A Neural Accumulator Model of Antisaccade Performance of Healthy Controls and Obsessive-Compulsive Disorder Patients

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Abstract

Antisaccade performance in obsessive-compulsive disorder (OCD) is related to a dysfunctional network of brain structures including the (pre)frontal and posterior parietal cortices, basal ganglia, and superior colliculus. 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 relative to healthy participants. Then a well-established neural accumulator model of antisaccade performance is employed to uncover the mechanisms giving rise to these observed OCD deficits. The model shows: i) 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 *almost* as confident about their decisions as healthy controls; iii) competition via local lateral inhibition between the correct and erroneous decision processes, and *not* a third top-down STOP signal of the erroneous response, accounts for both the antisaccade performance of healthy controls and OCD patients.

Keywords: Eye movements; superior colliculus; computer model; response inhibition; OCD.

Introduction

In the antisaccade paradigm participats suppress a reflexive saccade (error prosaccade) in favor of a saccade to a position in the opposite hemifield (correct antisaccade) (Hallett, 1978). At least two processes take place during this paradigm: (1) suppression (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, the error rate, the percentage of corrected errors, the amplitude of antisaccades and error prosaccades, and the final eye position of correct responses 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. 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. Carpenter (1981) proposed if the cumulative RT distribution is plotted against 1/RT on reciprobit scale, then the resulting straight line can be used as a diagnostic tool to assess the contribution of different factors influencing the experimental results. In a choice reaction time task such as the antisaccade paradigm, the various choices are represented by different straight lines. If the lines swivel by the threshold T_h, then the mean and variances of the lines are unequal (Reddi and Carpenter, 2000). If the lines are shifted by μ, then the slopes ($1/\sigma$) of the lines are equal, but their latency medians are not (Reddi et al., 2003). If the lines cross, then the slopes are not equal, but their medians are (Nakahara et al., 2006).

In the present study, the Cutsuridis and colleagues (2014) model of antisaccade performance was used and extended into the realm of OCD. 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) neural 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 *almost as confident* about their decisions as healthy controls (i.e. the decision threshold level T_h value is almost the same in healthy controls and 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.

Methods

Experimental data

Participants

Two groups of individuals participated in the study: healthy controls and OCD patients. Both participant groups were extensively described in two previously published studies (Evdokimidis et al., 2002; Damilou et al., 2016).

Antisaccade task

The antisaccade task for the healthy controls and OCD groups was identical to the experimental protocol used in the Evdokimidis and colleagues (2002) study. Stimuli were delivered through a 17-inch computer screen (LCD) located 1m away from the level of their eyes. Their head was immobilized using a chin rest. Subjects were informed about the requirements of the antisaccade task prior to its initiation. A calibration procedure was performed using a sequence of four saccadic eye movements, two to the left and two to the right of a central fixation target at an eccentricity of 10 deg. This process was then repeated with eye movements performed at 5 deg from the fixation point. During each antisaccade trial participants were instructed to fixate on a central fixation stimulus (white cross 0.3^o x 0.3^o of visual angle). After a variable period of 1–2 s, the central stimulus would disappear and a peripheral cue (the same white cross) would appear randomly at one of five positions ($2–10^o$ at 2^o intervals), either on the left or on the right hand side of the central fixation stimulus. The subject was instructed to make a saccade in the opposite direction from the peripheral target. Each subject performed 90 antisaccade trials (5 trials for each cue position) in a randomized order.

Eye movement recordings and analysis

For the control and OCD groups, eye movements were recorded from the right eye using the IRIS SKALAR infrared device. Stimulus presentation and recording of the responses was accomplished with a program written in Turbo Pascal 7.0 for DOS. A 12-bit A/D converter was used for data acquisition (Advantech PC-Lab Card 818L). Eye movement data were sampled at 600 Hz and stored in a PC for off-line data processing. Data pre-processing of all recordings was conducted using an interactive PC program (created using the Test-Point CEC Software). Trials with artifacts (blinks, etc.) in the analysis period or with any type of eye movement in the period of 100ms before the appearance of the peripheral stimulus were excluded from the analysis (Evdokimidis, et al., 2002). In addition, only the trials with response latency within the window of 80–600ms were included in the analysis. Based on these criteria, individuals who performed at least 30 valid antisaccade trials were only retained.

