Modelling cognitive processing of healthy controls and obsessive compulsive disorder subjects in the antisaccade task

Vassilis Cutsuridis

School of Computer Science, University of Lincoln, Lincoln, U.K. Email: vcutsuridis@lincoln.ac.uk, vcutsurdis@gmail.com

Abstract Antisaccade performance deficits in obsessive-compulsive disorder (OCD) include increased error rates and antisaccade latencies. These deficits are generally thought to be due to an impaired inhibitory process failing to suppress the erroneous response. The superior colliculus has been suggested as one of loci of this impaired inhibitory process. Previously recorded antisaccade performance of healthy and OCD subjects is re-analyzed to show greater variability in mean latency and variance of corrected antisaccades as well as in shape of antisaccade and corrected antisaccade latency distributions and increased error rates of OCD patients compared to healthy controls. A neural accumulator model of the superior colliculus is then employed to uncover the biophysical mechanisms giving rise to the observed OCD deficits. The model shows: (i) the increased variability in latency distributions of OCD patients is due to a more noisy accumulation of information by both correct and erroneous decision signals; (ii) OCD patients are *less* confident about their decisions than healthy controls; (iii) competition via lateral inhibition between the correct and erroneous decision processes, and *not* a third independent inhibitory signal of the erroneous response, accounts for the antisaccade performance of healthy controls and OCD patients.

Keywords: antisaccade paradigm, eye movements, superior colliculus, accumulator model with lateral inhibition, response inhibition, impulse control

Introduction

In the antisaccade paradigm participants suppress a reflexive saccade (error prosaccade) in favor of a saccade to a position in the opposite hemifield (correct antisaccade) (Hallett, 1978). Two processes take place during this paradigm: (1) sup-

pression (or inhibition) of an error prosaccade towards the peripheral stimulus, and (2) generation of a volitional saccade to the opposite direction (antisaccade) (Everling and Fischer, 1998; Munoz and Everling, 2004). The reaction times (RT) of error prosaccades, antisaccades and corrected antisaccades, and the error rate are some of the measures of antisaccade performance (Hutton and Ettinger, 2006) with the error rate being the most reliable measure of it. A large study of healthy young males has reported that error prosaccade and antisaccade RTs are highly variable and the error rate is about 20-25% (Smyrnis et al., 2002; Evdokimidis et al., 2002).

A recent experimental study reported an increase in error rates and in latency of corrected antisaccades in OCD patients (Damilou et al., 2016). The antisaccade performance deficit in OCD was speculated to be due a common dysfunctional network of brain structures including the (pre)frontal and posterior parietal cortices and superior colliculus (SC). In this network there is a reported deficit in erroneous response inhibition control (Chamberlain et al., 2005).

Models of decision making involves a gradual accumulation of information concerning the various potential responses (Cutsuridis et al., 2007; Cutsuridis, 2010; Noorani and Carpenter, 2013, 2014, Cutsuridis et al., 2014; Cutsuridis, 2015, 2017). As soon as the target appears, a decision process starting at some baseline level T_0 representing the prior expectation, begins to rise at a constant rate r until it reaches a threshold T_h representing the confidence level required before the commitment to a particular course of action. Once T_h is crossed, then a response towards the target is initiated. Response time (RT) is the time from the onset of the decision process till when the decision signal crosses T_h. The rate of rise is sometimes assumed to vary randomly from trial to trial, with a mean μ and variance σ^2 (Reddi and Carpenter, 2000). Changes in the baseline level of activity, the rate of rise or the threshold often result in changes in response latency. Prior expectation and level of activation of intention influence the baseline levels of activation.

