Neural competition via lateral inhibition between decision processes and not a STOP signal accounts for the antisaccade performance in healthy and schizophrenia subjects

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A commentary on

Re-starting a neural race: anti-saccade correction
by Noorani, I., and Carpenter, R. H. S. (2014). Eur. J. Neurosci. 39, 159–164. doi: 10.1111/ejn.12396

Decision making is the process of accumulating evidence about the world and the utility of possible outcomes (Cutsuridis, 2010). A paradigm often used by behavioral neuroscientists to investigate decision processes is the antisaccade paradigm (see Figure 1A; Hallett, 1978). In the antisaccade paradigm subjects are required to suppress an erroneous saccade (error prosaccade) toward a peripheral stimulus and instead make an eye movement to a position in the opposite hemifield (antisaccade). The response repertoire of a subject performing the antisaccade task has been reported to be: (1) the subject makes an erroneous response (i.e., looking toward the peripheral stimulus), (2) the subject makes the antisaccade (i.e., looking in the opposite direction of the peripheral stimulus), and (3) the subject makes an erroneous response followed by a corrected antisaccade (Evdokimidis et al., 2002).

Many computer models of decision making have been advanced. In these models, decision making involves a gradual accumulation of evidence with a variable rate \( r \). When this accumulation process crosses a threshold \( ST \), then a response is generated. Response time (RT) is then the time from the onset of the decision process till when the decision processes crosses \( ST \).

Recently the LATER (Linear Approach to Threshold at Ergodic Rate) model has been extended in the realm of the antisaccade task (see Figure 1B): (Noorani and Carpenter, 2014). The model consisted of three accumulator units racing to threshold: an “anti” unit, a “pro” unit, and a “stop” unit. The “stop” unit prevented the “pro” unit from reaching threshold, thus allowing the “anti” unit to reach a different threshold a little later. The authors hypothesized that the threshold level of the “pro” unit was higher than the “anti” unit’s threshold, reflecting this way the advice given by the experimenters to every subject to avoid errors. How often the “stop” unit canceled the “pro” unit depended on its rate of accumulation (\( \mu \)) and its variance (\( \sigma^2 \)). In the case the “pro” unit reached the threshold first, it restarted the “anti” unit allowing it to reach the threshold and generate the antisaccade response. The model’s performance was contrasted against the performance of five healthy subjects performing the antisaccade task. The model captured most of the response repertoire observed in the antisaccade task, namely the antisaccades and error prosaccades followed by corrected antisaccades, but not the error prosaccades, their corresponding latency distributions, and the error response rate. Despite the model’s successes, the model had several shortcomings.

First, the model is unable to produce just the error prosaccade. This shortcoming is inherent in the model. The authors postulated that if the STOP signal did not prevent the error prosaccade response, then the “pro” unit will always restart the “anti” unit (Noorani and Carpenter, 2014). This means the error prosaccades followed by corrected antisaccades will always be produced. If the “stop” unit did prevent the “pro” unit, then the “anti” unit would not re-start, and an antisaccade response would be generated (Noorani and Carpenter, 2014). In either scenario, just an error prosaccade response cannot be generated. Psychophysical studies of the antisaccade task (Evdokimidis et al., 2002) have reported that subjects make just erroneous prosaccades, but their response frequency is low.

Furthermore, the model implies that the latency of the corrected antisaccade is the result of the linear sum of latencies of the error prosaccade and the antisaccade minus the latency of the STOP activity. This shortcoming is also inherent in the model, because its units are considered linear encoders of the input information. In contrast, all neurons in the brain (units in the model) non-linearly transform the sum of the dendritic outputs before they generate a neuronal response. Dendritic subunits are perhaps the only linear encoders of incoming information (Polsky et al., 2004).

Moreover, the model postulates the existence of a STOP signal, which occasionally stops the error prosaccade.
A mirror antisaccade task. (B) Noorani and Carpenter (2014) model for antisaccades (reprinted with permission from Noorani and Carpenter, 2014). (C) Cutsuridis et al. (2014) neural network model of the superior colliculus (SC) for antisaccades in healthy and schizophrenia subjects (reprinted with permission from Cutsuridis et al., 2014). Neurons are represented as nodes. Short-range lateral excitation and long distance lateral inhibition was assumed between all nodes in the network. The left half of the network represented the left SC, whereas the right half represented the right SC. The left SC was activated by a reactive input $l_r$ (error prosaccade decision signal), whereas the right SC was activated by a planned input $p_p$ (antisaccade decision signal). The strengths of the inputs were not equal.

Overall, competition via lateral inhibition between non-linear accumulator neurons seems to be a better mechanism than the "stop-and-restart" mechanism of Noorani and Carpenter (2014), because it captures the full antisaccade performance (latencies of error prosaccades, antisaccades and corrected antisaccades and error rates) of healthy and diseased subjects performing the antisaccade task. Other parameters that may affect the antisaccade performance are the differential strengths of the erroneous prosaccade and the volitional antisaccade signals, or different baseline and/or threshold levels. Although recent experimental evidence has just demonstrated that lateral interactions within SC intermediate segment are more suitable for faithfully accumulating subthreshold signals for saccadic decision-making (Phongphanphanee et al., 2014), a lot more work needs to be done to conclusively show that a STOP-and-restart mechanism is unnecessary in the decision making process. An experimental study in that direction was recently published by Everling and colleagues and challenges the (prevailing) idea of a suppressive/inhibitory influence (STOP signal in the Noorani and Carpenter model).
of prefrontal cortical areas on reflexive, erroneous prosaccade generation in this paradigm (Everling and Johnston, 2013 for a recent review).

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