Metrics

The experimental control and patient saccade reaction times (RTs) were divided into three behavioral categories: (1) error prosaccades, (2) antisaccades, and (3) corrected antisaccades. Saccade reaction time (RT) was defined as the time interval from the onset of peripheral stimulus till the time of the first detectable eye movement. Corrected antisaccade RT was as the time between an error prosaccade and the subsequent corrected antisaccade.

Neural model

The model and its mathematical formalism were extensively described in Cutsuridis et al. (2014) study. Briefly, the model was a one-layer neural network of the superior colliculus (SC) with firing rate nodes (Fig. 1A). The total number of nodes in the network was assumed to be 100. Short-range lateral excitation and long distance lateral inhibition was also assumed between all nodes in model. The lateral interaction kernel w_{ij}, which allowed for lateral interactions between model nodes, was a shifted Gaussian, which depended only on the spatial distance between nodes and it was positive for nearby nodes to the node activated by the input and negative for distant nodes (Fig. 1B).

Model inputs were of two types: (1) a reactive input (I_r), which represented the error prosaccade decision signal and it was hypothesized to originate from the posterior parietal cortices (Munoz and Everling, 2004) and (2) a planned input (I_p), which represented the correct antisaccade decision signal and it was originated in the model from the frontal cortical areas (Munoz and Everling, 2004). In the model, each input was integrated in opposite model half according to the following way: if the reactive input activated a node and two of each nearest neighbors on each side in the left model half, then the planned input activated 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 were not equal ($I_p > I_r$).

In the model, the reactive input was presented first at time t = 50 ms, followed by the planned input, which was presented 50 ms later (t = 100 ms). Experimental evidence (Becker, 1989) reported that the difference in the afferent delays of the reactive and planned decision signals (inputs) is close to 50 ms. Both inputs remained active for 600 ms.

Results

As in the Cutsuridis and colleagues (2014) modeling study, to fit the experimental OCD data two model parameters were varied: the integration constant (τ) and the threshold (T_h). In the model, the integration constant was a parameter which indicated how fast or how slow the neuron integrated information. A large value of τ allowed the neuron to integrate information slowly. A small value of τ allowed the neuron to integrate information fast.

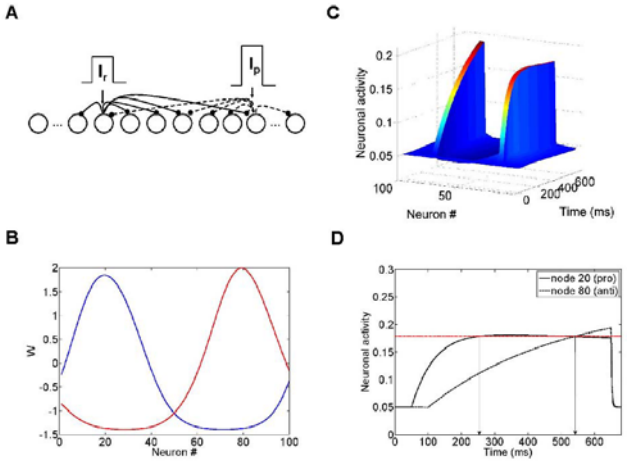

Figure 1. (A) Neural network model (reprinted with permission from Cutsuridis et al. (2014) study). (B) Lateral interaction kernels W for nodes 20 and 80 modelled as a shifted Gaussians (reprinted with permission from Cutsuridis et al. (2014) study). The kernels for nodes 20 and 80 were excitatory for the nearby nodes and inhibitory for the distant ones. (C) Neuronal activities of all nodes in the network as a function of time (ms) (reprinted with permission from Cutsuridis et al. (2014) study). (D) Neuronal activity of nodes 20 and 80 as a function of time (reprinted with permission from Cutsuridis et al. (2014) study). 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 followed by a corrected antisaccade.

