Supplement 1

Stop signal reaction time (SSRT) calculation

According to the race model, on each trial, the RT of the STOP and GO process are random variables. If, on a particular stop signal trial, the GO RT is less than the sum of the STOP RT and stop signal delay (SSD), the GO process 'wins', and the response is executed. Likewise, if GO RT is greater than the sum of STOP RT and SSD, the STOP process 'wins', and the response is inhibited. The trials that escape inhibition are from the fastest portion of the no-stop signal RT distribution. Thus, the race model accounts for the finding that the proportion of noncancelled trials increases with increasing SSD and that noncancelled RTs are shorter than no-stop signal RTs.

We estimated SSRT using data from the tracking procedure, which adjusted SSD so that subjects would fail to inhibit eye movements on approximately half of the stop signal trials (1). Under these conditions, the race between STOP and GO is tied (i.e., SSD + SSRT = GO RT), so SSRT can be estimated simply by subtracting mean SSD from mean no-stop signal RT (2). A series of simulations (3) showed that this tracking procedure provided more accurate estimates of SSRT than other methods.

Effects of antipsychotic medication

To examine the effect of medication on countermanding performance, we calculated chlorpromazine (CPZ) equivalent dosages for each subject taking antipsychotic medication (4) and correlated it with no-stop signal and noncancelled RTs, SSRT, slope of the inhibition function, post-error slowing, and post-cancelled slowing. CPZ equivalent dose is based on

antidopaminergic action and does not take into account other neurotransmitter systems, so it may not be ideal for evaluating potential drug effects. Nevertheless it is a standardized and accepted method.

One subject was excluded from this analysis because he was taking paliperidone, a newer atypical antipsychotic medication for which CPZ equivalent dosages have not been published. CPZ equivalent dose was not significantly related to any of the countermanding measures (*r* range: [-0.22, 0.22], *p* range: [0.42, 0.94]).

Interpretation of behavioral differences in schizophrenia in the context of computational models of countermanding performance

In the context of the independent horse race model of countermanding performance (5), which is described in the *Methods* section, our findings of longer SSRT and equal slopes of the inhibition function would suggest that the latency of the stop process is longer in schizophrenia. A variation of the independent race model, the interactive race model, accounts for both behavioral data and interactions between neurons associated with the STOP and GO processes, namely gaze-holding and gaze-shifting neurons in the frontal eye fields (FEF, 6). In this model, on cancelled trials, the STOP process inhibits the GO process and keeps it from reaching the threshold for response execution. The best fitting model accounted for the behavioral data by having a STOP process that became active only slightly before SSRT and exerted potent inhibition on the GO process. In the framework of this model, a longer delay for the STOP process to become active in schizophrenia, rather than weakened inhibition of the STOP process on the GO process, would be consistent with equal slopes of the inhibition functions and relations between no-stop and noncancelled RTs between groups.

Recently, Lo *et al.* (7) proposed a neural network model that considers the role of top-down control of pre-stop signal activity in gaze-holding neurons in countermanding saccades and described impaired inhibitory control when reducing input to neurons in the top-down control module of their network. Further explorations of neurobiologically plausible models to replicate countermanding performance in SZ have the potential to contribute to the understanding neural origins of inhibitory deficits.

Supplemental References

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