopaminergic projections from SNc modulate the firing rates of the STN and GPe neurons [21], which predominantly express D2 receptors [2]. Just as above, the dependence of activations of STN and GPe neurons on dopamine is modeled by making $\lambda^{STN}$ and $\lambda^{GPe}$ dependent on δ as,

$$\lambda^{STN} = 4*(\chi(6*(\theta_s - \delta)) + 1 \qquad (14)$$

$$\lambda^{GPe} = 4*(\chi(6*(\theta_g - \delta)) + 1 \qquad (15)$$

Where $\chi(x) = 1$ if $x>0$
$= 0$ if $x<0$

GPi performs action selection on the input "pulses." This selection is coded in the population activity of GPe neurons: the "left" pulse is considered to be selected if the sum of outputs of the "left" neurons (i=1 to 10) is higher than that of the "right" neurons (i=11 to 20). GPi is modeled as a 1D Continuous Attractor Neural Network (CANN) [22] which suitably embodies the competition necessary for action selection. Input to the GPi is a sum of outputs of D1 neurons of striatum (DP) and outputs of STN neurons (output of IP):

$$I^{GPi} = V^{D1} + U^{STN} W_{STN}^{GPi} \qquad (16)$$

where $W_{STN}^{GPi}$ is a 1 X n array in which the value of every entry equals 1/n.

Dynamics of GPi are given as:

$$\tau_u \frac{du_i^{GPi}}{dt} = -u_i^{GPi} + \sum_p w_{i,p}^{GPi} s_p^{GPi} \kappa_x + I_i^{GPi} \qquad (17)$$

where, $u_i^{GPi}$ is an internal variable of neuron at location i in GPi. The lateral connections consist of short-range excitation and long-range inhibition given as:

$$w_{i,p}^{GPi} = Ae^{-\frac{d^2}{2\sigma_w^2}} - C; \quad d = |i - p| \qquad (18)$$

$$s_i^{GPi} = \frac{(u_i^{GPi})^2}{1 + \frac{1}{2}\sum_p (u_p^{GPi})^2 \kappa_x} \qquad (19)$$

Action selection based on GPi neural activity is performed by defining $y_1$ and $y_2$ as:

$$y_1 = \sum_{i=1}^{n/2} s_i^{GPi}; \quad y_2 = \sum_{i=n/2+1}^{n} s_i^{GPi} \qquad (20)$$

Action 1 is selected if $y_1 > y_2$, and action 2 selected otherwise.

## RESULTS

***Dynamics of the STN-GPe on variation in connection strengths:*** We examine the effect of variation in connection strengths on the dynamics of STN-GPe network. We assume that the intra-nuclear lateral connection strengths in STN and GPe are equal in magnitude ($W_{ij,pq}^{slat} = W_{ij,pq}^{glat}$) and represented by σ. Similarly, the inter-nuclear connection between STN and GPe are also considered to be equal in magnitude ($w_{sg} = w_{gs}$) and represented by $w$. Fig. 2 shows the regions of ordered and chaotic behavior (determined based on the highest Lyapunov exponent,$\lambda_{ly}$) for different values of $w$ and σ. The extended lines indicate the borders between the regions where the $\lambda_{ly}$ changes sign from negative (ordered) to positive (chaotic) dynamics. The area around this line corresponds to the 'edge of chaos' which has been shown to have maximal computational capacity [23]. The dynamics become chaotic for high values of $w$ and low values of σ (fig. 2). We do not consider the second line occurring at high values of σ since it is not biologically realistic.

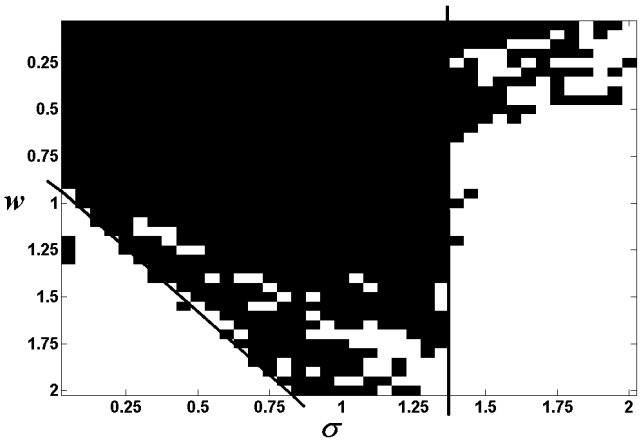


Fig. 2: x axis- σ, y axis- *w;* both varied between 0.05 and 2. Ordered regions correspond to positive $\lambda_{ly}$ (white); chaotic regions correspond to negative $\lambda_{ly}$ (black). Borders between the two regions are marked by the two lines extending out of the figure. We set the threshold for, $\lambda_{ly}$ to be 0.1. Plot shows average result from 20 runs for each (σ, w) pair.

