Simulating Parkinson's disease patient deficits using a COVIS-based computational model

Sebastien Helie, Erick J. Paul, and F. Gregory Ashby

***Abstract*—COVIS is a neurobiologically motivated model of perceptual category learning. It includes two competing systems: the *hypothesis-testing system* mediates learning and performance in tasks requiring explicit reasoning; the *procedural system* mediates learning and performance in tasks that are achieved procedurally through trial and error learning when no explicit rule/strategy exists. Here we describe a computational implementation of COVIS used to model the differential effects of dopamine depletion on performance in a perceptual category-learning task and the simplified Wisconsin Card Sorting Test (WCST).**

I. Introduction

Parkinson's Disease (PD) is caused by the accelerated death of dopamine (DA) producing neurons in the ventral extent of the Substantia Nigra pars compacta (SNpc) as well as the Ventral Tegmental Area (VTA). Symptoms appear after a loss of 60-70% of cells [1]. Non-demented PD patient deficits often resemble those observed in frontal damage patients. Patient groups have shown deficits in a variety of cognitive tasks related to memory, learning (e.g., rule generation), visuospatial skills, and attention (e.g., ignoring irrelevant and maintaining relevant information) [2]. While there have been several studies documenting cognitive deficits of PD patients (for a review, see [3]), very few computational models have been proposed to model the disease. In this article, we use the COVIS model of categorization [4] to simulate DA depletion and show that the model suffers from cognitive symptoms similar to those of human participants affected by PD.

The remainder of this article is organized as follows. First, we introduce COVIS and present a detailed computational implementation of the model. Second, we present how the DA deficits found in PD patients can be modeled within the COVIS framework. Third, we simulate two tasks where the performance of normal participants has been compared with the performance of PD patients: perceptual categorization and the simplified Wisconsin Card Sorting Test (WCST). Fourth, we conclude with a general discussion of the implications of computational PD modeling for future research.

Manuscript received May 5, 2011. This work was supported in part by the U.S. Army Research Office through the Institute for Collaborative Biotechnologies under grant W911NF-07-1-0072 and by the Intelligence Advanced Research Projects Activity (IARPA) via Department of the Interior (DOI) contract number D10PC20022 (through Lockheed Martin). The U.S. government is authorized to reproduce and distribute reprints for Governmental purposes notwithstanding any copyright annotation thereon. The views and conclusions contained herein are those of the authors and should not be interpreted as necessarily representing the official policies or endorsements, either expressed or implied, of IARPA, DOI, or the U.S. Government.

S. Helie is with the Department of Psychological & Brain Sciences, University of California, Santa Barbara, CA 93106-9660 USA (phone: 805-284-9474; fax: 805-893-4303; e-mail: helie@psych.ucsb.edu).

E. J. Paul is with the Department of Psychological & Brain Sciences, University of California, Santa Barbara, CA 93106-9660 USA (e-mail: paul@psych.ucsb.edu).

F. G. Ashby is with the Department of Psychological & Brain Sciences, University of California, Santa Barbara, CA 93106-9660 USA (e-mail: ashby@psych.ucsb.edu).

II. COVIS

A. Theory

COVIS [4] postulates two systems that compete throughout learning – an explicit, hypothesis-testing system that uses logical reasoning and depends on working memory and executive attention, and an implicit system that uses procedural learning.

The explicit, hypothesis-testing system of COVIS is thought to mediate *rule-based* category learning. Rule-based category-learning tasks are those in which the category structures can be learned via some explicit reasoning process. Frequently, the rule that maximizes accuracy (i.e., the optimal rule) is easy to describe verbally. In the most common applications, only one stimulus dimension is relevant, and the observer's task is to discover this relevant dimension and then to map the different dimensional values to the relevant categories. Even so, rule-based tasks can require attention to multiple stimulus dimensions. For example, any task where the optimal strategy is to apply a logical conjunction or disjunction is rule-based. The key requirement is that the optimal strategy can be discovered by logical reasoning and is easy for humans to describe verbally.

The implicit procedural-learning system of COVIS is hypothesized to mediate *information-integration* category learning. Information-integration tasks are those in which accuracy is maximized only if information from two or more stimulus components (or dimensions) is integrated at some pre-decisional stage. Perceptual integration could take many forms – from treating the stimulus as a Gestalt to computing a weighted linear combination of the dimensional values. Typically, the optimal strategy in information-integration tasks is difficult or impossible to describe verbally. Rule-based strategies can be applied in information-integration tasks, but they generally lead to sub-optimal levels of accuracy because rule-based strategies make separate decisions about each stimulus component, rather than integrating this information.

