

Research Article

ACTION-MONITORING DYSFUNCTION IN OBSESSIVE-COMPULSIVE DISORDER

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Abstract—Evidence suggests that a hyperactive frontal-striatal-thalamic-frontal circuit is associated with the symptoms of obsessive-compulsive disorder (OCD), but there is little agreement about the function of the exaggerated activity. We report electrophysiological evidence suggesting that part of this system monitors events and generates error signals when the events conflict with an individual's internal standards or goals. Nine individuals with OCD and 9 age-, sex-, and education-matched control participants performed a speeded reaction time task. The error-related negativity, an event-related brain potential component that reflects action-monitoring processes, was enhanced in the individuals with OCD. The magnitude of this enhancement correlated with symptom severity. Dipole modeling suggested that the locus of the enhancement corresponded to medial frontal regions, possibly the anterior cingulate cortex.

Obsessive-compulsive disorder (OCD) is an anxiety disorder characterized by two classes of symptoms, obsessions and compulsions. Obsessions are persistent, unwanted thoughts that often concern some feared circumstance. Compulsions are repetitive, sometimes ritualistic behaviors that are often undertaken to reduce anxiety associated with the obsessions (American Psychiatric Association, 1994). Here we report an electrophysiological study exploring the functional and neural basis of OCD.

Our study was motivated by theories and empirical findings regarding the cognitive neuroscience of OCD. Neuroimaging and neurosurgical evidence suggests that excessive activity in the orbitofrontal cortex, anterior cingulate cortex, caudate nucleus, and thalamus contributes to the symptoms of OCD (e.g., Baer et al., 1995; Breiter et al., 1996; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996). A number of investigators have suggested that these structures make up a cortical-striatal-thalamic-cortical circuit (e.g., Insel, 1992; Rapoport, 1991). The connections form a positive feedback loop, so that excessive activity persists. There is, however, no consensus on how this excessive activity produces the symptoms of OCD.

The present study explored the hypothesis that the excessive neural activity involved in OCD symptomatology represents, in part, "hyperactive error signals" (Pitman, 1987; Schwartz, 1997). According to this conception, the brain maintains internal standards (reference points) that represent desired internal and environmental states. A comparator system compares these standards with environmental stimuli, internal stimuli (thoughts, feelings), and actions. When conflicts are detected, the system generates an error signal. The error signal alerts cognitive, motoric, and affective systems of the need to correct the problem. These systems determine whether and how to act, according to the level of the error signal and the motivational significance of the alerting information.

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In individuals with OCD, the error signals are larger and persist longer than in individuals without the disorder. Because of the error signals, an individual with OCD feels that something is wrong and that action is needed to correct the problem. Thus, the error signals contribute to anxiety, doubt, feelings of incompleteness, and compulsive behavior. Consistent with the neural circuitry, the system forms a positive feedback loop: The individual, unable to reduce the error signals, repeats the actions, generating more error signals.

Our study used a component of the event-related brain potential (ERP) to test the part of this theory stating that actions are associated with excessive error-signal activity. The error-related negativity (ERN) is a negative-polarity component of the ERP that begins at about the moment of error onset in reaction time (RT) tasks (see Falkenstein, Hohnsbein, & Hoormann, 1995; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Coles, Meyer, & Donchin, 1995; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN is thought to reflect part of an action-monitoring system: Investigators have argued that it reflects error detection (Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996) or, alternatively, the detection of response conflict (Carter et al., 1998). A general working hypothesis is that the ERN reflects an error signal generated when a comparator process reveals that responses conflict in some manner with a desired state of affairs. Thus, the error signal could reflect error detection per se, conflict detection, or some other evaluative computation.

Gehring et al. (1993) proposed the anterior cingulate as a candidate neural generator of the ERN. Studies supporting this hypothesis have used BESA (Brain Electromagnetic Source Analysis; Scherg, 1990), a technique that models the dipoles (intracranial sources) generating the scalp ERP. These studies found that a midline frontal dipole consistent with an anterior cingulate source accounted well for the scalp topography of the ERN (Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998).

The literature therefore suggests two reasons to predict that individuals with OCD will show excessive ERN activity: the theoretical notion that error signals are hyperactive and the findings of excessive anterior cingulate activity in OCD. Thus, if the ERN reflects part of the system disrupted in OCD, then the ERN should be enhanced in individuals with OCD, the amplitude of the ERN should be correlated with the severity of OCD symptoms, and the ERN activity should show a medial frontal localization. To test these predictions, we examined ERN activity recorded from individuals with OCD and matched control participants during performance of a Stroop task (see MacLeod, 1991).

