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Impaired Cognitive Flexibility and Intact Cognitive Control in Autism: A Computational Cognitive Neuroscience Approach

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Abstract

In people with autism, the ability to enact a behavior in the presence of competing responses appears intact, while the ability to fluently adapt cognitive control in the face of changing task contingencies is impaired. In this paper, the Cross-Task Generalization model (Rougier et al., 2005), which offers a formal account of the effect of dopamine on frontal cortex function, is used to capture performance of both normally functioning individuals and people with autism on a classic test of cognitive control, the Stroop task (Stroop, 1935), and one of cognitive flexibility, the Wisconsin Card Sort Test (Berg, 1948). By weakening the effect of the dopamine's signal on frontal cortex, the model fits quantitative and qualitative results of autistic performance on these tasks and demonstrates the potential usefulness of this approach in autism research.

Introduction

Autism spectrum disorders (ASD) are pervasive developmental disorders with a prevalence estimated at 1 in 166 live births. Autism is characterized by severe social deficits, problems in both verbal and non-verbal communications, motor skill deficiencies, disruptive stereotypic movements, and occasionally self-injurious behavior. Genetic factors are evident in the disorder, shown through inheritability as well as the fact that most people with autism are male. There has been steady progress in early identification of the behavioral characteristics of the disorder, as well as early intervention techniques, but no consensus has been reached concerning the neural basis of autism.

People with autism are impaired across a range of cognitive tasks, including planning (Bennetto et al., 1996), theory of mind tasks (Baron-Cohen et al., 1985), and tasks requiring spontaneous generation of novel behaviors and ideas (Turner, 1999). Many cognitive faculties are spared, however, including working memory retention (Ozonoff and Strayer, 2001). One particularly interesting aspect of the cognitive profile demonstrated by people with autism is that cognitive flexibility has been shown to be impaired in experimental tasks such as the Wisconsin Card Sort Test, while cognitive control, as measured by tests such as the classic Stroop paradigm, remains robust and relatively unaffected (Ozonoff and Jensen, 1999). Cognitive control describes our ability to enact a behavior in the presence of a distracting or more automatic competing response. Cognitive flexibility can be described as our ability to fluently adjust cognitive

control as task contingencies change. Capturing this dichotomy is a considerable challenge for any theoretical account whose goal is explaining autistic behavior.

A potentially valuable and novel approach to autism research involves leveraging the tools of computational cognitive neuroscience to help formalize the neural mechanisms responsible for the pattern of behavior found in people with autism. Computational models of cognition force researchers to be explicit in their assumptions, as well as in the mechanisms employed, during scientific conjecture. The formal nature of these models allow us to form precise and testable hypotheses concerning the mechanisms responsible for the phenomena of interest.

The Cross-Task (XT) Generalization model (Rougier et al., 2005) is a model of cognitive control and flexibility grounded in contemporary accounts of the role of dopamine in prefrontal cortex (PFC) function. XT has been used to capture performance of both normal functioning and frontally damaged individuals on the Wisconsin Card Sort Test (WCST) and the Stroop task. We have found that, in the context of this model, weakening the effect of dopamine on frontal functioning is sufficient to capture autistic performance on both tasks, showing significantly increased perseverative errors during WCST, signaling impaired flexibility, while leaving cognitive control intact as demonstrated by a lack of effect on performance of the Stroop task. This observation suggests that "executive dysfunction" symptoms in autism may be mediated by PFC / dopamine interactions.

