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Event-Related Potential Activity in the Basal Ganglia Differentiates Rewards From Nonrewards: Response to Commentary

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Abstract: We recently demonstrated that the feedback negativity may be better understood as a reward-related positivity that is absent on nonreward trials, and source localization revealed that this reward response may reflect activity in the striatum. In a commentary on our report, Cohen et al. argue against this latter finding, claiming it is unlikely that the striatum contributes to the scalp-recorded event-related potential. We disagree with the line of reasoning presented by Cohen et al., and we respond here to each of their points. Based on all the available evidence, we argue that the striatum is a plausible generator of a reward-related response observed at the scalp, and this possibility warrants further investigation. *Hum Brain Mapp 32:2267–2269, 2011.* © 2011 Wiley Periodicals, Inc.

Key words: event-related potential; source localization; basal ganglia; striatum

INTRODUCTION

In a recent report, we used temporospatial principal components analysis (PCA) to parse the event-related potential (ERP) response to feedback indicating monetary gains and losses (Foti et al., 2011). The primary finding of this study was that the feedback negativity (FN) may be better understood as a reward-related *positivity* that is absent on nonreward trials. In other words, the apparent negative deflection in the ERP waveform actually reflects the *absence* of gain-related neural activity involved in reward processing. In fact, recent work from other laboratories has converged upon a similar conclusion, using both experimental manipulations (Baker and Holroyd, 2011; Holroyd et al., 2011; Holroyd et al., 2008) and time-frequency decomposition (Bernat et al., 2008; Bernat et al., 2011) to isolate this gain-related neural response. A second, preliminary finding in our report was that this reward-related positivity reflects activation of the striatum. Cohen et al. (this issue) suggest that this interpretation is "highly unlikely." We agree that the localization of scalp-recorded ERP activity to subcortical regions requires caution and ought to be further substantiated using complementary methods, such as fMRI and recordings from depth electrodes. For several reasons, however, we disagree with the line of reasoning presented by Cohen et al., and we reply here to each of their points.

(1 and 2) Intracerebral studies have demonstrated ERP-like activity in the striatum across a wide range of experimental tasks (Rektor, 2008). These studies show that it is possible to record electrical dipoles from within the basal ganglia, and that these dipoles create electrical fields that may be recorded from sites that are far away. As noted in our paper, combined evidence from depth and scalp electrodes indicates that subcortical activity—particularly in the striatum—can plausibly contribute to scalp-recorded activity (Rektor, 2002). The surface potential generated by a particular brain region depends not only on the distance to the scalp, however, but also the orientation of the neurons. Evidence from whole-brain anatomical models

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indicates that striatal activity is distinguishable from cortical activity using scalp-recorded potentials, and that it can be detected with ~20 trials (Attal et al., 2009).

In contrast, Cohen et al. cite work from their group (Cohen et al. (2009) in which they find only weak correlations between scalp-recorded ERPs and depth recordings from the basal ganglia during a reward learning task. Examining the scalp-recorded ERPs presented in the supplementary data section, however, suggests that unfavorable compared to favorable outcomes did not elicit an FN in the group of patients studied by Cohen et al. Following reward feedback, there was a positivity to losses and a negativity to gains, a pattern which is nearly opposite to the existing literature on the FN. The absence of an FN in their sample may be related to the fact that this group of individuals was currently depressed, which is known to attenuate neural responses to rewards (Eshel and Roiser, 2010)—and reduce the amplitude of the FN (Foti and Hajcak, 2009). Future studies in nondepressed patients that simultaneously record reward-related neural activity from depth electrodes and the scalp will certainly provide critical data on the correspondence between ERPs generated in the striatum and those recorded at the scalp.

(3 and 4) Previous studies have not separated the FN from overlapping responses, particularly the P300, which could influence source localization results. It is highly unlikely that the variation in the reward-related positivity is generated within the caudal anterior cingulate cortex (ACC): extensive neuroimaging evidence indicates that this region is not activated by monetary reward (Liu et al., 2011). However, it is possible that the FN is generated in part by the rostral ACC (Nieuwenhuis et al., 2005). For example, one study that combined ERPs and fMRI also localized the FN to the dorsal striatum, but, like Nieuwenhuis et al. found that the FN could be accounted for by a dipole in the rostral ACC (Martin et al., 2009). The relative contributions of the rostral ACC and the striatum will be relevant for future research. In a forthcoming report, we recorded both ERP and fMRI data from the same subjects performing the same gambling task (Carlson et al., under review). Using PCA, we again found that the FN was a reward-related positivity that source-localized to the striatum; moreover, FN magnitude correlated with reward-related hemodynamic activity throughout the mesocorticolimbic system, and the strongest correlations were between the FN and striatal activity. Seeding dipoles at each of the regions showing a significant BOLD response, we found that the FN was uniquely predicted by dipoles placed in the dorsal and ventral striatum, and not by the mPFC, orbitofrontal cortex, visual cortex, or motor cortex.