METHOD

Participants

Eleven individuals with a primary diagnosis of OCD were recruited from the Anxiety Disorders Clinic at the University of Michigan Medical Center. Diagnosis was confirmed using the Structured

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Table 1. *Composition of the participant groups*

Characteristic	Group	
	Obsessive-compulsive disorder	Control
Number of males, females	4, 5	4, 5
Age	39.6 (19.6–58.2)	40.5 (22.0–57.9)
Education (years post high school)	3.7 (0–7)	3.3 (1–5.5)
YBOCS total	14.6 (5–26)	—
BDI score	7.7 (1–18)	2.3 (0–5)
Number taking medication	6 ^a	0

Note. Group means are presented, with ranges in parentheses. BDI = Beck Depression Inventory; YBOCS = Yale-Brown Obsessive-Compulsive Scale.

^aTwo participants were taking fluoxetine hydrochloride, 1 was taking clomipramine hydrochloride, and 3 were taking sertraline hydrochloride.

Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P; First, Spitzer, Gibbon, & Williams, 1995), administered by L.G.N. or J.H. None of the 9 individuals in the final OCD group met criteria for current major depression. Seven had a history of past psychiatric disorder, including major depression, panic disorder with agoraphobia, anorexia nervosa, specific phobia, and alcohol abuse. Two of the 7 met criteria for current dysthymia and social phobia. One of those 2 met criteria for current panic disorder without agoraphobia and generalized anxiety disorder.

Each individual with OCD was matched with a control participant on the basis of sex, age, and educational history. Control participants underwent a SCID interview, and were excluded if they had a history of any psychiatric disorder. All participants were right-handed, fluent English speakers, with normal or corrected vision, including normal color vision. Participants received \$10.00 per hour. The final composition of the two groups is shown in Table 1.

Stimuli

Stimuli consisted of the words “red,” “green,” and “blue” presented on a 15-in. VGA color monitor, in either a red or a green font. Each letter subtended 1° of visual angle. A fixation mark (“+”) appeared 1° below the stimuli.

Psychophysiological Recording

The electroencephalogram (EEG) was recorded from 41 tin electrodes embedded in an elastic cap, with a ground electrode on the forehead. The reference was the left mastoid; an average mastoid reference was derived off-line using right mastoid data. Eye movements were recorded using electrooculogram (EOG) electrodes. Electromyogram (EMG) was recorded from the first dorsal interosseous muscle. Data were digitized at 1000 Hz. EEG and EOG were recorded from DC to 100 Hz. EMG was recorded from 10 to 200 Hz. After EMG rectification, all data were digitally low-pass filtered at 50 Hz and reduced to a sampling rate of 200 Hz. EEG data were corrected for ocular artifacts, as in Gehring et al. (1993). For BESA and for Figure 1, the data were digitally low-pass filtered at 12 Hz.

Procedure

On the day of testing, participants completed the Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988). Individuals with OCD