Background

Psychological & Neuroscientific Frameworks

Three main cognitive theories have been proposed for understanding cognitive differences in autism: theory of mind, weak central coherence, and executive dysfunction. Theory of mind (Baron-Cohen et al., 1985) describes the ability to attribute, evaluate, and manipulate mental states. This ability is believed to be impaired in autism, and this impairment is used as a possible explanation for the severe social deficits demonstrated by people with autism. Weak Central Coherence (WCC) is described as a difference in cognitive processing style, rather than a deficit in abilities (Happé, 1999). In WCC, it is believed that contextual processing of information is sacrificed in favor of a more focused "piecemeal" processing style. The Executive Dysfunction hypothesis views autism as emerging from a deficit in executive control

over behavior (Hughes et al., 1994). Executive functioning is used as an umbrella term for processes such as planning, inhibition, and cognitive flexibility, and is traditionally associated with frontal processes.

The success of these psychological frameworks in explaining many behavioral characteristics of ASD could be solidified if a formal account of the underlying biological mechanisms which give rise to observed behavior could be provided. Neuroscientific frameworks thus far have had little success in providing a unified view of the neural mechanisms responsible in autism. Current conjectures range from observed abnormalities in the cerebellum (Courchesne, 1987), to dysfunction of the serotonin system (Chugani, 2004), to specific prefrontal deficits (Mundy, 2003), to distributed theories implicating multiple brain areas such as widespread dysfunction throughout the hippocampus, the amygdala, the oxytocin-opiate system, and temporal and parietal association cortices (Waterhouse et al., 1996).

Psychological and neuroscientific theories have the potential to constrain and inform each other, unifying research concerning the neural basis of autism. However, it is unclear at this point, given the multiplicity of brain areas implicated in ASD, how best to integrate the cognitive neuroscience and cognitive psychology of autism.

Computational Approaches

The formal and explicit nature of the tools of computational cognitive modeling provide a novel method for approaching this problem. In order for computational models to be useful in this endeavor, they must be constrained by both bottom-up (neurobiological mechanisms) and by top-down (observed behavior) considerations. Most existing models of autism have been fairly abstract in nature, making little contact with specific neurobiological considerations (Cohen, 1994; McClelland, 2000; O’Loughlin and Thagard, 2000). Even those models of autism which have incorporated biology in their framework have thus far only matched qualitative patterns of behavior in people with ASD, not attempting to account for any quantitative behavioral data (Gustafsson, 1997). Models more tightly coupled with observed functional properties of neurobiological systems and constrained by actual behavioral data will be able to more precisely inform theories of ASD.

Dopamine & Temporal Difference Learning

The precise timing and firing rates of mesolimbic dopamine (DA) neurons have been demonstrated to encode a measure of change in future expected reward (Schultz et al., 1997). Linking machine learning and biology, this same change in expected future reward is also the key variable in a reinforcement learning paradigm known as Temporal Differences (TD) learning. This connection has led researchers to formalize the role of midbrain DA neurons in learning (Barto, 1994; Montague et al., 1996), equating the firing rate of the DA cells with the amount of change in expected future reward. In TD learning this change in expected reward across two consecutive time steps is given by:

$$\delta(t) = r(t) + \gamma V(t+1) - V(t) \quad (1)$$

Where $r(t)$ is a continuous reward value which is delivered at each time step based on performance, $V(t)$ and $V(t+1)$ are the expected future rewards for times t and $t+1$, $\delta(t)$ is the change in expected future reward, or TD Error, and γ is a constant scaling factor, where $0 < \gamma < 1$. Adjusting γ changes the amount by which temporally distant rewards are discounted as compared to rewards that can be attained in the near future.

Computational Models of PFC

PFC has been broadly implicated as playing a central role in cognitive control and cognitive flexibility. Under some accounts, the active maintenance of abstract rule-like representations in PFC is necessary to provide a task-appropriate processing bias to more posterior brain areas (Cohen et al., 1990). Our work builds on an existing body of modeling work having strong ties to biology, leveraging an existing formalism connecting phasic DA and PFC function (Braver and Cohen, 2000). This formalism states that a change in expected future reward, linked to the firing rate of DA neurons, can be used as a gating mechanism, informing PFC when to actively maintain its current control state and when to gate in a new one. These models have been successful in tying frontal disturbances, such as those found in schizophrenia, to deficits in cognitive control (Cohen and Servan-Schrieber, 1992) and cognitive flexibility (Braver and Cohen, 1999). A recent elaboration of this model, XT (Rougier et al., 2005), is the first neuroscientific model able to provide quantitative fits to a hallmark task of cognitive control, the Stroop task, and a widely used measure of cognitive flexibility, WCST, in both neurologically intact and frontally damaged people.