The scope of the present modelling study is to uncover what goes wrong neurally (i.e. the mechanisms) in OCD, so the model's behaviour best fits the experimental observations (error rates and response time distributions) from both participant populations (healthy controls and OCD patients). For this reason previously recorded error rates and latencies of healthy and OCD participants (Damilou et al., 2016; Evdokimidis et al., 2002) were re-analyzed to show that OCD patients display higher error rates, increases in mean latency and variance of corrected antisaccades, and greater variability in shape of antisaccade and corrected antisaccade latency distributions relative to healthy participants. The Cutsuridis and colleagues (2014) model was then employed to decipher the biophysical mechanisms that gave rise to these antisaccade performance deficits in OCD. The model showed that (i) increased variability in latency distributions of OCD patients was due to a more noisy accumulation of information by both (pre)frontal and posterior parietal centers representing the volitional (correct antisaccade) and reactive (erroneous prosaccade) decision signals, respectively, (ii) OCD patients were *less confident* about their decisions compared to healthy controls (i.e. the decision threshold level T_h value is lower in healthy controls than in OCD patients), and (iii) competition between the correct and erroneous decision processes, and *not* a third top-

down STOP of the erroneous response, accounted for the antisaccade performance of both healthy controls and OCD patients.

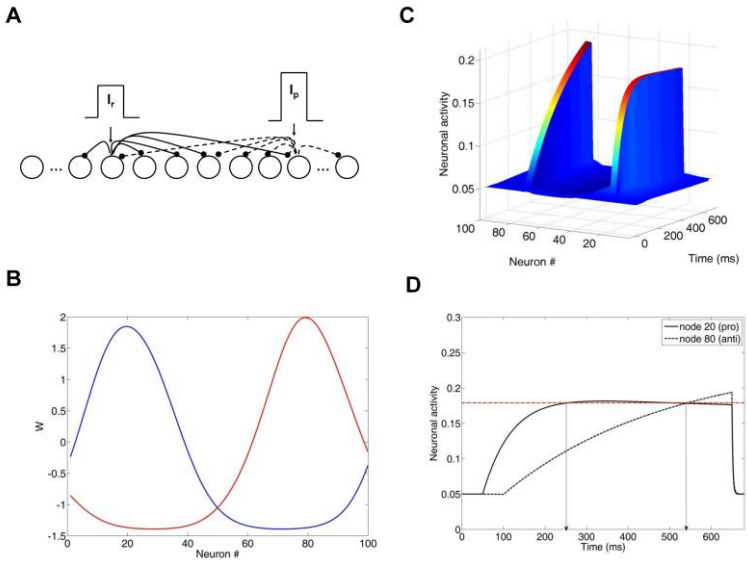

Fig. 1. Adapted with permission from Cutsuridis et al. (2014). (A) Neural network model. Neurons are represented as firing rate nodes. Short-range lateral excitation and long distance lateral inhibition was assumed between all nodes in the network. The left model half was activated by a reactive input I_r representing the error prosaccade decision signal, whereas the right model half was activated by a planned input I_p representing the antisaccade decision signal. The strengths of the inputs were not equal (I_p = 1.5*I_r). (B) Lateral interaction kernels W for nodes 20 and 80 modelled as a shifted Gaussians. The kernels for nodes 20 and 80 were excitatory for the nearby nodes and inhibitory for the distant ones. (C) Neuronal activities of all nodes in the network as a function of time (ms). (D) Neuronal activity of nodes 20 and 80 as a function of time. Node 20 encoded the reactive input (error prosaccade) and node 80 encoded the planned input (antisaccade). When both activities crossed the threshold (dotted horizontal line), then an eye movement decision was made. In this case, an error prosaccade was initiated first followed by a corrected antisaccade.

Experimental data

The data (antisaccade performance of healthy controls and OCD patients) were derived from two previously published studies (Evdokimidis et al., 2002; Damilou et al., 2016). Detailed description of these data including details about the participants, eye movement recordings, task description and analysis can be found in Cutsurdis (2017) study.