B. Implementation

The computational version of COVIS includes three separate components – namely a model of the hypothesis-testing system, a model of the procedural-learning system, and an algorithm that monitors the output of these two systems and selects a response on each trial. We describe each of these components in turn.

1) The Hypothesis-testing System: The hypothesis-testing system in COVIS selects and tests explicit rules that determine category membership. The simplest rule is one-dimensional. More complex rules are constructed from one-dimensional rules via Boolean algebra (e.g., to produce logical conjunctions, disjunctions, etc.). The neural structures that have been implicated in this process include the prefrontal cortex, anterior cingulate, head of the caudate nucleus, and hippocampus [4]-[6]. The computational implementation of the COVIS hypothesis-testing system is a hybrid neural network that includes both symbolic and connectionist components. The model's hybrid character arises from its combination of explicit rule selection and switching and its incremental salience-learning component.

To begin, denote the set of all possible explicit rules by $\boldsymbol{R} = \{R_1, R_2, \ldots, R_m\}$. In most applications, the set $\boldsymbol{R}$ will include all possible one-dimensional rules, and perhaps a variety of plausible conjunction and/or disjunction rules. On each trial, the model selects one of these rules for application by following an algorithm that is described below.

Suppose the stimuli to be categorized vary across trials on r stimulus dimensions. Denote the coordinates of the stimulus on these r dimensions by $\underline{x} = (x_1, x_2, \ldots, x_r)$. On trials when the active rule is R_i, a response is selected by computing a discriminant value $h_E(\underline{x})$ and using the following decision rule:

Respond A on trial n if $h_E(\underline{x}) < \varepsilon$;
Respond B if $h_E(\underline{x}) > \varepsilon$ (1)

where ε is a normally distributed random variable with mean 0 and variance σ_E^2. The variance σ_E^2 increases with trial-by-trial variability in the subject's perception of the stimulus and memory of the decision criterion (i.e., perceptual and criterial noise). In the case where R_i is a one-dimensional rule in which the relevant dimension is i, the discriminant function is

$$h_E(\underline{x}) = x_i - C_i \quad (2)$$

where C_i is a constant that plays the role of a decision criterion. Note that this rule is equivalent to deciding whether the stimulus value on dimension i is greater or less than the criterion C_i. The decision bound is the set of all points for which $x_i - C_i = 0$. Note that $| h_E(\underline{x}) |$ increases with the distance between the stimulus and this bound.

Suppose rule R_i is used on trial n. Then the rule selection process proceeds as follows. If the response on trial n is correct, then rule R_i is used again on trial $n + 1$ with probability 1. If the response on trial n is incorrect, then the probability of selecting each rule in the set $\boldsymbol{R}$ for use on trial $n + 1$ is a function of that rule's current weight. The weight associated with each rule is determined by the subject's lifetime history with that rule, the reward history associated with that rule during the current categorization training session, the tendency of the subject to perseverate, and the tendency of the subject to select unusual or creative rules. These factors are all formalized as described next.

Let $Z_k(n)$ denote the salience of rule R_k on trial n. Therefore, $Z_k(0)$ is the initial salience of rule R_k. Rules that participants have abundant prior experience with have high initial salience, and rules that a participant has rarely used before have low initial salience. In typical applications of COVIS, the initial saliencies of all one-dimensional rules are set equal, whereas the initial saliencies of conjunctive and disjunctive rules are set much lower. The salience of a rule is adjusted after every trial on which it is used, in a manner that depends on whether or not the rule was successful. For example, if rule R_k is used on trial $n - 1$ and a correct response occurs, then

$$Z_k(n) = Z_k(n - 1) + \Delta_C \quad (3)$$

where Δ_C is some positive constant. If rule R_k is used on trial $n - 1$ and an error occurs, then

$$Z_k(n) = Z_k(n - 1) - \Delta_E \quad (4)$$

where Δ_E is also a positive constant. The numerical value of Δ_C should depend on the perceived gain associated with a correct response and Δ_E should depend on the perceived cost of an error.

The salience of each rule is then adjusted to produce a weight, Y, according to the following rules. For the rule R_i that was active on trial n,

$$Y_i(n) = Z_i(n) + \gamma \quad (5)$$

where the constant γ is a measure of the tendency of the participant to perseverate on the active rule, even though feedback indicates that this rule is incorrect. If γ is small, then switching will be easy, whereas switching is difficult if γ is large. COVIS assumes that switching of executive attention is mediated within the head of the caudate nucleus, and that the parameter γ is inversely related to basal ganglia DA levels.