The XT Model

XT is a model of cognitive flexibility and cognitive control built using the biologically inspired Leabra framework (O’Reilly and Munakata, 2000). (See Figure 1.) The input of XT consists of a layer of units using a localist code to specify stimuli being used in the current task. We can think of the rows of the input layer representing different dimensions (e.g. color, shape, texture) and the columns indexing features across each dimension. (See Figure 2.) The response layer is analogous in structure to the input layer, with a winner-take-all mechanism used to simulate lateral inhibition between the units, facilitating a competition for a single output response. There is one additional unit—a “No Response” unit—included in the response layer, which provides the network with an alternative to the stimuli present in the input layer.

The PFC layer provides top-down cognitive control using abstract rule-like representations. In previous models, the PFC representations were hand coded by the modeler, with the question of how these representations develop brushed aside. In contrast, the rule-like PFC representations in XT are learned through extensive experience with the stimuli. This extended amount of experience provides a reasonable account for the protracted

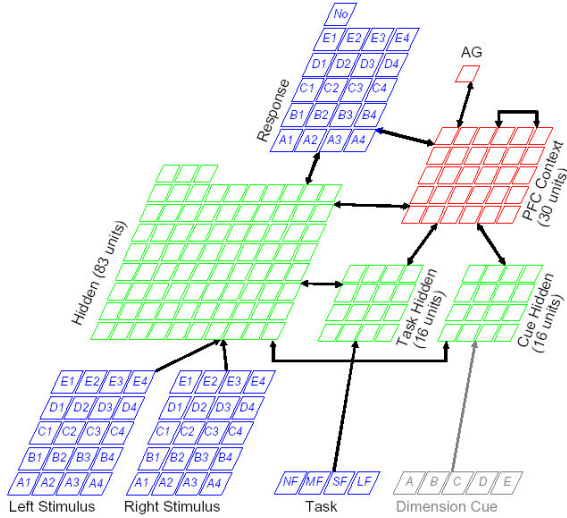


Figure 1: XT Model Architecture

period of the development of PFC during adolescence, as well as how control can emerge through experience, supported by biologically based self-organizing mechanisms.

The Dimension Cue layer is used in the model to inform the network of what stimulus dimension (e.g. color) is relevant. For example, the Dimension Cue layer is used in the Stroop task to inform the network when it should prefer color naming to word reading, or vice versa. Each unit in the Dimension Cue corresponds to a dimension in the stimulus (input) layer being available. If no Dimension Cue unit is activated, the network is uninformed as to what dimension is currently relevant, and must rely on a random search method in order to succeed. This uninformed search strategy is used during the modeling of WCST performance.

The Task layer is vital in the training of the XT network, with each unit representing a different task for the network to perform. Rougier et al (2005), show that a large breadth of experience is necessary for useful rule-like representations to develop in PFC using the XT framework, necessitating the exposure of the network to multiple tasks during training. For our simulations, the Task layer is held constant after training, always requiring the network to perform the “Naming Feature” task. “Naming Feature” requires the network to name one feature of the input stimuli, using feedback to adjust the dimensional representation in PFC in order to name the correct feature.