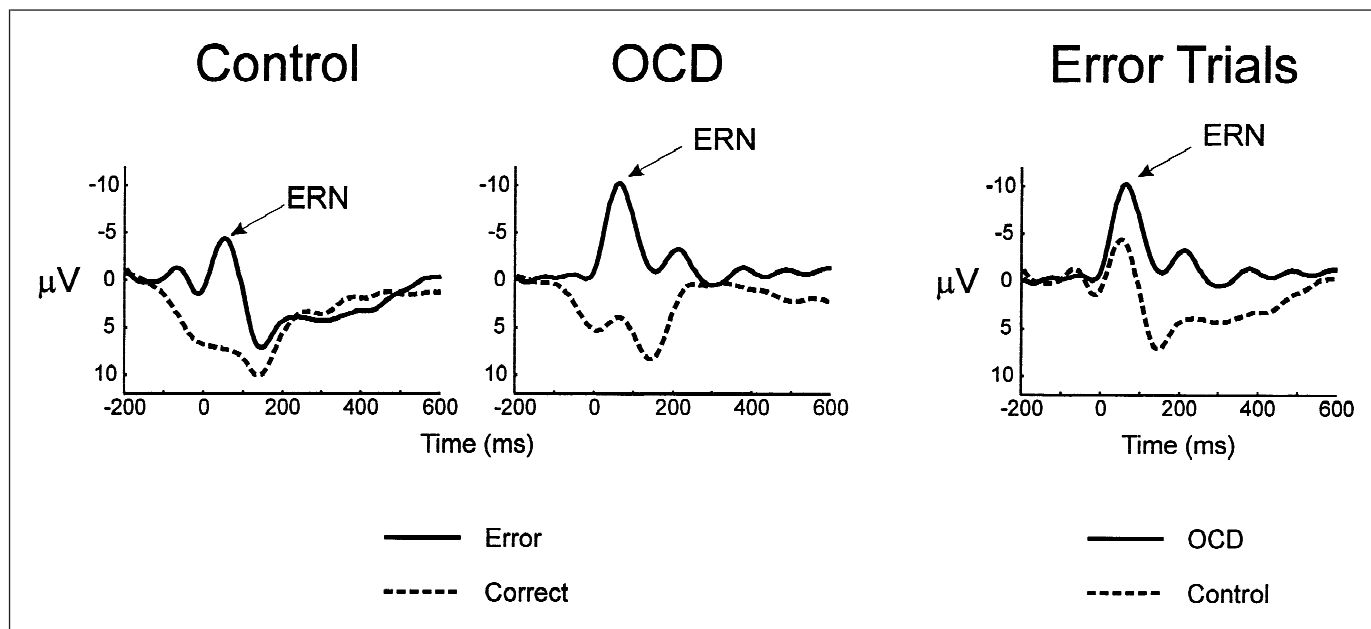


Fig. 1. Response-locked event-related potential waveforms at the Cz electrode location. The left panel compares correct-trial and error-trial waveforms for control participants and for individuals with obsessive-compulsive disorder (OCD). The right panel compares error-trial waveforms for the two groups. Times are plotted relative to the latency of the button-press response. ERN = error-related negativity.

Table 2. Analysis of the error-related negativity

Time post reponse (ms)	Error trials		Difference (error trials – correct trials)	
	Group effect	Correlation with YBOCS	Group effect	Correlation with YBOCS
0–50	–2.76	–.141	–0.761	–.197
50–100	–6.61* (–2.57)	–.592*	–3.48	–.479*
100–150	–8.18* (–2.16)	–.479*	–6.34* (–2.66)	–.535*
150–200	–6.90	–.479*	–5.39	–.592*
200–250	–6.07	–.535*	–3.37	–.535*
250–300	–3.70	–.366	–0.83	–.479*

Note. The group-effect values represent the mean difference (in microvolts) in the Cz electrode waveforms between the obsessive-compulsive disorder (OCD) group and the control group. The values were calculated across the nine OCD-control pairs, for each of the six time windows (relative to the button press). A negative value indicates that the OCD group's waveform was more negative-going than the control group's waveform in that interval. Significant paired *t* values (one-tailed, *df* = 8) are shown in parentheses. The correlation values (computed for the OCD group only) represent Kendall's Tau rank-order correlation between the Yale-Brown Obsessive-Compulsive Scale (YBOCS) total score and mean amplitude calculated over the indicated time interval. A negative correlation indicates that individuals with higher YBOCS scores (more severe symptoms) showed more negativity in that interval than individuals with lower scores. Significant correlations (one tailed; *N* = 9) are indicated by an asterisk.

**p* < .05.

were administered the Yale-Brown Obsessive-Compulsive Scale (YBOCS; Goodman et al., 1989) by J.H. or L.G.N.

Participants responded to each stimulus by pressing a button with one index finger if the color of the word was red and pressing a button with the other index finger if the color was green. Hand of response was counterbalanced. Each word was presented for 0.2 s, at 1.5 to 2 s following the preceding response. There were 48 trials per block. Following 2 practice blocks, each participant completed 24 blocks. The Stroop conditions were congruent (e.g., "red" printed in red), incongruent (e.g., "red" printed in green), and neutral (e.g., "blue" printed in red). Bonuses and verbal feedback encouraged participants to respond quickly, with an error rate of 5 to 10%.

RESULTS

Performance Measures

The groups showed virtually identical accuracy and error RTs. RT and accuracy were analyzed with 2 (group) × 3 (Stroop condition) repeated measures analyses of variance, with Greenhouse-Geisser corrected *p* values. The control and OCD groups did not differ on proportion correct (control: .943; OCD: .945; *F* = 0.02) or on error RT (control: 438 ms; OCD: 434 ms; *F* = 0.06). On correct trials, the OCD group (473 ms) tended to be faster than the control group (514 ms), *F*(1, 8) = 4.81, *p* = .06, *MSE* = 4,708. Stroop condition affected all three measures but did not interact with group.