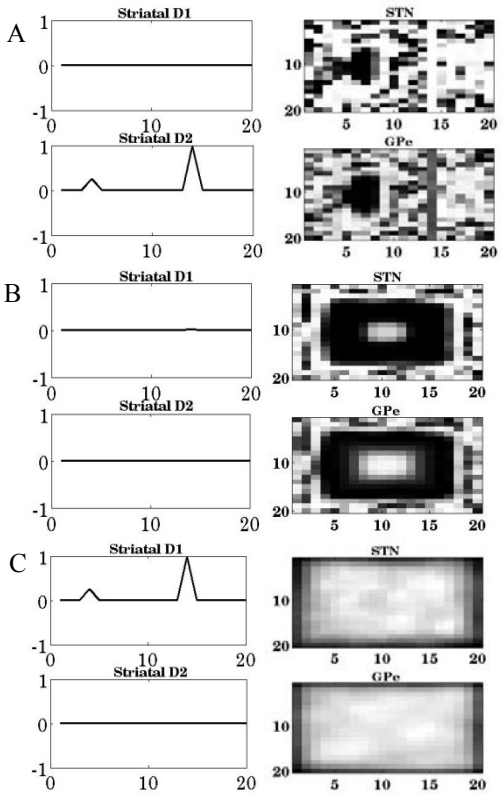


Fig. 3: The dynamics of the different nuclei of the BG for increasing levels of DA. In each panel, the left figures represent the output of the striatal D1 (above) and D2 neurons (below) while the right panels represent the activity of STN(above) and GPe(below). (A) for low values of DA ($\delta$ =-2). (B) for intermediate values of DA ($\delta$=-0.2) and (C) for high values of DA ($\delta$=1).

***Effect of Dopamine(DA) on action selection:*** Since one the most important functions of the BG is action selection and since DA levels are known to play a critical role in this process, we studied the effect of DA levels on selection between two input pulses of different amplitudes (fig. 1). Fig. 3 shows the response of the different modules of the BG to two pulses of different amplitudes for different DA values, with the STN-GPe network showing chaotic dynamics. It can be seen from the figure that for low values of DA, the STN-GPe is maximally active and results in suppression of the higher amplitude pulse and thus selection of the weaker pulse, whereas for high DA values, the STN-GPe is not active and the stronger pulse gets selected via the Direct pathway. For intermediate values of DA, since STN-GPe and the direct pathway are nearly equally balanced, the selection between the two pulses becomes probabilistic because of the chaotic nature of the STN-GPe dynamics.

***Effect of STN-GPe dynamics and DA on action selection:*** The intrinsic dynamics of the STN-GPe network is thought to play a crucial role in the function of these nuclei. Several studies have shown that the normal dynamics of STN is chaotic and a loss of the complexity in dynamics due to increased correlations in firing activity in STN is observed under dopamine-deficient conditions.[24][25]. Our model of the STN-GPe network exhibits two dynamic regimes (ordered/chaotic) of intrinsic dynamics on varying the different connection strengths. We therefore investigate the role of these regimes and DA levels on action selection. Fig. 4 shows the probability of selection of the stronger input as a function of the DA level in the ordered, intermediate and chaotic modes of function of the STN-GPe network. In the case of the ordered regime, the stronger input is always selected. On the border between the ordered and chaotic regimes, there is a region of low DA where the probability of selection of stronger input is 0.5, indicating that in this region other alternatives can be explored. In the case of the chaotic region, the probability of selection of stronger input varies from very low values (0.2) to 1 as a function of DA, showing that for very low values of DA, the stronger input is suppressed and for high values of DA, the stronger one is selected whereas for intermediate values of DA, either choice can be selected. This region of intermediate probability of selection of stronger input which we see for intermediate DA levels is what we define as the 'exploratory' regime of DA action. We can see that on the border between the ordered/chaotic regimes of STN-GPe dynamics, the exploratory regime is largest whereas it is smaller deep in the chaotic regime and nearly non-existent in the ordered regime.

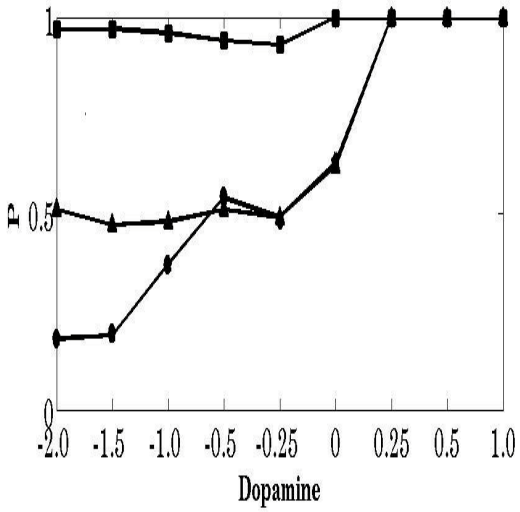


Fig. 4: The probability of selection of stronger input (Y-axis) as a function of the DA levels (X-axis) in the ordered regime (■), the border between ordered/chaotic (▲) and the chaotic regime of the STN-GPe dynamics (●).