The flexible adaptation of cognitive control is implemented using a DA-based adaptive gating (AG) mechanism, depicted in XT by the AG unit. (See Figure 1.) The AG mechanism computes the expected future reward based on the TD learning algorithm, with reward delivered based on the network’s performance. When the model performs better than expected (positive TD Error, $\delta(t) > 0$) the PFC representations are strengthened using an intrinsic maintenance current to stabilize PFC. When the model performs worse than expected

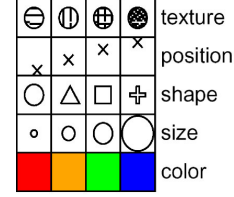


Figure 2: Stimulus Input Layer: Caricature of input to the XT model, with rows portraying stimulus dimension (color, shape, size, etc) and columns indexing feature values across dimensions (small, medium, large, etc.)

(negative TD Error, $\delta(t) < 0$), the PFC representations are destabilized, allowing a new, possibly more appropriate, PFC representation to be maintained. In the model, the $\delta(t)$ value directly modulates excitatory ionic maintenance currents (g_m below) of units in PFC by:

$$g_m(t-1) = 0 \text{ if } |\delta(t)| > \theta \quad (2)$$

$$g_m(t) = g_m(t-1) + \delta(t)a \quad (3)$$

Therefore, a positive $\delta(t)$ will result in an increase in active maintenance of PFC representations, while a negative $\delta(t)$ will destabilize PFC. The value θ represents a threshold value for the ionic currents. If the TD error, $\delta(t)$, exceeds this amount ($\theta = .5$ in all simulations), then the maintenance currents, g_m , are effectively reset.

Using this mechanism and a unified computational framework, XT has been successful in providing strong quantitative fits to human performance on tasks measuring both cognitive control and flexibility.

Modeling Approach

Dopamine levels have been studied in people with autism, and a difference in activation in PET studies (Fernell et al., 1997) as well as an overall difference in HVA (a DA metabolite) (Martineau et al., 1992) have been observed. In clinical studies, haloperidol, a DA antagonist, has produced clinical benefits in mitigating some of the disruptive attributes of autism (Posey and McDougle, 2000). Given the formalized relationship between the role of DA on PFC functioning and PFC’s role in cognitive control and the flexible adaptation of control, we hypothesize a deficit in DA functioning can account for the impaired cognitive flexibility seen in people with autism, while leaving cognitive control robust and relatively unaffected. Using the XT framework, we test this by reducing the effect of the DA analog in the model by scaling the TD Error, $\delta(t)$, by a constant factor, κ , where $\kappa = 1$ for normally functioning individuals and $\kappa < 1$ for people with autism¹. The TD Error $\delta(t)$ now becomes:

$$\delta(t) = \kappa[r(t) + \gamma V(t+1) - V(t)] \quad (4)$$

¹The scaling of $\delta(t)$ by κ is the only parameter modified from the original XT model to capture autistic performance.

...where $0 < \kappa \leq 1$. When modeling autistic performance in both WCST and Stroop, a κ value of 0.53 produced the best results and was used in all simulations. Qualitatively, this reduction of the DA signal can be seen as decreasing the efficacy of the PFC gating system, resulting in less efficient destabilization of the representations in PFC. It is worth noting that the modeled DA signal remains agnostic as to the precise quantitative nature of actual DA levels. It is possible, for instance, that an optimal firing rate of the midbrain DA neurons exists for efficient PFC gating. Thus, *either* too much or too little DA could have deleterious results on the effectiveness (a lower κ value) of the DA based PFC gating system. Alternatively, tonic and phasic components of the DA signal could have differential effects on PFC gating (Cohen et al., 2002).

Modeling WCST

The WCST consists of a deck of cards, which contain stimuli varying along three dimensions (e.g. color, shape, quantity) and across four different features per dimension (e.g. for color dimension: red, blue, green, & yellow). The participants in the study are told to sort the cards into piles, but are not given any explicit instructions on how to correctly do this. Instead, subjects are given sparse feedback —“Correct” or “Incorrect”— until the proper sorting strategy is discovered. After the sorting rule (e.g. sort by color) is learned by the participant, and 10 consecutive correct sorts are accomplished, the rule is changed without informing the subject. This procedure continues until either 6 correct categories (sets of 10 correct consecutive sorts) are achieved, or all 127 cards in the deck are exhausted. Errors are recorded as incorrect sorts, with perseverative errors scored as an incorrect sort that used the last correct sorting rule. Success at WCST requires the ability to flexibly change the dimension being maintained by PFC as the sorting rules change.