ERN

To isolate the error effect, we analyzed difference waveforms consisting of the correct-trial waveform subtracted from the error-trial waveform. We also analyzed error-trial waveforms, which were more closely matched in RT and error rate between the groups, thus avoiding performance differences that could affect the ERN (see Gehring et al., 1993, 1995).

Group differences in ERN amplitude

The waveforms from the central electrode site Cz (see Fig. 1) are consistent with our central prediction that the individuals with OCD would show an enhancement in ERN activity relative to the control participants. We partitioned the Cz data following the button press into six 50-ms time windows and computed the mean amplitude in each window, relative to a baseline epoch of 200 to 100 ms prior to the button press (i.e., just prior to EMG onset). As shown in Table 2, the groups differed significantly in the 50- to 100-ms window in error-trial waveforms. In both comparisons (error-trial waveforms and error – correct difference waveforms), the significant difference included the 100- to 150-ms epoch, just after the ERN peak.

ERN effects and symptom severity

We predicted that individuals with more severe OCD symptoms would show greater enhancements in ERN activity. Restricting the analysis to the OCD group, we computed Kendall's Tau rank-order correlations (cf. Baxter et al., 1992) between the ERN amplitude and total YBOCS score for the six time windows (see Table 2). As predicted, significant correlations were evident in both waveforms.¹

Scalp topography and localization

We predicted that the ERN in each group and the group difference would show a medial frontal localization. For localization, we analyzed each group's difference waveform (error trials – correct trials), as well as a group difference waveform consisting of the control group's error-trial waveform subtracted from the OCD group's error-trial waveform.

We mapped the scalp topography of the ERN by computing scalp

1. Other analyses suggested that depressive symptoms did not account for our effects. The correlation between BDI and YBOCS scores was small and not significant, $\tau = .145$ (*N* = 9). Partial correlations (partial Tau; Gibbons, 1985) between ERP measures (50- to 100-ms time window) and YBOCS score (with the effects of BDI score removed) were $-.589$ (*p* < .01, one-tailed; *N* = 9) for error-trial amplitude and $-.466$ (*p* < .025, one-tailed; *N* = 9) for the difference between the error and correct trials.