Choose a rule at random from $\boldsymbol{R}$. Call this rule R_j. The weight for this rule is

$$Y_j(n) = Z_j(n) + \mathbf{X} \quad (6)$$

where $\mathbf{X}$ is a random variable that has a Poisson distribution with mean λ. Larger values of λ increase the probability that rule R_j will be selected for the next trial, so λ is called the selection parameter. COVIS assumes that selection is mediated by a cortical network that includes the anterior cingulate and the prefrontal cortex, and that λ increases with cortical DA levels. For any other rule R_k (i.e., $R_k \neq R_i$ or R_j),

$$Y_k(n) = Z_k(n) \quad (7)$$

Finally, rule R_k (for all k) is selected for use on trial $n + 1$ with probability

$$P_{n+1}(R_k) = \frac{Y_k^a(n)}{\sum_{s=1}^{m} Y_s^a(n)} \quad (8)$$

where a is a parameter that determines the decision stochasticity. When $a < 1$, the decision is noisy and the probability differences are diminished (making the decision probabilities uniform). When $a > 1$, the decision tends to become more deterministic. Hence, COVIS assumes that a increases with cortical DA [7]. This algorithm has a number of attractive properties. First, the more salient the rule, the higher the probability that it will be selected, even after an incorrect trial. Second, after the first trial, feedback is used to adjust the selection probabilities up or down, depending on the success of the rule type. Third, the model has separate selection and switching parameters, reflecting the COVIS assumption that these are separate operations. The random variable **X** models the selection operation. The greater the mean of **X** (i.e., λ) in (6), the greater the probability that the selected rule (R_j) will become active. In contrast, the parameter γ from (5) models switching, because when γ is large, it is unlikely that the system will switch to the selected rule R_j. It is important to note, however, that with both parameters (i.e., λ and γ), optimal performance occurs at intermediate numerical values. For example, note that if λ is too large, some extremely low salience rules will be selected, and if γ is too low then a single incorrect response could cause a participant to switch away from an otherwise successful rule.

Each one-dimensional rule has an associated decision criterion (e.g., the C_i in (2)) and each conjunction rule has two. These are not free parameters: they are learned using a conventional gradient-descent process with learning rate δ. Thus, the full COVIS hypothesis-testing system has 7 free parameters: σ_E^2 (noise variance), γ (perseveration), λ (selection), a (decision stochasticity), Δ_C (salience increment when correct), Δ_E (salience decrement when incorrect), and δ (gradient-descent learning rate).

2) The Procedural System: The current implementation of the procedural system is called the Striatal Pattern Classifier (SPC: [8]). The SPC learns to assign responses to regions of perceptual space. In such models, a decision bound could be defined as the set of all points that separate regions assigned to different responses, but it is important to note that in the SPC, the decision bound has no psychological meaning. As the name suggests, the SPC assumes the key site of learning is at corticostriatal synapses within the striatum.

The SPC architecture is shown in Fig. 1 for an application to a categorization task with two contrasting categories. This is a straightforward three-layer feedforward network with up to 10,000 units in the input layer and two units each in the hidden and output layers. The only modifiable synapses are between the input and hidden layers. The more biologically detailed version of this model proposed in [8] included lateral inhibition between striatal units and between cortical units. In the absence of such inhibition, the top motor output layer in Fig. 1 represents a conceptual placeholder for the striatum's projection to premotor areas. This layer is not included in the following computational description.

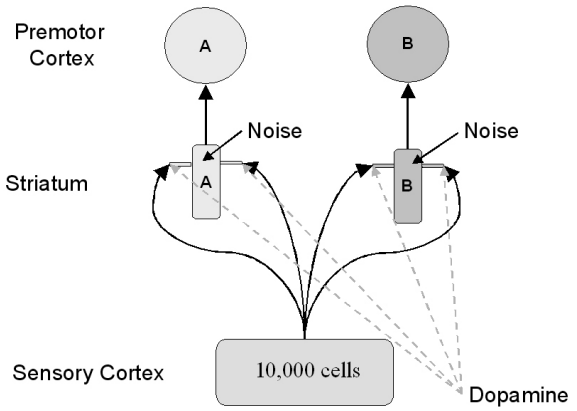

Fig. 1. A schematic illustrating the architecture of the COVIS procedural system.

