A Lack Of Inhibitory Components in the Nogo N2 and the ERN
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Purpose
To see whether the amplitudes of the nogo N2 and ERN reflect activity in the same underlying physiological sources.

Background
The argument has been made that the generator of the nogo N2 is the same as for the ERN, both in the ACC, reflecting some level of conflict processing [2] or inhibitory control [1]. However, fMRI shows enhanced activation in the left MFC associated with errors on nogo trials and right ACC and DLPFC on correct nogo trials [1].

If the nogo N2 and ERN both represent ACC responses to inhibition (or conflict), then individuals with a larger nogo N2 should also have a larger ERN (taken from nogo error trials), i.e., individual differences should be preserved.

Hypotheses:
(1) The nogo N2 should be larger than the go N2.
(2) Individual differences in the N2 should correlate with individual differences in the ERN, especially for the inhibitory aspect alone.
(3) Dipole models of generators of the nogo N2 should differ from those for the go N2 and should match those for the ERN.

Methods
Participants
• 35 18-19 year old male university students

Procedure
• Go-Nogo task. Two letters (e.g., x and y) were presented in an alternating pattern.
• Go trial = when current letter is different from previous letter.
• Nogo lure trial = when current letter is the same as previous letter. (adapted from [1])
• Duration = 100 ms;
• ISI = 1000 ms, increasing after 2 errors and decreasing after 2 correct responses
• # trials: 550 trials with 172 (31%) lures.

ERPs
• 128 site EGI system; 500 points per second; offline filtered 1 to 30 Hz
• ERN was scored 3 ways: against early baseline, as peak-to-peak from preceding positivity, and with the previous positivity partialed out by regression.

Results
The nogo N2 and the go N2 were maximal at FCz (see Figure 1) as was the ERN (not shown). Correlation results reflect all the various ways of measuring the ERN.

1) N2 was significantly more negative for nogo trials at Cz and Pz but nogo and go N2 are not different when P2 amplitudes are partialed out by regression.

2) The ERN correlated significantly with the nogo N2, but only slightly less strongly with the go N2.

ERN did not correlate with the nogo N2 once the go N2 was partialed

3) The dipole modeling for the go N2, nogo N2 (with 2 symmetric pairs modeled at 200-270 ms post-stimulus) and ERN (with 2 symmetric pairs, modeled at -10 to +66 ms post-response) produced good fits with a pair in the MFC accounting for the frontocentral negativity at the scalp (see Figure 2 and Table 1).

Conclusions
In this data set, the N2 is not greater for the nogo trials when the P2 is taken into account.

-Similarly, the significant correlations between the nogo N2 and the ERN are removed when the go N2 is controlled for.

Thus, the nogo N2 and the ERN appear to be similar in topography (less so in dipole sources), but must represent different sources because individual differences are not maintained.

Whatever the ERN and the N2 have in common as functional generators, it is not the response-inhibition factor.

Table 1. Dipole moments for the medial frontal cortex symmetric pair of generators, taken at the FCz maximum.

<table>
<thead>
<tr>
<th></th>
<th>Left</th>
<th>Right</th>
<th>R.V. at maximum</th>
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<tbody>
<tr>
<td>Go N2</td>
<td>21.38 nAmps</td>
<td>5.76 nAmps</td>
<td>9.23%</td>
</tr>
<tr>
<td>Nogo N2</td>
<td>28.14 nAmps</td>
<td>8.14 nAmps</td>
<td>3.94%</td>
</tr>
<tr>
<td>ERN</td>
<td>18.93 nAmps</td>
<td>8.47 nAmps</td>
<td>6.10%</td>
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Figure 2. Scalp topographies and dipole models are similar but not identical.

References

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