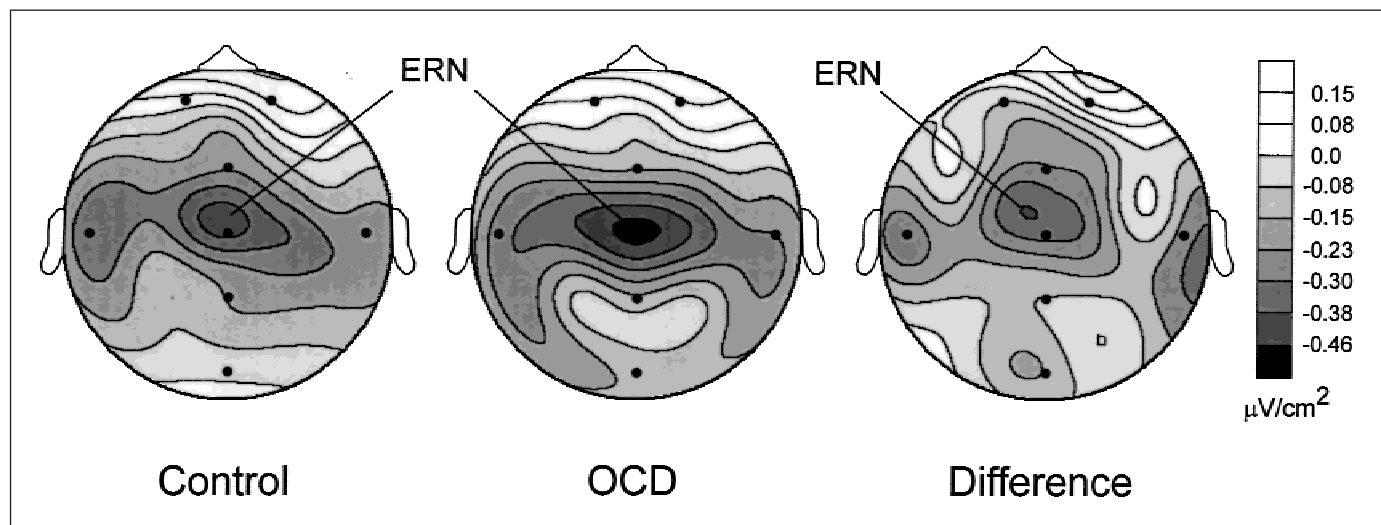


Fig. 2. Scalp current-density maps, representing the scalp topography of the error-related negativity (ERN). The left and middle maps are derived from the control and obsessive-compulsive disorder (OCD) groups' average difference waveforms (error trials – correct trials) and represent the ERN effect within each group. The “Difference” map is derived from the average difference waveform (OCD group – control group) for error trials and represents the group difference in the ERN. The view is of the top of the head with the nose pointing upward. The maps were computed with 43 scalp electrodes; electrode sites Fp1, Fp2, Fz, Cz, Pz, Oz, T7, and T8 are shown as black dots. White regions indicate positive values (regions where current flowed out of the head, or current sources). Shaded regions indicate negative values (regions where current flowed into the head, or current sinks), with darker shading representing larger negative values. Each region between contour lines represents a particular range of current-density values. The ERN is evident as a focal current sink at Cz.

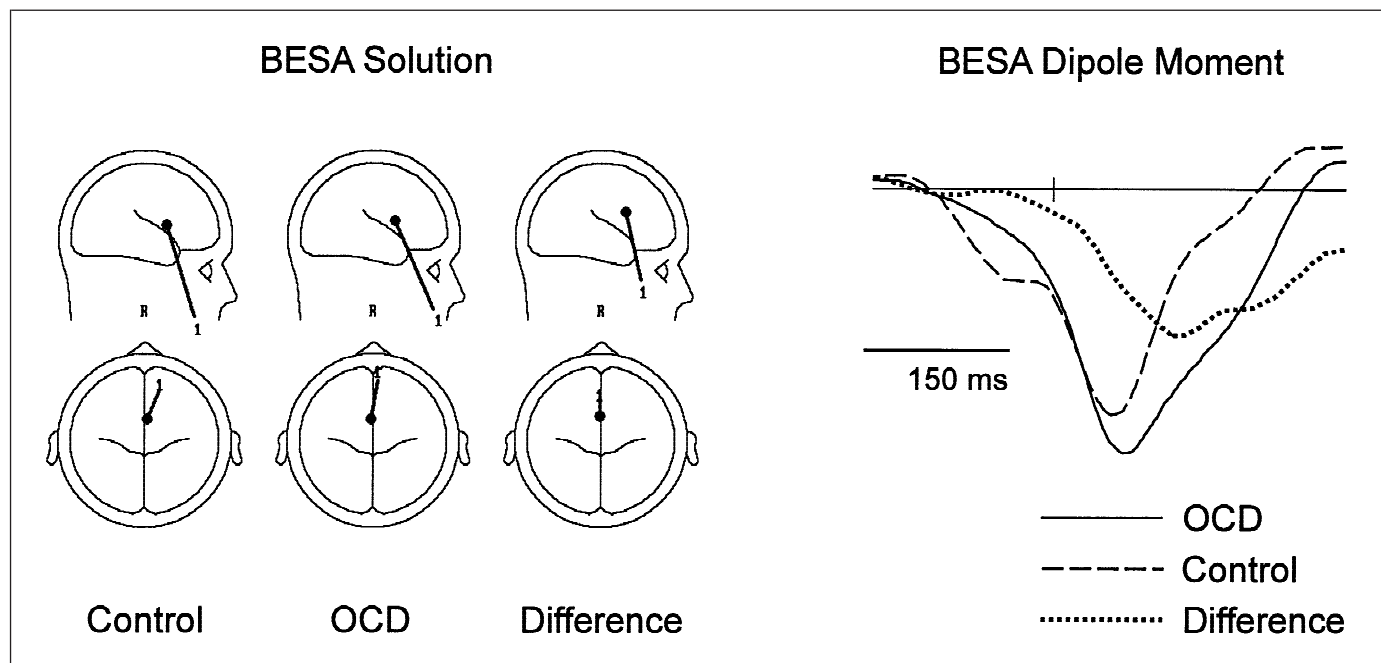


Fig. 3. Single-dipole models of the within- and between-group error-related negativity (ERN) effects. Dipole solutions for the control and obsessive-compulsive disorder (OCD) groups (left panel) were derived from the groups' average difference waveforms (error trials – correct trials) and represent the ERN effect within each group. The “Difference” solution (left panel) was derived from the average error-trial difference waveform (OCD group – control group) and represents the group difference in the ERN. The right panel shows dipole source waveforms (dipole moment as a function of time) for each dipole, plotted in arbitrary units. The vertical line denotes the moment of button press. BESA = Brain Electromagnetic Source Analysis.

current-density maps, which highlight local, radial current flow. (We used the spherical spline interpolation method of Perrin, Pernier, Bertrand, & Echallier, 1989, with $\lambda = .00001$.) Figure 2 shows these maps at the time of each waveform's peak (control: 55 ms; OCD: 65 ms; difference: 130 ms). In each waveform, the ERN appears as a focal maximum at Cz, which is consistent with a medial frontal generator.