(5) We agree that PCA will only effectively separate responses that have distinct temporal and/or spatial distributions. In the sample figure provided by Cohen et al, the two simulated headmaps are nearly identical, but the time course of activity is not considered. Activity in the pair of ACC/PCC sources would need to be highly similar to the striatal source temporally, not just spatially, and also similar to one another. Although it is possible to have coherent electrical activity in two disparate regions such as the ACC and PCC, a more parsimonious explanation is that this activity is happening in a single region. Instead, as with any source analysis of ERP data, the relevant competing explanations are a single point source (i.e., the striatum) or a more superficial distributed source (i.e., a relatively broad region of the cingulate). Converging evidence from other methods is necessary to distinguish between these possibilities and, as noted above, in light of the existing fMRI and intracerebral evidence we favor the striatum as the more likely source.

Overall, we agree with Cohen et al. that interpreting ERP activity in terms of subcortical sources should be done with caution, and be strongly guided by existing anatomical and empirical evidence. The localization of the FN to the striatum is noteworthy insofar as it provides a link between the ERP literature and the vast neuroimaging and animal literatures on reward processing. Based on the available evidence, we argue that a medial frontal ERP response resulting from a dipole in the striatum is *plausible*, and warrants further investigation.

REFERENCES

Attal Y, Bhattacharjee M, Yelnik J, Cottereau B, Lefèvre J, Okada Y, Bardinet E, Chupin M, Baillet S, et al (2009): Modelling and detecting deep brain activity with MEG and EEG. IRBM 30:33–138.

Baker TE, Holroyd CB (2011): Dissociated roles of the anterior cingulate cortex in reward and conflict processing as revealed by the feedback error-related negativity and N200. Biol Psychol 87:5–34.

Bernat EM, Nelson LD, Holroyd CB, Gehring WJ, Patrick CJ (2008): Separating cognitve processes with principal components analysis of EEG time-frequency distributions. Proc SPIE 7074:1–10.

Bernat EM, Nelson LD, Steele VR, Gehring WJ, Patrick CJ (2011): Externalizing psychopathology and gain-loss feedback in a simulated gambling task: Dissociable components of brain response revealed by time-frequency analysis. J Abnorm Psychol 120:352–364.

Carlson JM, Foti D, Mujica-Parodi LR, Harmon-Jones E, Hajcak G (in press): Ventral striatal and medial prefrontal BOLD activation is correlated with reward-related electrocortical activity: A combined ERP and fMRI study. NeuroImage.

Cohen MX, Axmacher N, Lenartz D, Elger CE, Sturm V, Schlaepfer TE (2009): Neuroelectric signatures of reward learning and decision-making in the human nucleus accumbens. Neuropsychopharmacology 34:649–1658.

Eshel N, Roiser J (2010): Reward and punishment processing in depression. Biol Psychiatry 68:118–124.

Foti D, Hajcak G (2009): Depression and reduced sensitivity to non-rewards versus rewards: Evidence from event-related potentials. Biol Psychol 81:1–8.

Foti D, Weinberg A, Dien J, Hajcak G (2011): Event-related potential activity in the basal ganglia differentiates rewards from non-rewards: Temporospatial principal components analysis and source localization of the feedback negativity. Hum Brain Mapp DOI is: 10.1002/hbm.21182.

Holroyd CB, Krigolson OE, Lee S (2011): Reward positivity elicited by predictive cues. Neuroreport 22:49–252.

Holroyd CB, Pakzad-Vaezi KL, Krigolson OE (2008): The feedback correct-related positivity: sensitivity of the event-related brain potential to unexpected positive feedback. Psychophysiology 45:688–697.

Liu X, Hairston J, Schrier M, Fan J (2011): Common and distinct networks underlying reward valence and processing stages: A meta-analysis of functional neuroimaging studies. Neurosci Biobehav Rev 35:219–1236.

Martin LE, Potts GF, Burton PC, Montague PR (2009): Electrophysiological and hemodynamic responses to reward prediction violation. Neuroreport 20:1140–1143.

Nieuwenhuis S, Slagter HA, von Geusau NJ, Heslenfeld DJ, Holroyd CB. (2005): Knowing good from bad: differential activation of human cortical areas by positive and negative outcomes. Eur J Neurosci 21:3161–3168.

Rektor I (2002): Scalp-recorded Bereitschaftspotential is the result of the activity of cortical and subcortical generators - a hypothesis. Clin Neurophysiol 113:1998–2005.

Rektor I (2008):The cognitive event-related potentials in the basal ganglia: invasive studies. In: Ikeda A, Inoue Y. editors. Event-Related Potentials in Patients with Epilepsy: From Current State to Future Prospect, John Libbey Eurotext: Montrouge, France. pp 177–190.