where bison were excluded, there was a negative relation between abundance of forbs and fire frequency. In contrast, there was a positive relation between fire frequency and forb abundance on watersheds that were grazed by bison (Fig. 1). Thus, grazing increased the abundance of forbs under conditions that would otherwise promote dominance by C_4 grasses and lower species diversity.

Although burning is essential to maintain tallgrass prairie (*15*), fire alone is not a sufficient management solution for restoring prairie biodiversity as some have proposed (*16*). More frequent fires are now needed to resist invasion by exotics and woody species in remaining grassland fragments (*16*), but as shown here frequent burning dramatically increases the dominance of C_4 grasses and reduces plant species diversity (*17*). Whereas fire is used as a conservation tool throughout much of the tallgrass region, the use of grazing by bison or cattle as a management tool for maintaining species diversity is less common (*18*). Yet herbivores such as bison historically served as keystone species in tallgrass ecosystems because they reduced the competitive dominance of the C_4 grasses, increased habitat heterogeneity, and increased species diversity (*19*).

One consequence of anthropogenically driven global change has been the extinction or dramatic reduction in populations of keystone species (*20*). The role that keystone species play in community structure and ecosystem functioning is now widely recognized (*21*). In some systems, loss of a keystone species may decouple the critical interplay between trophic interactions and community structure (*20*). Our research demonstrates that by adding or maintaining top-down forces such as grazing, at least in ecosystems like grasslands that were affected historically by keystone herbivores (*22*), diversity in native vegetation can be retained under conditions that would otherwise lead to a decline in species richness.

REFERENCES AND NOTES

1. T. R. Seastedt and A. K. Knapp, *Am. Nat*. **141**, 621 (1993); J. M. Blair, *Ecology* **78**, 2359 (1997).
2. S. L. Collins, *Ecology* **68**, 1243 (1987).
3. A. K. Knapp and T. R. Seastedt, in *Grassland Dynamics: Long-term Ecological Research in Tallgrass Prairie*, A. K. Knapp, J. M. Briggs, D. C. Hartnett, S. L. Collins, Eds. (Oxford Univ. Press, London, in press).
4. D. C. Hartnett and P. A. Fay, in (*3*).
5. S. J. McNaughton, *Ecol. Appl*. **3**, 17 (1993); F. B. Samson and F. L. Knopf, *BioScience* **44**, 18 (1994).
6. J. P. Grime, *Nature* **242**, 344 (1973); D. Tilman, *Resource Competition and Community Structure* (Princeton Univ. Press, Princeton, NJ, 1982); A. DiTommaso and L. W. Aarrsen, *Vegetatio* **84**, 9 (1989).
7. This rate of N addition was chosen to add sufficient N to allow for a detectable plant response in a short period of time [J. D. Aber *et al*., *Ecol. Appl*. **3**, 156 (1993)]. Studies have shown that supplemental N in this range was sufficient to affect productivity and community structure [T. R. Seastedt, J. M. Briggs, D. J. Gibson, *Oecologia* **87**, 72 (1991); J. W. Silvertown, *J. Appl. Ecol*. **17**, 491 (1980); D. A. Wedin and D. Tilman, *Science* **274**, 1720 (1996)]. These accelerated responses are indicative of long-term cumulative effects of chronic elevated N deposition that occur in grasslands and other regions [P. M. Vitousek *et al*., *Ecol. Appl*. **7**, 737 (1997)].
8. Spatially integrated photon flux density (0.4 to 0.7 μm; reported in micromoles per square meter per second) was measured with a 1-m ceptometer (Decagon) in four locations in each plot in September. Measurements were made at midday under clear sky conditions. Mowing occurred in late June of each year.
9. S. L. Collins and S. M. Glenn, *Ecology* **72**, 654 (1991); C. L. Turner and A. K. Knapp, *ibid.* **77**, 1738 (1996).
10. L. C. Hulbert, *Prairie Scout* **5**, 63 (1985).
11. In 1987, 30 bison were reintroduced to Konza Prairie, and the herd is now maintained at 200 individuals. Bison have unrestricted access to 10 watersheds subjected to different frequencies of late spring prescribed fire in a 1012-ha portion of prairie. This animal density was selected so that ~25% of aboveground primary production is consumed annually.
12. J. M. Briggs and A. K. Knapp, *Am. J. Bot*. **82**, 1024 (1995).
13. N. T. Hobbs, D. S. Schimel, C. E. Owensby, D. S. Ojima, *Ecology* **72**, 1374 (1991); S. J. McNaughton, F. F. Banyikwa, M. M. McNaughton, *Science* **278**, 1798 (1997).
14. D. J. Gibson and L. C. Hulbert, *Vegetatio* **72**, 175 (1987); S. L. Collins and E. M. Steinauer, in (*3*).
15. R. Daubenmire, *Adv. Ecol. Res*. **5**, 209 (1968); R. J. Vogl, in *Fire and Ecosystems*, T. T. Kozlowski and C. E. Ahlgren, Eds. (Academic Press, New York, 1974), pp. 139–194.
16. M. K. Leach and T. J. Givnish, *Science* **273**, 1555 (1996).
17. S. L. Collins and S. M. Barber, *Vegetatio* **64**, 87 (1985); S. L. Collins, S. M. Glenn, D. J. Gibson, *Ecology* **76**, 486 (1995).
18. F. Berendse, M. J. M. Oomes, H. J. Altena, W. T. Elberse, *Biol. Conserv*. **62**, 59 (1992); E. M. Steinauer and S. L. Collins, in *Prairie Conservation: Preserving North America's Most Endangered Ecosystem*, F. B. Sampson and F. L. Knopf, Eds. (Island Press, Washington, DC, 1996), pp. 39–52. The functional equivalency of bison and cattle grazing in North American grasslands remains uncertain [G. E. Plumb and J. L. Dodd, *Ecol. Appl*. **3**, 631 (1993); (*4*)].
19. S. L. Collins and T. R. Benning, in *Biodiversity: A Biology of Numbers and Difference*, K. J. Gaston, Ed. (Blackwell, London, 1996), pp. 253–280.
20. M. E. Power *et al*., *BioScience* **46**, 609 (1996); D. A. Wardle, O. Zachrisson, G. Hörnberg, C. Gallet, *Science* **277**, 1296 (1997); D. Tilman *et al*., *ibid.*, p. 1300; D. U. Hooper and P. M. Vitousek, *ibid.*, p. 1302.
21. E.-D. Schulze and H. A. Mooney, Eds., *Biodiversity and Ecosystem Function* (Springer-Verlag, Berlin, 1993); C. G. Jones and J. H. Lawton, Eds., *Linking Species and Ecosystems* (Chapman & Hall, New York, 1995); F. S. Chapin *et al*., *Science* **277**, 500 (1997).
22. D. G. Milchunas, O. E. Sala, W. K. Lauenroth, *Am. Nat*. **132**, 87 (1988).