The key structure in the model is the striatum (mostly the putamen), which is a major input region of the basal ganglia. In humans and other primates, all of extra-striate cortex projects directly to the striatum and these projections are characterized by massive convergence, with the dendritic field of each medium spiny cell innervated by the axons of approximately 380,000 cortical pyramidal cells [9]. COVIS assumes that, through a procedural-learning process, each striatal unit associates an abstract motor program with a large group of sensory cortical cells (i.e., all that project strongly to it).

The dendrites of striatal medium spiny cells are covered in protuberances called spines. These play a critical role in the model because glutamate projections from sensory cortex and DA projections from the SNpc converge (i.e., synapse) on the dendritic spines of the medium spiny cells. COVIS assumes that these synapses are a critical site of procedural learning.

a) Activation equations: Sensory cortex is modeled as an ordered array of up to 10,000 units, each tuned to a different stimulus. The model assumes that each unit responds maximally when its preferred stimulus is presented, and that its response decreases as a Gaussian function of the distance in stimulus space between the stimulus preferred by that unit and the presented stimulus. Specifically, when a stimulus is presented, the activation in sensory cortical unit K on trial n is given by

$$I_K(n) = e^{-\frac{d(K,\,\text{stimulus})^2}{\alpha}} \quad (9)$$

where α is a constant that scales the unit of measurement in stimulus space and *d(K, stimulus)* is the distance (in stimulus space) between the stimulus preferred by unit K and the

presented stimulus (smaller α produces a smaller unit of measurement). Equation (9), which is an example of a radial basis function, is a popular method for modeling the receptive fields of sensory units in models of many cognitive tasks.

COVIS assumes that the activation in striatal unit *J* (within the middle or hidden layer) on trial *n*, denoted $S_J(n)$, is determined by the weighted sum of activations in all sensory cortical cells that project to it:

$$S_J(n) = \sum_K w_{K,J}(n) I_K(n) + \varepsilon \tag{10}$$

where $w_{K,J}(n)$ is the strength of the synapse between cortical unit *K* and striatal cell *J* on trial *n*, $I_K(n)$ is the input from visual cortical unit *K* on trial *n*, and ε is normally distributed noise (with mean 0 and variance σ_P^2; in all the present simulations, $\sigma_P^2 = 0.0125$).

In a task with two alternative categories, A and B, the decision rule is:

Respond A on trial *n* if $S_A(n) > S_B(n)$;
Otherwise respond B. (11)

Hence, smaller σ_P^2 tend to produce more deterministic behaviors. The synaptic strengths $w_{K,J}(n)$ are adjusted up and down from trial-to-trial via reinforcement learning, which is described below. To run the model however, initial values must be selected for these weights. This is done by randomly sampling from some uniform distribution.

b) Learning equations: The three factors thought to be necessary to strengthen corticostriatal synapses are 1) strong pre-synaptic activation, 2) strong post-synaptic activation, and 3) DA levels above baseline (e.g., see [10]). According to this model, the synapse between a cell in sensory association cortex and a medium spiny cell in the striatum is strengthened if the cortical cell responds strongly to the presented stimulus, the striatal cell is also strongly activated (i.e., factors 1 and 2 are present) and the participant is rewarded for responding correctly (factor 3). On the other hand, the strength of the synapse will weaken if the participant responds incorrectly (factor 3 is missing), or if the synapse is driven by a cell in sensory cortex that does not produce much activation in the striatum (i.e., factor 2 is missing).

Let $w_{K,J}(n)$ denote the strength of the synapse on trial *n* between cortical unit *K* and striatal unit *J*. COVIS models reinforcement learning as follows:

$$\begin{aligned} w_{K,J}(n+1) &= w_{K,J}(n) \\ &+ \alpha_w I_K(n) [S_J(n) - \theta_{NMDA}]^+ [D(n) - D_{base}]^+ [1 - w_{K,J}(n)] \\ &- \beta_w I_K(n) [S_J(n) - \theta_{NMDA}]^+ [D_{base} - D(n)]^+ w_{K,J}(n) \\ &- \gamma_w I_K(n) \left\{ [\theta_{NMDA} - S_J(n)]^+ - \theta_{AMPA} \right\}^+ w_{K,J}(n). \end{aligned} \tag{12}$$