The model

The model with its mathematical formalism was initially introduced in Cutsuridis et al. (2014) study. Interested readers are referred to this study for detailed description of the model. To assist the readers of this chapter and increase the readability of it a brief description of the model is provided here. The model is a one-layer SC neural network with lateral inhibition and firing rate nodes (neurons) representing the SC build-up neurons (Fig. 1A). The total number of nodes in the network is N. Short-range lateral excitation and long distance lateral inhibition is assumed between all nodes in model. The lateral interaction kernel w_{ij}, which allows for lateral interactions between model nodes, is a shifted Gaussian, which depends only on the spatial distance between nodes and it is positive for nearby nodes to the node activated by the input and negative for distant nodes (Fig. 1B). Model inputs are of two types: (1) a reactive input (I_r), which represents the error prosaccade decision signal and is hypothesized to originate from the posterior parietal cortices (Munoz and Everling, 2004) and (2) a planned input (I_p), which represents the correct antisaccade decision signal and originates in the model from the frontal cortical areas (Munoz and Everling, 2004). Each input is integrated in opposite model half according to the following way: if the reactive input activates a node and two of each nearest neighbors on each side in the left model half, then the planned input activates the mirror node and its two nearest neighbor nodes on each side in the right model half, and vice versa. The strengths of the external inputs are not equal ($I_p > I_r$; see Table 3 for values). The reactive input is presented first at time t = 50 ms, followed by the planned input, which is presented 50 ms later (t = 100 ms) in accordance to experimental evidence (Becker, 1989). Both inputs remain active for 600 ms.

	Median RT in ms						% Error rate	
	Error prosaccade		Antisaccade		Corrected antisaccade			
	Sim	Exp	Sim	Exp	Sim	Exp	Sim	Exp
Controls	214.72	211.09±49.71	262.72	268.61±46.76	136.97	128.84±53.62	31.24	20.79±0.19
OCD Patients	207.84	203.81±53.17	277.58	275.73±52.68	188.92	160.34±42.55	41.58	47.96±0.3

Table 1. Simulated and experimental median saccade reaction times and their standard deviations and percent error rates for controls and patients with OCD.

Results

Experimental latency distributions

The controls and OCD patient experimental data (Evdokimidis et al., 2002; Damilou et al., 2016) are re-analysed here using the methodology presented in Cutsuridis and colleagues (2014) study. The mean inter-individual of the median intra-individual RT for the error prosaccades was found to be 211.09 ms (SD: 49.71) for the controls and 203.81 ms (SD: 53.17) for the patients (Fig. 2A; see Table 1). This 7.28 ms difference was not statistically significant ($t_{64} = 0.57$, P = 0.57). The RT distributions for patients were not much broader than those for the controls, indicating a smaller RT variability. The group coefficient of variation of RT defined as the inter-quartile RT range ($Q_{75} - Q_{25}$) divided by the median RT was not significantly different for the patients (0.35, SD: 0.21) and the controls (0.30, SD: 0.21) ($t_{64} = 0.4$, P = 0.85) (see Table 2).

An average cumulative RT distribution for each group (controls vs. patients) (Fig. 3A) was computed to further investigate if there is a shape difference between the controls and patients distributions by organizing the RT for each subject in ascending order and percentile values were calculated (e.g. the RT for the 5% percentile, the 10% percentile, the 15% percentile, …, the 95% percentile, the 100% percentile). The percentile values were then averaged across the group to give average group percentile values. It has been shown that the average distribution retains the basic shape characteristics of the individual distributions (Ratcliff, 1977). To test the difference between the group distributions for patients and controls, a Wilcoxon rank sum test was used. It can be observed that the two cumulative distributions did not differ in shape (Z = 1.008, P =0.31).

	Coefficient of variation (CV)					
	Error prosaccade		Antisaccade		Corrected Antisaccade	
	Simulated	Experimental	Simulated	Experimental	Simulated	Experimental
Controls	0.22	0.30± 0.21	0.19	0.24±0.07	0.77	0.83±0.41
OCD Patients	0.32	0.35±0.21	0.26	0.31±0.12	0.77	0.54±0.24

Table 2. Simulated and experimental coefficients of variation (CV) of error prosaccades, antisaccades and corrected antisaccades for controls and patients with OCD performing the antisaccade task.