We applied the BESA procedure (Scherg, 1990; software version 2.2) to the data from the 41 cap electrodes, fitting single-dipole solutions to the three waveforms during the epoch between the button press and each waveform's peak at Cz. The starting model was a single vertically oriented dipole at the center of the head. For each waveform, BESA converged on a midline frontal source (Fig. 3, left panel). The residual variance (RV) computed over the interval suggested that each model accounted well for the observed data (OCD RV = 2.73%, control RV = 4.43%, difference RV = 7.92%). The source waveforms (Fig. 3, right panel), representing the strength of the dipole (dipole moment) as a function of time, suggest that the ERN was prolonged in the OCD group relative to the control group, with the point of maximal difference following the peak of the ERN itself.

DISCUSSION

Individuals with OCD show a heightened and prolonged electrophysiological response to errors that correlates with the severity of OCD symptoms. The likely medial frontal source for this activity is consistent with a locus in the anterior cingulate cortex (Dehaene et al., 1994; Holroyd et al., 1998). These results support theories linking OCD symptoms to hyperactive error signals in a frontal-striatal-thalamic-frontal circuit (Pitman, 1987; Schwartz, 1997). Our data point to medial frontal action-monitoring activity as one source of the error signals. Other error signals may arise when problems are detected in the environment and in internal states.

There are a number of cognitive explanations for excessive error signals (see Pitman, 1987). One way to understand how the ERN data might be relevant to the symptoms of OCD is to examine the data in the context of these hypotheses. OCD is, of course, a complex disorder, with many symptoms and variants. Our attempt to relate the ERN data and theory to symptomatology must be rudimentary, and it may not apply to all types of OCD.

The classification of events can cause excessive error signals, either through dysfunctions in the comparator mechanism or through conflict between the reference points of several comparators (Pitman, 1987; see also Reed, 1985). A comparator dysfunction could explain the tendency of individuals with OCD to feel that something is wrong when the situation seems satisfactory to an outside observer (e.g., when the individual continues to check the stove after successfully turning it off). Such a dysfunction could therefore cause seemingly correct actions to elicit error signals. We did not find a significant group difference in ERN-like activity on correct trials. Note, however, that a comparator dysfunction does not necessarily predict such an effect in a Stroop task. Rather, the dysfunction could be focused on symptom-relevant events (e.g., pertaining to cleanliness), which would then result in domain-specific symptoms (e.g., washing). Future studies should examine symptom-relevant events. The comparator-deficit hypothesis predicts that ERPs recorded when an individual with checking compulsions turns off a stove would show unusual amounts of ERN activity.

Alternatively, the excessive activity may result from problems in a separate alerting system or in systems that modulate the error signal (e.g., attention; Pitman, 1987). Our observation of enhanced ERN responses to errors in a non-symptom-relevant task would be consistent with a generalized enhancement in such a system. Domain specificity could then arise in systems that evaluate the error signals and generate further compensatory behavior.

An example of a theory that postulates such a source for domain specificity is the conflict-displacement model of OCD (Holland, 1974; Pitman, 1987, 1989). In this account, compulsive behaviors are analogous to the displacement behaviors of animals: purposeless, repetitive actions such as grooming and burying performed by animals in states of conflict. These may be primitive, adaptive behaviors (reducing risks of disease and danger) that are activated inappropriately in OCD (Pitman, 1989). As Rapoport (1991) has suggested, this ethological framework is consistent with neural circuitry in which error signals that signify the need for action arise from the cingulate cortex (and possibly orbitofrontal cortex; see Schwartz, 1997), triggering basal ganglia systems responsible for particular learned or genetically programmed behaviors. Although the conflict-displacement account has been criticized (Jakes, 1996), our data suggest that the general principle is worthy of further study.

The ERN provides a unique window on medial frontal error processing. Our study has shown how it can help to build a comprehensive theory of OCD, by illuminating the links between neuroscience, cognitive theory, and OCD symptomatology.

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