The function $[g(n)]^+ = g(n)$ if $g(n) > 0$, and otherwise $g(n) = 0$. The constant D_{base} is the baseline DA level, $D(n)$ is the amount of DA released following feedback on trial *n*, and α_w, β_w, γ_w, θ_{NMDA}, and θ_{AMPA} are all constants. The first three of these (i.e., α_w, β_w, and γ_w) operate like standard learning rates because they determine the magnitudes of increases and decreases in synaptic strength (in all the simulations herein, $\alpha_w = 0.4$, $\beta_w = 0.19$, and $\gamma_w = 0.02$). The constants θ_{NMDA} and θ_{AMPA} represent the activation thresholds for post-synaptic NMDA and AMPA (more precisely, non-NMDA) glutamate receptors, respectively. The numerical value of $\theta_{NMDA} > \theta_{AMPA}$ because NMDA receptors have a higher threshold for activation than AMPA receptors. This is critical because NMDA receptor activation is required to strengthen corticostriatal synapses [11]. Note that the values assigned to θ_{NMDA} and θ_{AMPA} are used to discriminate between the postsynaptic activation of the different striatal cells. As such, mid-level values should be selected (because values too high or too low will not allow for such discrimination).

The first line in (12) describes the conditions under which synapses are strengthened (i.e., striatal activation above the threshold for NMDA receptor activation and DA above baseline) and lines two and three describe conditions that cause the synapse to be weakened. The first possibility (line 2) is that post-synaptic activation is above the NMDA threshold but DA is below baseline (as on an error trial), and the second possibility is that striatal activation is between the AMPA and NMDA thresholds. Note that synaptic strength does not change if post-synaptic activation is below the AMPA threshold.

c) Dopamine model: The Equation (12) model of reinforcement learning requires that we specify the amount of DA released on every trial in response to the feedback signal (the $D(n)$ term). The key empirical results are [12]: 1) midbrain DA cells fire spontaneously (i.e., tonically), 2) DA release increases above baseline following unexpected reward, and the more unexpected the reward the greater the release, and 3) DA release decreases below baseline following unexpected absence of reward, and the more unexpected the absence, the greater the decrease. One common interpretation of these results is that over a wide range, DA firing is proportional to the reward prediction error (RPE):

RPE = Obtained Reward - Predicted Reward (13)

A simple model of DA release can be built by specifying how to compute Obtained Reward, Predicted Reward, and exactly how the amount of DA release is related to the RPE. Our solution to these three problems is as follows.

In applications that do not vary the valence of the rewards (e.g., as in designs where some correct responses are rewarded more than others), the obtained reward R_n on trial *n* is defined as +1 if correct or reward feedback is received, 0 in the absence of feedback, and -1 if error feedback is received.

We use a simplified version of the well-known Rescorla-Wagner model [13] to compute Predicted Reward. Consider a trial where the participant has just responded for the n^{th} time to some particular stimulus. Then COVIS

assumes that the reward the participant expects to receive equals

$$P_n = P_{n-1} + .025(R_{n-1} - P_{n-1}) \quad (14)$$

It is well known that when computed in this fashion, P_n converges exponentially to the expected reward value and then fluctuates around this value until reward contingencies change [13].

Bayer and Glimcher [14] reported activity in midbrain DA cells as a function of RPE. A simple model that nicely matches their results is

$$D(n) = \begin{cases} D_{\max} & \text{if } \mathrm{RPE} > 1 \\ D_{\mathrm{slope}} \times \mathrm{RPE} + D_{base} & \text{if } -.25 < \mathrm{RPE} \le 1 \\ 0 & \text{if } \mathrm{RPE} < -.25 \end{cases} \quad (15)$$

where D_{max}, D_{slope}, and D_{base} are constant. Note that the baseline DA level is D_{base} (i.e., when the RPE = 0) and that DA levels increase linearly with the RPE. However, note also the asymmetry between DA increases and decreases (which is evident in [14]) – that is, a negative RPE quickly causes DA levels to fall to zero, whereas there is a considerable range for DA levels to increase in response to positive RPEs. Specifically, higher values of D_{max} allow for a larger increase in DA following unexpected reward, higher values of D_{base} allow for a larger decrease of DA following the unexpected absence of reward, and higher values of D_{slope} increase the effect of RPE on DA release. Thus, increasing the value of any of these constants should improve learning in the procedural system (up to a point).