Threshold was a model parameter that indicated how confident the model was to make a decision. When the neuronal activity crossed the threshold (see Fig. 1C), then a decision was made (i.e. an eye movement was generated).

In each trial run the integration constant τ values of the two nodes that encoded the erroneous prosaccade and the antisaccade decision signals took values from two normal distributions with different means and standard deviations. The model was then 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.

Table 1: Model parameters

Symbol	Value		Symbol	Value	
	Controls	OCD		Controls	OCD
T_h	0.1767	0.177	σ	2π/10	2π/10
C	0.35	0.35	Δx	2π/N	2π/N
I_r	1	1	A	1	1
I_p	1.5	1.5	N	100	100
μ_1	0.01787	0.0165	β	0.5	0.5
σ_1	0.003	0.005	θ	0.5	0.5
μ_2	0.0056	0.0047	μ_n	0	0
σ_2	0.0016	0.002	σ_n	0.05	0.05
T	50 ms, unless mentioned otherwise		ntrials	5000	5000

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 (see Table 1 for parameter values). In both conditions, the threshold value at which as a decision was reached (parameter T_h in Table 1) was slightly higher in OCD patients than in healthy controls. The parameter values (μ_1, σ_1, μ_2, σ_2 and T_h) that best fitted the experimental data were found via exhaustive search of the parameter value space. The remaining model parameter values were the same as in Cutsuridis et al. (2014) study. 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 (see Table 2). 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).

To compare the experimental and simulated error prosaccade, antisaccade and corrected antisaccade RT distributions for both groups (healthy controls vs OCD patients) I replicated the measures reported in Cutsuridis and colleagues (2014) study. First, I estimated the experimental average cumulative distribution for error prosaccades, antisaccades and corrected antisaccades for both healthy controls and OCD patients by organizing the RTs for each subject (control subject or OCD patient) in ascending order and calculating the percentile values in increments of 5% (at 5, 10,15,20,...,95, 100%). The

calculated percentile values from each subject were then averaged across each subject group (healthy controls or OCD patients) to give the experimental average group percentile values for error prosaccades, antisaccades and corrected antisaccades, which were then plotted in the average cumulative distribution (controls vs. patients) (see left plots of Figs 2A, 2B, and 2C). Ratcliff (1977) showed that the average distribution retains the basic shape characteristics of the individual distributions. Second, I repeated the same procedure for the error prosaccade, antisaccade and corrected antisaccade RTs of the virtual control and OCD subjects. The percentile values were then averaged across trial runs (5000 trial runs) for each subject group (virtual control subject vs virtual OCD patient) 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 (see left plots of Figs 2A, 2B and 2C). A best-fitting regression line was computed for each behavioural category (error prosaccade, antisaccade and corrected antisaccade) in each simulated subject group (simulated controls vs simulated patients). An R correlation coefficient was estimated to assess how good fit was the regression line (simulated data) to the experimental data (open circles and dark squares). 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).

Table 2: Simulated median saccade reaction times, their standard deviations and coefficients of variation (CV) for healthy controls and OCD patients. Bold values in parentheses correspond to experimentally estimated means of medians of saccade RTs, their standard deviations and CVs for controls and patients.

	Median RT in ms		
	Error prosaccade	**Antisaccade**	**Corrected antisaccade**
Controls	214.72 (**211.09, SD: 49.71**)	262.72 (**268.61, SD: 46.76**)	136.97 (**128.84, SD: 53.62**)
OCD Patients	207.84 (**203.81, SD: 53.17**)	277.58 (**275.73, SD: 52.68**)	188.917 (**160.34, SD: 42.55**)
	Coefficient of variation (CV)		
Controls	0.22 (**0.30, SD: 0.21**)	0.19 (**0.24, SD: 0.07**)	0.77 (**0.83, SD: 0.41**)
OCD Patients	0.32 (**0.35, SD: 0.21**)	0.26 (**0.31, SD: 0.12**)	0.77 (**0.54, SD: 0.24**)

Discussion

What have learned from the model

Previously recorded antisaccade performance of healthy and OCD subjects (Damilou et al., 2016) was 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 neural 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 *mostly* in the rate of accumulation of information (μ and σ) and *less on* the threshold level T_h (confidence level required before commitment to a particular course of action). As we can see from Table 1 parameters μ_1 and μ_2 (see Table 1 for values) are greater in control condition than in the 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 1 for values) are smaller in healthy control condition than in the 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 variability in the rate of accumulation of information (variability in parameter τ) is variability of NMDA based rate of evidence integration (Cutsuridis et al., 2007b). Experimental (Lewis, 2012) and computational (Kahramanoglou et al., 2008) studies have shown that NMDA hypofunction is implicated in neurodegenerative disorders such schizophrenia and OCD.