***Effect of thresholds on the exploratory region of STN-GPe function:*** The existence of the exploratory region is a function of the threshold parameters ($\theta$) that define the dependence of neural firing rates on DA in striatum (D1 and D2) (5) (6), $\theta_{D1}$ and $\theta_{D2}$), STN ((14), $\theta_s$) and GPe ((15), $\theta_g$). These thresholds determine how dopamine controls the relative activations of DP and IP. Fig. 5 shows the effect of increasing this threshold for D2 striatum ($\theta_{D2}$) on the exploratory region. When the threshold for selection of the IP, $\theta_{D2,}$ approaches the selection threshold for DP, $\theta_{D1}$ ,then the exploration is reduced and probability of selection of

stronger input approaches either 0 (for low δ) or 1(for high δ).

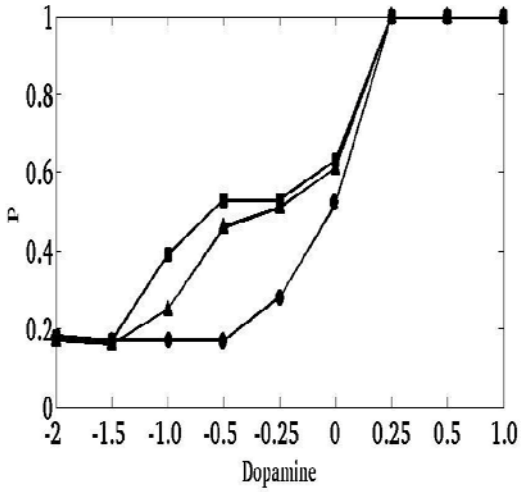


Fig. 5: The probability of selection of stronger input (Y-axis) as a function of the DA levels (X-axis) for $\theta_{D2,}$= -0.9(■),$\theta_{D2,}$ =-0.5 (▲) and $\theta_{D2,}$ = 0.1 (●).

## DISCUSSION

We present a model of basal ganglia that seeks to expand the classical Go/NoGo depiction of the action of the two BG pathways – DP and IP. The classical description maintains that activation of DP facilitates movement while activation of IP inhibits movement. Experimental evidence for such a picture does exist. Theoretical arguments in support of such a picture are often based on the number (odd or even) of excitatory/inhibitory links in the chains of the two BG pathways. Dopamine released from SNc to BG targets is thought to result in a transition from Go (high DA) to NoGo (low DA) regime. We argue that the classical Go/NoGo dichotomy is too simplistic and posit between the Go and NoGo regimes an intermediate regime (for moderate dopamine levels) dubbed as "Explore" regime. We propose that the IP is the substrate to this new regime, in addition to the NoGo regime.

We proposed this dopamine-dependent three regime model of BG function, recently in a *lumped* model of BG involved in driving reaching movements. However, in [13], the three regimes were proposed as a hypothesis whose justification is found solely in the fact that the model is able to explain features of Parkinsonian reaching movements. In the present work we show that the three regimes emerge naturally in a *network* model of BG, simply by combining familiar models of BG components ( striatum model similar to [26]; STN-GPe model similar to [19] in a novel way. Earlier models have shown synchronous oscillations and complex spiking in STN-GPe system and have linked oscillations with Parkinsonian tremor. Models of DBS proposed that the DBS acts by desynchronizing STN-GPe network from its pathological oscillatory mode, thereby leading to decrease in tremor [27]. However, these models do not discuss the reason why desynchronized, complex activity is necessary for normal BG function and therefore why the emergence of synchrony should lead to pathology. Describing the BG function within the framework of RL, we show why complex activity is necessary for the normal function of the BG in that it provides the activity for the Explore regime between Go and NoGo.

Our model also uses the concept of activity at the 'edge of chaos' which has been proposed as the optimal zone for functioning of some biological systems [28][29]. Systems living on the "edge of chaos" are thought to have the flexibility to rapidly adapt to changing environmental conditions. This idea originated from studies of cellular automata (CA), which found that CAs operating near the edge of chaos are capable of universal computation [23]. The possibility of brain operating near edge of chaos has been discussed for some years, with mounting evidence [29]. In our model, the highest exploration was seen near the edge of chaos, when the model selects the two possible outputs with equal probability (fig. 4). Deep in the ordered regime, the system exhibits a kind of "perseverative behavior" mostly choosing action #1; deep in the chaotic regime, the system is biased towards action #2. Such perseverative activity has been observed in STN damaged rats which show decreased exploration of new options even when the old ones are no longer rewarding [30].

Dopamine controls the activity of the BG at several levels, of which we have not included some in our simplified model. For instance, we have not considered the connection strength between STN and GPe to be explicit functions of dopamine [31]. Further work incorporating these effects needs to be carried out to extend and validate the model.

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