3) Resolving the Competition Between the Hypothesis-testing and Procedural Systems: Since on any trial the model can make only one response, the final task is to decide which of the two systems will control the observable response. In COVIS, this competition is resolved by combining two factors: the confidence each system has in the accuracy of its response, and how much each system can be trusted. In the case of the hypothesis-testing system, confidence equals the absolute value of the discriminant function $| h_E(n) |$. When $| h_E(n) | = 0$, the stimulus is exactly on the hypothesis-testing system's decision bound, so the model has no confidence in its ability to predict the correct response. When $| h_E(n) |$ is large, the stimulus is far from the bound and confidence is high. In the procedural system, confidence is defined as the absolute value of the difference between the activation values in the two striatal units:

$$| h_P(n) | = | S_A(n) - S_B(n) | \quad (16)$$

The logic of (16) is similar to that of the hypothesis-testing system: When $| h_P(n) | = 0$, the stimulus is equally activating both striatal units, so the procedural system has no confidence in its ability to predict the correct response, and when $| h_P(n) |$ is large, the evidence strongly favors one response over the other.

One problem with this approach is $| h_E(n) |$ and $| h_P(n) |$ will typically have different upper limits, which makes them difficult to compare. For this reason, these values are normalized to a [0,1] scale on every trial. This is done by dividing each discriminant value by its maximum possible value.

The amount of trust that is placed in each system is a function of an initial bias toward the hypothesis-testing system, and the previous success history of each system. On trial n, the trust in each system increases with the system weights, $\theta_E(n)$ and $\theta_P(n)$, where it is assumed that $\theta_E(n) + \theta_P(n) = 1$. In typical applications, COVIS assumes that the initial trust in the hypothesis-testing system is much higher than in the procedural system, partly because initially there is no procedural learning to use. A common assumption is that $\theta_E(1) = 0.99$ and $\theta_I(1) = 0.01$. As the experiment progresses feedback is used to adjust the two system weights up or down depending on the success of the relevant component system. This is done in the following way. If the hypothesis-testing system suggests the correct response on trial n then

$$\theta_E(n+1) = \theta_E(n) + \Delta_{OC}[1 - \theta_E(n)] \quad (17)$$

where Δ_{OC} is a parameter. If instead, the hypothesis-testing system suggests an incorrect response then

$$\theta_E(n+1) = \theta_E(n) - \Delta_{OE}\theta_E(n) \quad (18)$$

where Δ_{OE} is another parameter. The two regulatory terms on the end of (17) and (18) restrict $\theta_E(n)$ to the range $0 < \theta_E(n) < 1$. Finally, on every trial, $\theta_P(n+1) = 1 - \theta_E(n+1)$. Thus, (17) and (18) also guarantee that $\theta_P(n)$ falls in the range $0 < \theta_P(n) < 1$. The value assigned to Δ_{OC} should be positively related to the model persistence toward using the hypothesis-testing system, whereas the value assigned to Δ_{OE} should be positively related to the model willingness to switch to the procedural system.

The last step is to combine confidence and trust. This is done multiplicatively, so the overall system decision rule is: Emit the response suggested by the hypothesis-testing system if $\theta_E(n) | h_E(n) | > \theta_P(n) | h_P(n) |$; Otherwise emit the response suggested by the procedural system.

III. Modeling Parkinson's disease with COVIS

PD is caused by the death of DA cells in the SNpc and the VTA, which results in decreased DA levels in the prefrontal cortex and the striatum. In COVIS, DA has a differential effect on the hypothesis-testing and procedural systems.

In the hypothesis-testing system, COVIS assumes that selection and switching both depend on brain DA levels. In particular, selection should improve as levels of DA rise in frontal cortex (up to some optimal level), and switching should improve if levels of DA rise in the head of the caudate nucleus. Thus, the parameter λ should increase with DA levels in frontal cortex, and γ is assumed to decrease with DA levels in the caudate. In addition, DA in the prefrontal cortex has been shown to play a role in focusing

cell activation [7]. Hence, a should increase with DA levels (similar to λ), and σ_E^2 should decrease with more DA (similar to γ).

In the procedural system, DA plays a crucial role in learning: it provides the reward signal in (12). A decreased DA baseline or range can affect the ability of the procedural system to learn stimulus-response associations. Hence, decreasing DA levels in the striatum should decrease the values assigned to D_{base}, D_{slope}, and D_{max}.

Although we currently have no methods for directly measuring brain DA levels in humans (microdialysis can be used in animals), many factors known to affect these levels include: age, mood, genetic predisposition, drug-taking history, and neuropsychological patient status. For example, brain DA levels are known to decrease by approximately 7% per decade of life, and PD patients are thought to have lost at least 70% of their birth DA levels [2]-[3]. Hence, in COVIS, we model an ordinal relationship where DA(Young adults) > DA(Old adults) > DA(PD) (where more DA results in lower γ and σ_E^2, and higher λ, a, D_{base}, D_{slope}, and D_{max}). This general principle guides the following simulations.