23. We thank J. H. Brown, L. Gough, J. H. Lawton, S. J. McNaughton, G. Shaver, D. Tilman, and P. M. Vitousek and four anonymous reviewers for comments on an earlier version of the manuscript. Supported by the NSF Long-Term Ecological Research Program, Kansas State University, and the Kansas Agricultural Experiment Station. Opinions expressed in this report do not necessarily reflect an endorsement by the National Science Foundation.

12 December 1997; accepted 16 March 1998

Anterior Cingulate Cortex, Error Detection, and the Online Monitoring of Performance

Cameron S. Carter,* Todd S. Braver, Deanna M. Barch, Matthew M. Botvinick, Douglas Noll, Jonathan D. Cohen

An unresolved question in neuroscience and psychology is how the brain monitors performance to regulate behavior. It has been proposed that the anterior cingulate cortex (ACC), on the medial surface of the frontal lobe, contributes to performance monitoring by detecting errors. In this study, event-related functional magnetic resonance imaging was used to examine ACC function. Results confirm that this region shows activity during erroneous responses. However, activity was also observed in the same region during correct responses under conditions of increased response competition. This suggests that the ACC detects conditions under which errors are likely to occur rather than errors themselves.

It has been proposed that the ACC plays a prominent role in the executive control of cognition (*1*). This hypothesis is based, in part, on functional neuroimaging studies that show ACC activity during tasks that engage selective attention, working memory, language generation, and controlled information processing (*2*). Disturbances in this brain region have been reported in disorders associated with cognitive impairment, including schizophrenia and depression (*3*). This account of ACC function is consistent with the rich anatomical connectivity of this region with association, limbic, and motor cortices (*4*). However, it is lacking in detail regarding the precise contribution of the ACC to cognitive control.