Modeling WCST in the XT framework required using only three of the five possible input dimensions, facilitating a tighter link to the actual WCST. All of the rules mentioned above for administering WCST were followed in the model’s implementation, recording errors and perseverative errors as they occur.

Modeling the Stroop Task

The Stroop task is a classic measure of cognitive control and the ability to inhibit a prepotent response. In Stroop, the stimuli are different color words, presented in various colored fonts. The participants are asked to either read the word or to name the color of the font in which the text is presented. People are faster overall at reading the word as opposed to naming the color of the word. Furthermore, when comparing congruent (e.g. the word “red” in red font) versus the incongruent (the word “red” written in green font) conditions, people only show an interference effect when naming the color and not when reading the word. In other words, there is an increase in reaction time for color naming, but not for word reading, when comparing congruent to incongruent cases.

Cohen and Servan-Schreiber (1990) provide a computational account of the Stroop task, positing that the greater overall strength of the word reading pathway is due to greater familiarity with word reading, making this pathway stronger and more automatic compared to the color naming pathway. In their model, a PFC-like mechanism provides top-down biasing on the respective pathways based on the current goal (e.g. “read the word” or “name the color”). The control provided from PFC is necessary to overcome the pre-potent word reading pathway in the trials when the network is required to name the color. This results in an increase in reaction time in the color naming incongruent condition, but not in the word reading incongruent condition. This is attributed to the greater overall competition created when the network needs to overcome the stronger word reading pathway.

In order to simulate this imbalance of processing strengths in our model, we manipulated the frequency in which one dimension was experienced during training, making the dimension relevant only 25% as often as the other dimensions. The competition between the color naming and word reading pathways is simulated by co-activating features in this weaker dimension, corresponding to the color naming pathway, and a strong dimension, representing the word reading pathway. The PFC layer provides the crucial top-down biasing mechanism, consistent with the model of Cohen & Servan-Schreiber, to help resolve the competition appropriately, based on the goal of the task. The settling time of the network resulting from this competition is used as an analog to reaction time, and is scaled using a single free parameter allowing us to directly compare model results to human data.

Results

In order to compare WCST and Stroop performance, 100 networks were fully trained using the XT framework standard training procedure, stopping after a maximum 100 epochs of training or when the network achieved a stringent performance criteria. Following this training, each network was tested under both conditions of DA modulation, on each of the WCST and the Stroop tasks. The 100 networks were each treated as individual subjects for data analysis.

WCST Results

Four main measures were used in evaluating the performance on the WCST task:

1. Total Number of Errors
2. Percentage of Total Errors
3. Total Number of Perseverative Errors
4. Percentage of Perseverative Errors

Our simulations of both normally functioning persons and individuals with autism provide reasonable results for all measures.

All differences between the simulated performance of normally functioning individuals and that of people with autism were statistically reliable (see Table 1), and consistent with previous studies (Prior and Hoffman, 1990).

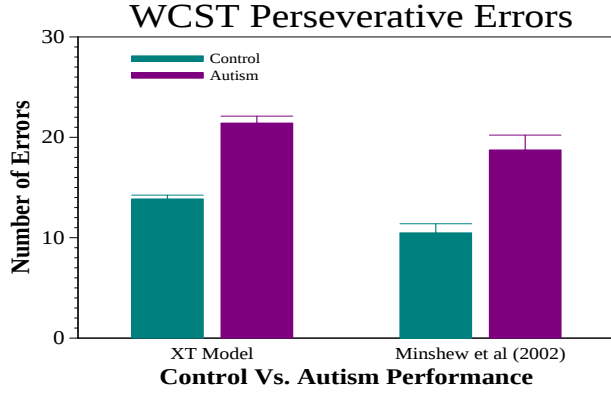


Figure 3: Comparing model performance on WCST perseverative errors for normally functioning and individuals with autism to a previous study. (Minshew et al., 2002)