IV. Simulations

In this section, we show the adequacy of COVIS to model PD patient performances in two separate tasks, namely perceptual categorization and the simplified WCST.

A. Perceptual Categorization

1) Human Experiment: Ashby and his colleagues [15] compared PD patient performances in rule-based and information-integration categorization tasks with aged-matched (old) control and undergraduate student (young control) performances. The stimuli were four-dimensional binary-valued and varied on background color, symbol color, symbol shape, and number of symbols. The resulting 16 possible stimuli were separated into two categories of equal size with a different stimulus assignment in each of the categorization conditions. In the rule-based condition, a stimulus dimension was selected randomly, and stimuli were assigned to different categories based on their value on the selected dimension. In the information-integration condition, one dimension was randomly selected to be irrelevant. Next, one value from each of the remaining dimensions was randomly assigned the numerosity 1 and the other the numerosity 0. One category was composed of the stimuli whose summed numerosity was higher than 1.5, while the other category was composed of the stimuli whose summed numerosity was smaller than 1.5. Note here that rule-based categories where easily verbalizable (e.g., blue vs. yellow background) whereas no such simple verbalizable rule was available for the information-integration categories.

If a participant was able to correctly classify 10 consecutive stimuli before reaching the 200-trial limit, s/he was classified as a 'learner'. Otherwise, the participant was classified as a 'non-learner'. The dependent measure is the proportion of 'non-learners' in each subject group in each categorization condition. The results show that, compared with old controls, PD patients were impaired in learning the rule-based categories but not information-integration categories (see Fig. 2, black bars) [15].

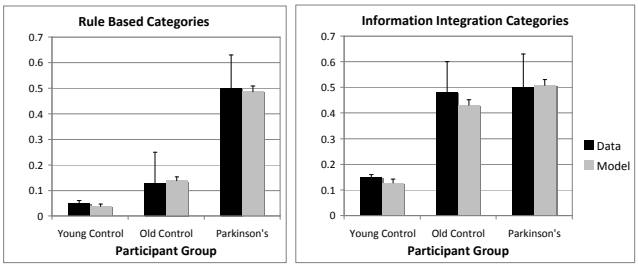

Fig. 2. Human and simulation data for the categorization task from [15].

2) Simulation: Five hundred simulations were run for each subject group in each categorization condition with the COVIS model described in Section II. The stimuli presented to the procedural system were 16-dimensional binary vectors (one vector position for each stimulus, because the stimuli are not visually confusable) while the stimuli presented to the hypothesis-testing system were 4-dimensional binary vectors (one position for each dimension-value pair, again because the stimuli were not confusable). Hence, the procedural system received an object-based representation of the stimuli while the hypothesis-testing system received a feature-based representation of the stimuli. Each system received a separate copy of the feedback. The parameter values are shown in Table I. Note that D_{base} and D_{max} were calculated to reflect the proportion of DA cells remaining as a function of age and diagnosis (i.e., young control = 80% of birth DA cells, old control = 50% of birth DA cells, and PD = 30% of birth DA cells) [2]-[3]. Hence, these were not free parameters. As a result, only three free parameters were used to fit the data (i.e., λ, γ, and D_{slope}). None of the parameters were optimized; reasonable values were assigned using grid search. The simulation results are shown in Fig. 2 (gray bars).

TABLE I
COVIS PARAMETERS IN THE CATEGORIZATION TASK OF [15]

Parameter	Young Control	Old Control	PD
Explicit			
Δ_C	0.0025	0.0025	0.0025
Δ_E	0.02	0.02	0.02
γ	10	10	55
λ	1.5	1.5	.15
a	1	1	1
σ_E^2	0.5	0.5	0.5
Procedural			
θ_{NMDA}	0.002	0.002	0.002
θ_{AMPA}	0.001	0.001	0.001
D_{base}	0.2	0.15	0.1
D_{max}	1	0.6	0.35
D_{slope}	0.8	0.25	0.2
System			
Δ_{OC}	0.01	0.01	0.01
Δ_{OE}	0.04	0.04	0.04

3) Results and Discussion: As seen, COVIS did a good job of simulating the young control, old control, and PD patient performances regardless of the category structure.

This good fit was achieved by varying only three free parameters, and each one of these parameters had a clear conceptual meaning related to the amount of DA found in humans. These results suggest the adequacy of the COVIS model of categorization to account for aging subject and PD patient data in rule-based and information-integration perceptual categorization.