To date, the most explicit hypothesis regarding ACC function comes from event-related brain potential (ERP) studies during speeded response tasks. These studies have reported an error-related negativity (ERN), peaking 100 to 150 ms after

a person shows electromyographic evidence of initiating an incorrect response. Dipole modeling suggests that the ERN has a medial frontal generator, possibly the ACC (*5*). These and other characteristics of the ERN have led to the hypothesis that the ACC is involved in monitoring and compensating for errors. Specifically it has been proposed that this involves a comparator process in which a representation of the intended, correct response is compared to a representation of the actual response (*5*, *6*).

We propose that rather than implementing a comparator process, the ACC monitors competition between processes that conflict during task performance. For example, response competition arises when a task elicits a prepotent but inappropriate response tendency (manifested as activity in the incorrect response channel) that must be overcome to perform correctly. These conditions are also more likely to elicit incorrect responses, possibly accounting for the relationship of ACC activity to errors. However, our hypothesis predicts that response competition will activate the ACC even when a correct response is made. The present study employed event-related functional magnetic resonance imaging (fMRI) to accomplish two goals. First, we sought to test the hypothesis, suggested by ERN studies, that the ACC shows error-related activity. More important, we also sought to test our alternative hypothesis regarding the functional significance of the ERN and the performance-monitoring function of the ACC.

Thirteen people underwent fMRI (*7*) while performing variants of the Continuous Performance Test (AX-CPT) (*8*) that were designed both to increase error rates and to manipulate response competition (Fig. 1) (*9*). We observed a transient increase in ACC activity (*10*) occurring during incorrect responses (*11*) (Fig. 2). However, as predicted by our hypothesis, greater ACC activity was also seen during correct responses, under conditions that elicited greater response competition (*12*). We interpret these results as suggesting that the contribution of the ACC to performance monitoring may be the detection of response competition rather than detection of errors per se. Sensitivity to response competition should also produce error-related effects, because error trials are often likely to be those in which activity in the incorrect response channel competes with, and prevails over, activity in the correct response channel.

To determine whether error and competition effects were specific to the ACC, we used two additional analyses. First, we examined error- and competition-related effects in three other regions of interest (ROIs) previously shown to be activated by stimulus degradation (two right inferior frontal regions and a right striatal region) (*8*). We observed error-related activity in one right frontal region (BA 44/45), but neither this nor the two other ROIs showed competition effects (*13*). Next, we examined error and competition effects in an exploratory analysis of all brain regions scanned (*14*). Three regions in addition to the ACC showed significant transient increases in activity associated with errors. These were the right (BA 9) and left (BA 46/9) dorsolateral prefrontal cortex and the left premotor cortex (BA 6). However, none showed significant effects of response competition.

Our reconceptualization of ACC activity as being related to response competition, rather than errors per se, has a number of important implications. First, it links the wealth of literature concerning the role of this region in higher level cognition [including the hypothesis that it is involved with late selection or "attention to action" (*1*)] with the ERP literature suggesting that it is responsive to errors. In particular, it reconciles the observation that reliable ACC activation is observed in some tasks that are associated with low error rates, such as the Stroop task and verbal fluency (*15*, *16*). Second, it is computationally parsimonious. A

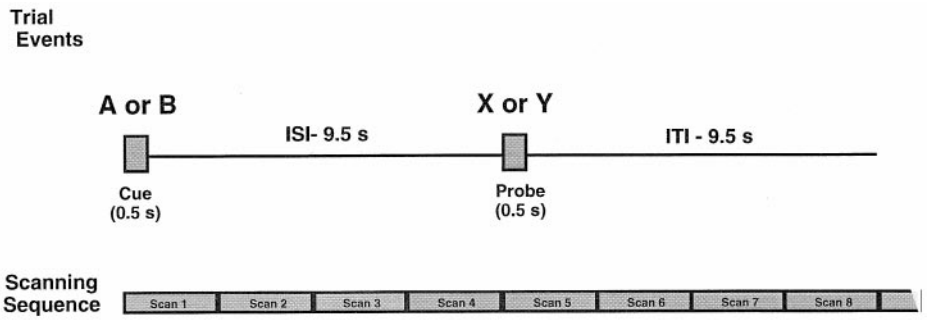

Fig. 1. The AX-CPT task. Stimuli were presented centrally on a visual display projected into the scanner [duration of cue and probe, 0.5 s; duration of interstimulus interval (ISI), 9.5 s; and duration of intertrial interval (ITI), 9.5 s]. Trials occurred as sequences of cue-probe pairs. Stimuli were either degraded or nondegraded letters, presented as cue-probe pairs. Eight time-locked scans were acquired during each 20-s trial, one every 2.5 s, and included sufficient time for the MRI signal to decay to baseline after transient response-related activity.