A similar analysis was used for the antisaccades and corrected antisaccades for both the controls and patients. The mean inter-individual of the median intra-individual RT for the antisaccades was 268.61 ms (SD: 46.76 ms) for the controls and 275.73 ms (SD: 52.68 ms) for the patients (Fig. 2A; see Table 1). This 7.12 ms

difference was not statistically significant ($t_{64} = 0.57$, P = 0.57). The coefficient of variation of antisaccade RTs was also not significantly different for the patients (0.31, SD: 0.12) and for the controls (0.24, SD: 0.07) ($t_{64} = 2.62$, P = 0.31) (see Table 2).

The average cumulative RT distribution for each group (controls vs. patients) (Fig. 3B) was computed as before. To test the difference between the antisaccade group distributions for patients and controls, a Wilcoxon rank sum test was used. It can be observed that the two cumulative distributions differ in shape and this difference was significant (Z = 2.65, P = 0.008).

The mean inter-individual of the median intra-individual RT for the corrected antisaccades was 128.84 ms (SD: 53.62 ms) for the controls and 160.34 ms (SD: 42.55 ms) for the patients (Fig. 2A; see Table 1). This 31.5 ms difference was statistically significant ($t_{64} = 2.60$, P = 0.0115). The coefficient of variation of RT was found to be significantly different for the controls (0.83, SD: 0.41) and the patients (0.54, SD: 0.24) ($t_{64} = 3.42$, P = 0.0011) (see Table 2).

The average cumulative RT distribution for each group (controls vs. patients) (Fig. 3C) was similarly computed and a Wilcoxon rank sum test was used to test the difference between the group distributions for patients and controls. The two cumulative distributions differed in shape and this difference was significant (Z = 3.92, $P < 10^{-3}$).

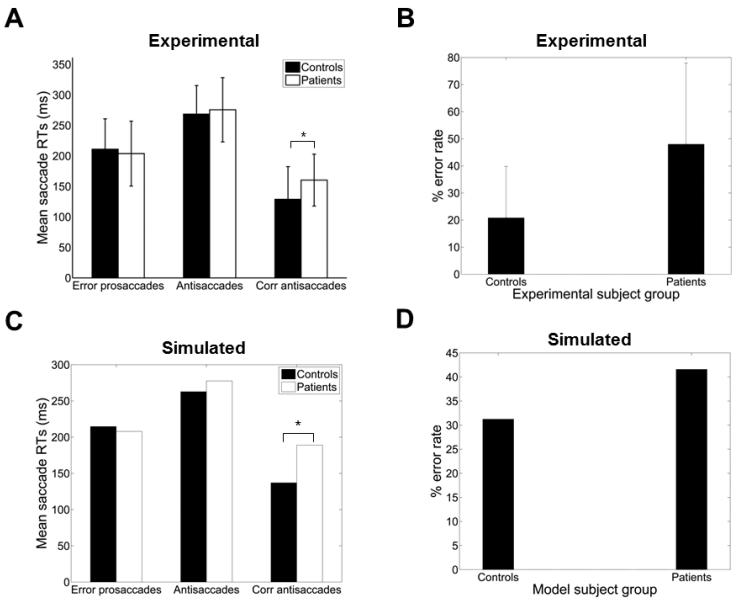

Fig. 2. (A) Mean of median error prosaccade, antisaccade, and corrected antisaccade reaction times (RTs) for controls and OCD patients. (B) Mean percent error rate of controls and OCD patients performing the antisaccade task. (C) Simulated median error prosaccade, antisaccade and corrected antisaccade reaction times (RTs) for controls and OCD patients. (D) Simulated percent error rate for controls and OCD patients performing the antisaccade task.