B. The Simplified Wisconsin Card Sorting Test (WCST)

1) Human Experiment: Gotham, Brown, and Marsden [2] compared PD patient performances with age-matched control performances on a battery of psychological tests. One of the tasks used is the simplified WCST [16]. The WCST is a popular clinical measure of conceptual ability and hypothesis testing. In short, the experimenter has a deck of cards with a number of figures on each card. The cards differ in the shape of the figures, the number of figures, and the figures' color. Each one of these dimensions has four possible values (for a total of 64 different cards). On each trial, the participant is shown a card and asked to categorize the card using a rule on one of the dimensions. After a certain number of consecutive correct categorizations, the dimension relevant for categorization is switched. In the simplified WCST, better control is achieved by using only a subset of 24 cards to make the classification of error trials unambiguous. The criterion for rule switching is 6 consecutive correct responses. The experiment ends after 6 sorts have been completed or the entire set of cards has been seen twice. The dependent measures are the number of sorts completed, the number of perseverative errors (error trials where the previously correct rule is used) and number of nonperseverative errors (error trials that are not perseverative errors). The results show that PD patients achieved fewer sorts and made more perseverative errors than age-matched controls (see Fig. 3, black bars).

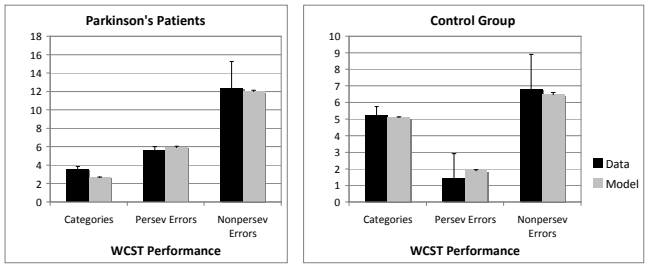

Fig. 3. Human and simulation data for the simplified WCST from [2].

2) Simulation: Five hundred simulations were run for each subject group with the COVIS model described in Section II. The stimuli presented to the procedural system were 64-dimensional binary vectors (one vector position for each stimulus, because the stimuli are not visually confusable) while the stimuli presented to the hypothesis-testing system were 12-dimensional binary vectors (one position for each dimension-value pair, again because the stimuli were not confusable). Hence, the procedural system received an object-based representation of the stimuli while the hypothesis-testing system received a feature-based representation of the stimuli. Note that, while the whole deck of WCST cards was represented, only 24 stimuli were used (corresponding to the subset of cards used in the simplified WCST). Like in the previous simulation, each system received a separate copy of the feedback. The parameter values are shown in Table II. D_{base} and D_{max} were calculated as in Section IV.A.2. Hence, 5 free parameters were varied to simulate the data, and none were optimized (reasonable values were chosen using grid search). The simulation results are shown in Fig. 3 (gray bars).

TABLE II
COVIS PARAMETERS IN THE SIMPLIFIED WCST OF [2]

Parameter	Control	PD
Explicit		
Δ_C	0.05	0.05
Δ_E	0.09	0.09
γ	0.25	6.4645
λ	15	14.2107
a	1.5	0.8385
σ_E^2	0.29	0.4491
Procedural		
θ_{NMDA}	0.057	0.057
θ_{AMPA}	0.0001	0.0001
D_{base}	0.2	0.1
D_{max}	1	0.35
D_{slope}	0.8	0.15
System		
Δ_{OC}	0.05	0.05
Δ_{OE}	0.001	0.001

3) Results and Discussion: The COVIS simulation shows a good match to both simulated PD patients and simulated controls on all three dependent measures. It is interesting to note that this good fit was achieved by only varying DA-related parameters, without any *ad hoc* hypotheses or arbitrary parameter changes. This suggests that COVIS is an adequate model of PD patient performance deficits both in hypothesis testing and perseverance.

V. Conclusion

This article presented a simulation-based account of PD patient symptoms using the COVIS model of categorization. The model achieved a good fit to the data in categorization and in the simplified WCST. It is noteworthy that differential performance between young adults, old adults, and PD patients was achieved simply by changing the amount of DA available in the model. This suggests that COVIS is not only an adequate model of human categorization and the simplified WCST but also of the general role of DA in these tasks. Changing the amount of DA available to the model reproduces behavioral patterns of different human subject populations who correspondingly have different number of DA producing cells in the SNpc and the VTA. Future work should be devoted to the simulation of other tasks with PD patients and the exploration of the interaction of DA depletion with feedback magnitude.

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