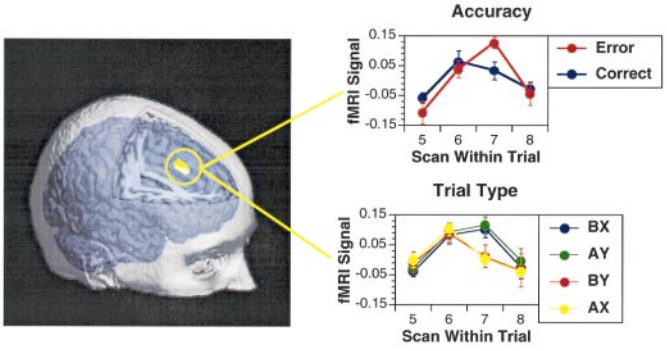

Fig. 2. The location of the region of the ACC (BA 24/32) and its associated effects. On the left, the functional image from the confirmatory analysis of the 39-voxel ACC ROI (yellow) is rendered onto a three-dimensional structural MRI image for anatomic visualization. On the right, the temporal dynamics of activity in this region are shown for both the error and the trial-type effects, plotted as percent change from the mean MRI signal for each condition. Error bars indicate the standard error of the mean. The error effect (observed as an error × scan interaction during the degraded condition) occurred during the time of response as a transient increase in activity on incorrect trials. The competition effect (observed as a cue × probe × scan interaction) is shown only for correct trials and also occurred during the time of response. A transient increase in activity was found for trials with high response competition (AY and BX) relative to trials with low competition (AX and BY).

C. S. Carter, T. S. Braver, D. M. Barch, M. M. Botvinick, Western Psychiatric Institute and Clinic, School of Medicine, University of Pittsburgh, 3811 O'Hara Street, Pittsburgh, PA 15213, USA.
D. Noll, Magnetic Resonance Research Center, Presbyterian University Hospital, B-804, University of Pittsburgh Health System, 200 Lothrop Street, Pittsburgh, PA 15213, USA.
J. D. Cohen, Western Psychiatric Institute and Clinic, School of Medicine, University of Pittsburgh, 3811 O'Hara Street, Pittsburgh, PA 15213, USA, and Department of Psychology, Carnegie Mellon University, 5000 Forbes Avenue, Pittsburgh, PA 15213, USA.

*To whom correspondence should be addressed. E-mail: cscarter+@pitt.edu

comparator function requires the ACC to have access to representations of intended states (for example, the correct action) as well as the outcome of processing, whereas detection of competition requires only information about the state of the response system.

This study focused on ACC activity associated with response competition. The question remains open as to whether the ACC is responsive to competition only at this level or is also responsive to competition earlier in processing. Whichever is correct, the present findings demonstrate how error-related activity can occur without the need for a comparator and how such activity might represent one instance of a more general performance-monitoring function of the ACC.