Simulated latency distributions

To simulate the experimental data, the Cutsuridis and colleagues neural network model of the antisaccade performance of healthy controls and schizophrenia patients (Cutsuridis et al., 2014) was employed and extended it into the realm of OCD (Fig. 1A). To fit the experimental data, in each trial run in the left and right SC the time constants τ of the internal states of each node took values from two different normal distributions with means μ_1 and μ_2 and standard deviations σ_1 and σ_2, respectively. The model was run for 5000 trials. In each trial the error prosaccade, antisaccade and corrected antisaccade latencies were recorded. In the model the error prosaccade reaction time was estimated as the time interval from the onset of the reactive input until the time the activity of the node encoding the reactive input reached a preset threshold (T_h) plus an additional 30 ms (Fig. 1D). The antisaccade reaction time was estimated as the time interval from the onset of the reactive input until the time the activity of the node encoding the planned input reached the threshold plus 30 ms (Fig. 1D). The corrected antisaccade reaction time was the time interval from threshold crossing of the error node activity until the threshold crossing of the correct node activity.

To simulate the error prosaccade, antisaccade and corrected antisaccade RT distributions as well as the error rates of both healthy controls and OCD participant groups, the integration constants τ (μ and σ) for both nodes that integrated the reactive (μ_1 and σ_1) and planned (μ_2 and σ_2) inputs were varied. In the control condition, $\mu_1 = 0.01787$, $\sigma_1 = 0.003$, $\mu_2 = 0.0056$ and $\sigma_2 = 0.0016$, whereas in OCD condition $\mu_1 = 0.0165$, $\sigma_1 = 0.005$, $\mu_2 = 0.0047$ and $\sigma_2 = 0.002$. In both conditions, the threshold value at which as a decision was reached (parameter T_h in Table 3) was higher in OCD patients than in healthy controls. The simulated median RTs for the error prosaccades, antisaccades and corrective antisaccades were 214.72 ms, 262.72 ms and 136.97 ms, respectively for the model controls and 207.84 ms, 277.58 ms and 188.917 ms, respectively for the model patients. The simulated median RT values are very close to the experimental ones (Fig. 2C; see also Table 1). The simulated coefficients of variation (CVs) for the error prosaccades, antisaccades and corrected antisaccades were 0.22, 0.19 and 0.77, respectively for the controls and 0.32, 0.26 and 0.77, respectively for the patients. The simulated CV values are very close to the experimental ones (see Table 2).

As before the simulated average cumulative RT distributions for error prosaccades, antisaccades and corrected antisaccades for both groups (model controls vs. model patients) by organizing the RT for each subject group from each trial run in ascending order and calculating the percentile values (e.g. the RT for the 5% percentile, the 10% percentile, the 15% percentile, …, the 95% percentile) were computed. The percentile values were then averaged across trial runs (5000 trial runs) for each subject group to give average subject group percentile values. Carpenter and Williams (1995) showed that if the cumulative RT distribution is plotted using 1/RT in a reciprobit plot, then the RTs will fall on a straight line. Thus, the average cumulative distribution data of RT (error prosaccade, antisaccade and corrected antisaccade) for the experimental and simulated controls and patients in a reciprobit plot were transformed. A best-fitting regression line was

computed for each behavioural category (error prosaccade, antisaccade and corrected antisaccade) in each subject group (controls and patients). An R correlation coefficient was estimated to assess how good fit was the modelled regression line to the experimental data. The model fit for each behavioural category and for subject group was excellent (correlation coefficient R was 0.99 for error prosaccades and antisaccades and 0.96 for corrected antisaccades in the healthy control group and 0.99 for error prosaccades and antisaccades and 0.97 for corrected antisaccades in the OCD group). To compare the two simulated regression lines for the patient and control groups the homogeneity of slopes and intercepts regression analysis described in Wuensch (2007) was used. The coefficients (slope and intercept) were extracted and fitted to the experimental 1/RT data (see right plots of Figs. 3A, 3B, and 3C). A comparison of the homogeneity of slopes and intercepts showed that both (controls and patients) fitted error prosaccade lines were statistically different in slope (t_{36} = 5.53305, p = 0.005) and in intercept (t_{36} = 2.9, p = 0.005). A similar comparison of the slopes and intercepts were made for the antisaccades and corrected antisaccades for the controls and patients. The fitted antisaccade lines were not statistically different in slope (t_{36} = 2.10387, p = 0.005) and in intercept (t_{36} = 1.75, p = 0.005). The fitted corrected antisaccade lines were not statistically different in slope (t_{36} = 2.49, p = 0.005) and in intercept (t_{36} = 0.193, p = 0.005).