On the other hand, the value of T_h (threshold level) is almost the same in the OCD patient case as in healthy control one meaning that the OCD patients are as confident about their decisions as the healthy controls.

Comparison with other models

An 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 (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 constructive critiques of Noorani and Carpenter (2013, 2014) models). In favor of the major finding of the current study that "competition via local lateral inhibition between the correct and erroneous decision processes, and *not* a third top-down STOP signal of the erroneous response, accounts for both the antisaccade performance of healthy controls and OCD patients" 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.

Reciprobit plot as an insights tool of antisaccade performance

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 (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/ σ) 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). Along these lines we observed from the simulations that when the lines crossed (error prosaccade (right plot of Fig. 2A) and antisaccade (right plot of Fig. 2B)), then the median values of error prosaccade and antisaccade latencies are not significantly equal. When the lines are parallel and shifted (corrected antisaccades; right plot of Fig. 2C), then the median latencies are significantly different.

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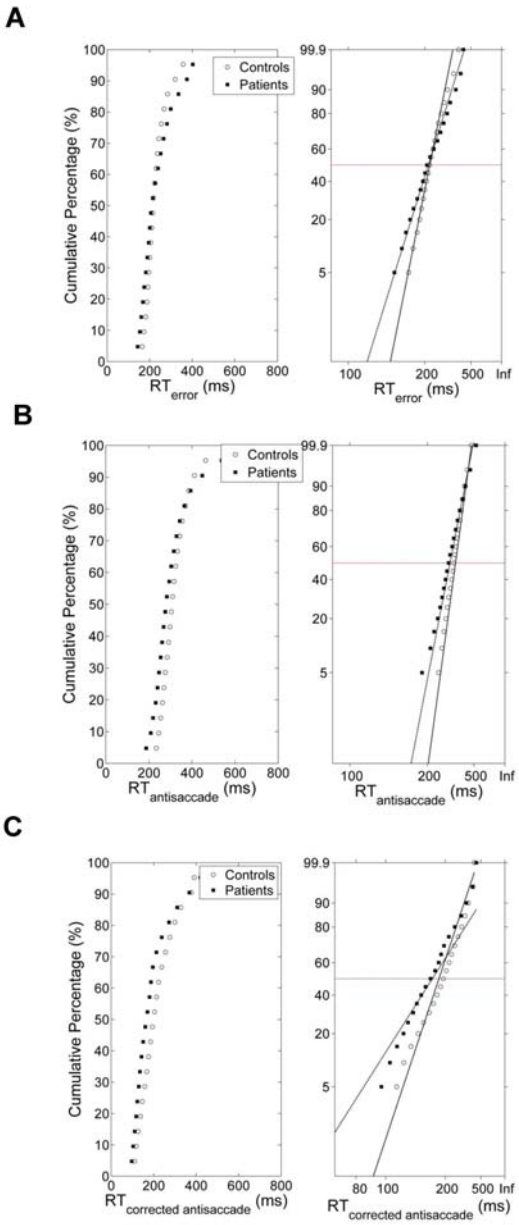

Figure 2. (*Left*) Experimental average cumulative RT distribution for controls (white empty circles) and patients (black squares). (*Right*) Reciprobit plots of the experimental (white empty circles and black squares) and simulated (solid lines) average cumulative RT distributions. The x-axis represents 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 (simulated data) on the experimental data (white circles and black squares) of each distribution (controls vs. patients). (A) Error prosaccades. (B) Antisaccades. (C) Corrected antisaccades.

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