REFERENCES AND NOTES

1. M. I. Posner and S. Dahaene, *Trends Neurosci.* **17**, 75 (1994); M. D'Esposito *et al.*, *Nature* **378**, 279 (1995).
2. R. Cabeza and L. Nyburg, *J. Cogn. Neurosci*. **9**, 1 (1997).
3. C. S. Carter, M. Mintun, T. Nichols, J. D. Cohen, *Am. J. Psychiatry* **154,** 1670 (1997); C. J. Bench *et al.*, *Psychol. Med.* **22**, 607 (1992).
4. B. Vogt, D. M. Finch, C. R. Olsen. *Cereb. Cortex* **2**, 435 (1992); O. Devinsky, M. J. Morrel, B.Vogt, *Brain* **118**, 279 (1995).
5. W. J. Ghering, M. G. Coles, D. E. Meyer, E. Donchin, *Psychophysiology* **27** (4A suppl.), S34 (1990); M. J. Falkenstein, J. Hohnsbein, J. Hoorman, L. Blanke, *Electroencephalogr. Clin. Neurophysiol.* **78**, 447 (1991); W. J. Ghering, B. Goss, M. G. H. Coles, D. E. Meyer, E. Donchin, *Psychol. Sci*. **4**, 385 (1993); S. Dehaene, M. I. Posner, D. M. Tucker, *ibid.* **5**, 303 (1994).
6. M. Falkenstein, J. Hohnsbein, J. Hoorman, in *Perspectives of Event Related Potentials Research* (EEG Suppl. 44), G. Karmos, M. Molnar, V. Csepe, I. Czigler, J. E. Desmedt, Eds. (Elsevier Science BV, Amsterdam, 1995), pp. 287–296; M. G. H. Coles, M. K. Sheffers, L. Fourneir, *Acta Psychol.* **90**, 129 (1995).
7. Images were acquired with a 1.5-T GE Signa whole-body scanner with a standard head coil. Sixteen axial slices (with 3.75 mm^3 voxels) were obtained parallel to the AC-PC line. Functional scans were obtained with a two-shot T2*-weighted spiral scan [D. Noll, J. D. Cohen, C. H. Meyer, W. Schneider, *J. Magn. Reson. Imaging* **5**, 49 (1995)]. The pulse sequence was as follows: relaxation time, 1250 ms; echo time, 35 ms; flip angle, 60°; and field of view, 24 cm. Structural images were obtained with a standard T1-weighted pulse sequence. Images for all participants were coregistered to a common reference structural MRI scan by means of a 12-parameter automatic algorithm [R. P. Woods, S. R. Cherry, J. C. Mazziotta, *J. Comput. Assisted Tomogr.* **16**, 620 (1992)]. Images were then smoothed with an 8-mm, full width at half maximum, three-dimensional Gaussian filter to accommodate individual differences in anatomy. Finally, data were pooled across participants to increase the signal-to-noise ratio [T. S. Braver *et al.*, *NeuroImage* **5**, 49 (1997)].
8. D. M. Barch *et al.*, *Neuropsychologia* **35**, 1373 (1997).
9. The AX-CPT is a modified Continuous Performance Test in which sequences of single letters are presented as cue-probe pairs (Fig. 1). During the task, participants were instructed to make a target response whenever the probe was an X preceded by a cue that was an A and to make a nontarget response to all other stimuli. Target (AX) trials occurred with 70% frequency, and each of the three nontarget trial types (AY, BX, and BY) occurred with 10% frequency. This frequency manipulation was introduced to produce higher levels of response competition in BX trials (non-A cue followed by X) and AY trials (A followed by non-X probe) than in AX and BY trials (non-A cue followed by non-X probe). In BX trials, response competition occurs because the correct (nontarget) response conflicts with the tendency to give a target response to an X probe (the correct response 87.5% of the time). In AY trials, response competition occurs because participants are primed to give a target response after an A (correct 87.5% of the time) and must override this tendency to give the correct, nontarget response. AX and BY trials involve no such conflicts and thus elicit less response competition. These predictions regarding response competition have been validated in previous empirical work and arose from an existing computational model of the AX-CPT [J. D. Cohen, T. S. Braver, R. O'Reilly, *Philos. Trans. R. Soc. London Ser. B* **351**, 1515 (1996)]. Error rates in the present study were also consistent with this formulation, being increased for BX (mean 6.2%, SE 3.6%) and AY (mean 6.5%, SE 5.1%) trial types, as compared with AX (mean 1.5%, SE 0.8%) and BY (mean 1.9%, SE 2.0%) trial types. Participants also performed a degraded stimulus version of the task. In the degraded condition, 90% of the pixels were randomly removed from the stimuli. In a previous study (*8*), this manipulation was found to reliably increase error rates and to activate the ACC (however, because event-related methods were not used, it was not possible to specifically examine error-related activity). Participant performance in the current study confirmed that errors were greatly increased under stimulus degradation [baseline: mean = 2.5%, SE = 0.6%; degraded: mean = 29.8%, SE = 2.8%; t (12) = 10.5, $P < 0.001$]. However, degraded performance remained significantly above chance [mean d' = 1.41, SE = 0.13; t (12) = 11.2, $P < 0.001$]. Trials in both conditions lasted for 20 s, and scan acquisition occurred in an event-related manner (see Fig. 1). Each condition had five blocks of 10 trials, with the order counterbalanced across participants. Under both conditions, participants were given standard instructions to respond both quickly and accurately. Participants provided written informed consent in accordance with the Institutional Review Board at the University of Pittsburgh.