Symbol	Value		Symbol	Value
	Controls	OCD		
T_h	0.16	0.177	σ	$2\pi/10$
C	0.35		Δx	$2\pi/N$
I_r	1		A	1
I_p	1.5		N	100
μ_1	0.01787	0.0165	β	0.5
σ_1	0.003	0.005	θ	0.5
μ_2	0.0056	0.0047	μ_n	0
σ_2	0.0016	0.002	σ_n	0.05
T	50 ms, unless mentioned otherwise		ntrials	5000

Table 3. Model parameters and values.

Error rates

The experimental error rate was found to be 20.79% for the controls and 47.96% for the patients (Fig. 2B; see also Table 1). In the model an error was considered when the firing activity of the node encoding the reactive input (error prosaccade) crossed a preset threshold level. The model error rate was estimated to be 31.24% for the controls and 41.58% for the patients (Fig 2D; see Table 1), thus qualitatively reproducing the increasing error rate trend reported in OCD patients.

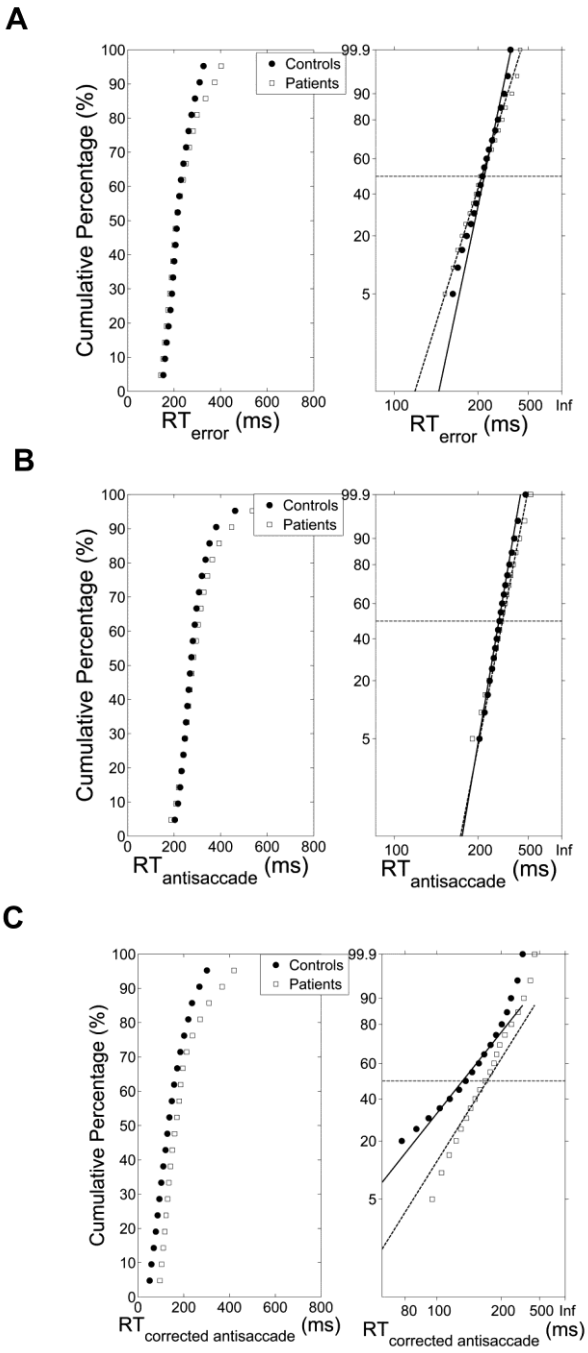
A
Cumulative Percentage (%)
Controls
Patients
RT error (ms)
RT error (ms)
B
Cumulative Percentage (%)
Controls
Patients
RT antisaccade (ms)
RT antisaccade (ms)
C
Cumulative Percentage (%)
Controls
Patients
RT corrected antisaccade (ms)
RT corrected antisaccade (ms)