Importantly, the perseverative error measure is significantly higher in the DA modified version of our model compared to the model of normal function (Figure 3), matching a pattern of results reported in the literature (Ozonoff and Jensen, 1999).

Stroop Results

Model performance on the Stroop task is able to qualitatively and quantitatively fit human performance (Figure 4). The model of intact DA function shows the classic Stroop reaction time results. The pre-potent word reading dimension shows uniform reaction times across both congruent and conflict conditions, while the weaker color naming dimension shows a slowing in reaction times when the stimuli are incongruent. Autistic performance, obtained by scaling the strength of the DA signal in the model, showed the same pattern of results with no significant increase in the overall Stroop effect ($F(1,198) = 0.62$; $p > 0.43$) consistent with past findings (Ozonoff and Jensen, 1999).

Discussion & Future Work

Leveraging a formal characterization of the effect of DA on PFC functioning, we have shown that a single manipulation, reducing the efficacy of the DA signal, is sufficient to capture the performance of people with autism on basic tests of cognitive flexibility (WCST), and cognitive control (Stroop). In WCST, our models of autistic performance commit significantly more perseverative errors compared to controls, while in Stroop there is no

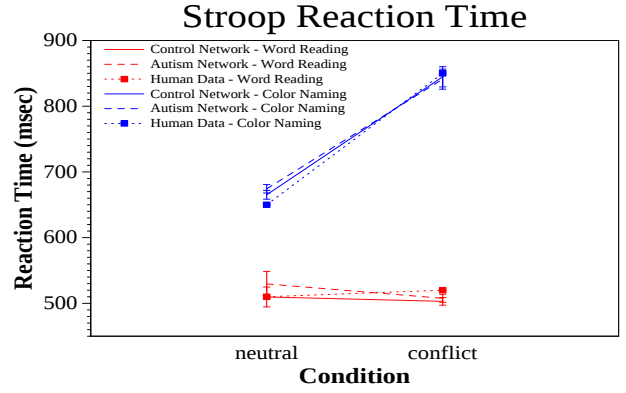


Figure 4: Stroop reaction time plot comparing the simulated autistic and normally functioning network’s performance to human data. Human data from (Dunbar and Macleod, 1984)

significant change in performance, matching the cognitive profile of impaired cognitive flexibility and relatively robust cognitive control found in the performance of people with autism.

It is interesting to note that our model ties a difference in DA function to frontal lobe dysfunction in people with autism. This provides a possible bridge to Executive Dysfunction, traditionally linked to frontal processes, in people with autism (Hughes et al., 1994).

By reducing the effect of DA modulation only after the network is completely trained and PFC representations are fully developed, we limit our model’s ability to capture the time course of autism. Autism is, at its core, a developmental disorder. A major direction of our future work will involve observing how DA modulation affects the developmental trajectory of the model. Specifically, we will examine how the difference in DA strength affects the learning of PFC representations and how this subsequently affects performance.

Using computational models inspired and constrained by our existing knowledge of biology is a relatively untapped resource for exploring the neurological underpinnings of autism. Our initial results using these tools are encouraging and show a promising future direction for research on autism spectrum disorders.

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Table 1: WCST Model Results

WCST Measure	Normal	Autism	F(1,198)	p <
Total Errors	34.66	44.95	15.33	.001
% Total Errors	35.80%	41.76%	13.23	.001
Total Perseverative Errors	13.84	21.40	88.13	.001
% Perseverative Errors	15.07%	20.38%	63.47	.001

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