10. Data were analyzed with a confirmatory ROI approach. Current fMRI data were registered to the same reference brain used in our previous study (*8*), and a 39-voxel ACC region identified in that study was used to precisely define the ROI (Talairach coordinates 4, 25, and 43). The confirmatory analysis for each ROI was performed as follows. We conducted analyses of variance (ANOVAs) for each voxel within the ROI to determine how many showed significant predicted effects ($P < 0.05$), using the Huynh-Feldt correction for nonindependence of repeated measures. We then determined the likelihood of this number of voxels showing a significant effect by chance alone. This was achieved through Monte Carlo simulations (10,000 iterations), in which 13 random data sets were generated (corresponding to the 13 participants in the study). The smoothness of the empirical data was estimated to be 10.46 mm (full width at half maximum) [S. D. Forman *et al.*, *Magn. Reson. Med.* **33**, 636 (1995)], and a smoothing kernel of these dimensions was applied to the simulated data. Then ROI determination and assessment of statistical significance were performed in the same manner as for the empirical data. All P values reported for the confirmatory analyses were obtained from these simulations.
11. A task (degraded versus nondegraded) × scan (1-8) interaction revealed 12 of 39 contiguous voxels within the ACC ROI that were significant ($P < 0.004$). The main effect of task was not significant (0 out of 39 voxels). These results precisely replicated previous findings with this task (*8*). The error-by-scan interaction, pooled across degraded and nondegraded conditions, was highly significant (23 of 39 voxels, $P < 0.0001$). Because the task × scan analysis demonstrated condition-related effects on ACC activity, we also performed the error analysis using only the degraded condition (although this reduced power in the analysis because half of the images were removed). This analysis revealed that 11 out of 39 voxels were significant for the error effect (error × scan interaction, $P < 0.008$). Similarly, when we considered only AX trials, in the degraded condition 11 of 39 voxels showed the error effect ($P < 0.008$). Activity was found to increase during the period of probe response, when the 5-s hemodynamic lag that underlies the fMRI signal is taken into account [K. K. Kwong *et al.*, *Proc. Natl. Acad. Sci.* **89**, 5675 (1992)].
12. Only the baseline AX-CPT was used for this analysis, as degradation is likely to add additional variability and perhaps obscure the effects of trial type. The analysis revealed that 11 out of 39 voxels were significant for the competition effect (cue × probe × scan interaction, $P < 0.008$). Both error and trial-type effects occurred within the exact same region of the ACC, as 10 of 39 pixels were significant for both effects ($P < 0.0001$).
13. The additional ROIs were taken from our earlier study (*8*), as they had also shown a significant task × scan within-trial interaction. In the current study, 8 out of 21 voxels ($P < 0.004$) in BA 45/47 and 13 out of 21 ($P < 0.0001$) voxels in BA 44/45 showed a significant task × scan interaction, again replicating previous results. The effect did not replicate in the striatal ROI (none of the 10 voxels were significant). Five out of 21 voxels in BA 44/45 showed a significant error by scan interaction ($P < 0.03$), but none showed a trial-type effect. None of 21 voxels in BA 45/47 showed a significant error effect, and 3 showed a trial-type effect ($P > 0.05$)
14. This analysis used voxel-by-voxel ANOVAs, with accuracy- and response-related scan-within trial (scans 5 through 8) as factors. The effects of response competition were also examined by testing for significant cue × probe × scan interactions. Statistical maps of F ratios for each voxel were calculated, and the threshold for significance was set with the use of a cluster size algorithm (*11*), which takes account of the spatial extent of activation to correct for multiple comparisons. A cluster size of eight contiguous voxels with alpha set for each voxel at 0.005 was chosen, corresponding to false positive rate for the entiere image of 0.005.
15. J. V. Pardo, J. Pardo, W. Janer, M. E. Raichle, *Proc. Natl. Acad. Sci. U.S.A.* **87**, 256 (1990); C. S. Carter, M. Mintun, J. D. Cohen, *NeuroImage* **2**, 264 (1995); C. D. Frith, K. J. Friston, P. F. Liddle, R. J. S. Frackowiak, *Neuropsychologia* **29**, 1137 (1991).
16. In preliminary computational modeling work by our group [M. Botvinick, C. S. Carter, T. S. Braver, J. D.Cohen, Technical Report PDP.CNS 98.1 (Center for the Neural Basis of Cognition, Carnegie Mellon University, 1998)], we have shown how performance monitoring could occur by measuring competition through the activity level of processing units. In this work, competition is indexed by the sum of the pairwise products of the activity level of the response units in the model. We have examined how this competition measure might correspond to ACC activity in the AX-CPT, Stroop, and stem completion tasks, as well as the behavior of the ERN in the Eriksen flanker task.

17. We gratefully acknowledge A. Sanders, K. Fissell, S. Forman, L. Nystrom, and A. Herbster for their contributions to this project. This study was supported in part by grants K08MH01306 and a NARSAD Young Investigator Award to C.S.C. and by RO1MH52864 to J.D.C.

26 November 1997; accepted 2 March 1998