Fig. 3. (*Left*) Average cumulative RT distribution for controls (white empty circles) and patients (black squares). (*Right*) Reciprobit plots of the average cumulative RT distributions. The x-axis represents 1/RT and it has been reversed so that RTs increase to the right. Instead of 1/RT values the axis is marked with the corresponding RT values. The fitted lines correspond to linear regression on the data of each distribution (controls vs. patients). (A) Error prosaccades. (B) Antisaccades. (C) Corrected antisaccades.

Discussion

What have we learned from this model?

Previously recorded antisaccade performance of healthy and OCD subjects (Damilou et al., 2016) is re-analyzed to show greater variability in mean latency and variance of corrected antisaccades as well as variability in shape of antisaccade and corrected antisaccade latency distributions and increased error rates of OCD patients relative to healthy participants. A well-established neural non-linear accumulator model of antisaccade performance is then employed to uncover the biophysical mechanisms giving rise to these observed OCD deficits. The major finding of this study is that the brains of OCD participants when they performing the antisaccade task are noisier than the brains of healthy controls. This noise is reflected in the rate of accumulation of information (μ and σ) and the threshold level S_T (confidence level required before commitment to a particular course of action). As we can see from Table 3 the value of T_h (threshold level S_T) is higher in the OCD patient case than in healthy control one meaning that the OCD patients are less confident about their decisions than the healthy controls. Their lack of confidence is reflected in their latencies, which are longer than the control ones (see Table 1). Parameters μ_1 and μ_2 (see Table 3 for values) are greater in control condition than in OCD condition meaning that error prosaccades, antisaccades and corrected antisaccades are slower in OCD patients than in healthy controls. Similarly, σ_1 and σ_2 (see Table 3 for values) are smaller in healthy control condition than in patient one, which means that error prosaccade, antisaccade and corrected antisaccade latencies are more variable in OCD patients than in healthy participants. A physiological interpretation of the time constant, τ, and its variability maybe variability of NMDA based rate of evidence integration (Cutsuridis et al., 2007b). Experimental studies have shown that NMDA hypofunction is implicated in neurodegenerative disorders such schizophrenia and OCD (Lewis, 2012).

Another important finding of this study is the absence of a third signal, inhibitory in nature, necessary to prevent the error prosaccade from being expressed when the antisaccade reached the threshold first. Such a third inhibitory signal has been speculated to exist by Noorani and Carpenter (2012, 2013, 2014) in the form

of a "stop-and-restart" mechanism that partially captures the antisaccade performance of healthy participants (see the Cutsuridis (2015, 2017) studies for a critique of Noorani and Carpenter (2014) model limitations). Recent experimental evidence has demonstrated that lateral interactions within SC intermediate segment are more suitable for faithfully accumulating subthreshold signals for saccadic decision- making (Phongphanphanee et al., 2014). Another experimental study by Everling and colleagues (2013) challenges the idea of a third suppressive/inhibitory influence (STOP signal in the Noorani and Carpenter model) of prefrontal cortical areas on reflexive, erroneous prosaccade generation in the antisaccade paradigm. My study has provided quantitative evidence that such a third inhibitory STOP process is not necessary, but instead competition via lateral inhibition in the SC between the neurons encoding the erroneous response (error prosaccade) and neurons encoding the voluntary one (antisaccade) is sufficient to prevent in some trials the error prosaccade from crossing the threshold when the antisaccade has reached it first. My model simulated accurately the latency distributions of the error prosaccades, antisaccades and corrected antisaccades of both healthy controls and OCD patients.

It has been suggested that when data are plotted on the reciprobit plot, then the resulting straight line on the reciprobit plot could be used a diagnostic tool to assess the contribution of different factors influencing the experimental results (Carpenter, 1981). When straight lines swivel by the threshold S_T (Reddi and Carpenter, 2000), then the mean and variances of the lines are unequal. When the lines are parallel and shifted by μ, then the slopes ($1/\sigma$) of the lines are equal, but their latency medians are not (Reddi et al., 2003). When the lines cross, then the slopes are not equal, but their medians are (Nakahara et al., 2006). In my model we observed that when the lines crossed (error prosaccade (Fig. 3A) and antisaccade (Fig. 3B)), then the median values of error prosaccade and antisaccade latencies are not significantly equal. When the lines are parallel and shifted (corrected antisaccades; Fig. 3C), then the median latencies are significantly different.

Comparison with other models

Neurodegenerative disorders such as schizophrenia, bipolar disorder, and major depression share same dimensions of clinical symptoms and same genetic vulnerabilities (American Psychiatric Association, 2013). OCD, however, does not share with them the same clinical and genetic vulnerability factors (American Psychiatric Association, 2013). On the other hand, both OCD and schizophrenia have been associated with dysfunctions of similar cortical and subcortical circuits (Tekin & Cummings, 2002). These dissimilarities are reflected in the antisaccade performances of patients with schizophrenia and those with OCD. Patients with schizophrenia consistently report increased error rate, increased *both* antisaccade and corrected antisaccade latencies, while their erroneous prosaccade ones are not significantly different from those of healthy controls (Karoumi et al., 1998; Smyrnis et al., 2009; Theleritis et al., 2014). On the other end, OCD patients show increas-

es in error rates, increases in latencies of *just* the corrected antisaccades, and significant differences in the shapes of OCD latency distributions of antisaccades and corrected antisaccades compared with those of the healthy controls (Damilou et al., 2016; this study).

The computational study of Cutsuridis and colleagues (2014) showed that the differences in the antisaccade performance of healthy controls and schizophrenia patients is due to a more noisy accumulation of information process (μ and σ) in both frontal (voluntary; antisaccade) and posterior parietal (reactive; erroneous prosaccade) decision centers, but both groups' prior confidence level (decision threshold level S_T) required before commitment to a particular course of action were not affected by disease (schizophrenia). In the present computational study of antisaccade performance of healthy controls and OCD patients, the accumulation of information process (μ and σ) in both frontal (voluntary; antisaccade) and posterior parietal (reactive; erroneous prosaccade) decision centers is still noisy compared to healthy controls, but the OCD patients' confidence level value (decision threshold level S_T) is higher than that of the healthy controls. This means that the OCD patients less confident to respond that the healthy controls. The difference in the confidence level value between schizophrenia (see Table 3 in Cutsuridis et al., 2014) and OCD (Table 3 in this study) participant groups is *maybe* due to the accuracy constraints of the mirror antisaccade task reported in the Cutsuridis and colleagues (2014) study making the schizophrenia patients less confident (more hesitant) to respond, which is reflected in the observed increases in their latencies (compare latency values in Tables 1 in Cutsuridis et al. (2014) study and this one).

Conclusion

Overall, the model showed in a quantitative way why the antisaccade performance of patients with OCD is so poor, that this performance is not due to a deficit in the top-down inhibitory control of the erroneous response as many speculated, but instead it is a product of a neuronal competition via lateral inhibition between the erroneous prosaccade and the antisaccade. The model accurately reproduced the error rates, the median antisaccade, median error prosaccade and median corrected antisaccade latencies as well as the antisaccade, error prosaccade and corrected antisaccade distributions of healthy controls and OCD patients. The model showed that the experimentally observed antisaccade performance deficits of OCD patients are due to: (i) a more noisy accumulation of information by both erroneous and correct decision signals, and (ii) a higher confidence level of the OCD patients. The results presented here illustrate the benefits of tightly integrating psychophysical studies with computational neural modeling, because the two methods complement each other and they may provide together a strong basis for hypothesis generation and theory testing regarding the neural basis of decision making in health and in disease.

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