cortex 48 (2012) 519–520

Available online at www.sciencedirect.com

SciVerse ScienceDirect

Journal homepage: www.elsevier.com/locate/cortex

Discussion forum

More on fMRI and the locus of perceptual learning

Dusana Dorjee [a,*] *and Jeffrey S. Bowers* [b]

[a] *School of Psychology, Bangor University, Bangor, Wales, UK*
[b] *School of Experimental Psychology, University of Bristol, Bristol, England, UK*

We would like to thank Yuko Yotsumoto as well as Karsten Rauss and Sophie Schwartz for their constructive comments on our paper (Dorjee and Bowers, 2012). We agree with many of the points made by Yotsumoto, but would like to address the claim that Yotsumoto et al. (2008) did not make claims regarding the locus of learning. The key finding of this study was that the blood-oxygenation-level-dependent (BOLD) signal in V1 changed as a function of texture discrimination task (TDT) practice, and on our reading, the authors linked the BOLD signal to learning within V1 in a number of passages. For example, Yotsumoto et al. (2008) wrote: “It should be noted that the activated region size did not expand in the trained V1 as learning proceeded. The absence of expansion in the activated region suggests that the learning and reorganization were localized.” (p. 830) and: “If the degree of BOLD activation is indicative of the degree of synaptic activity (Logothetis et al., 2001; Viswanathan and Freeman, 2007) the reduced BOLD activation that we observed is in accord with synaptic downscaling” (p. 830). We would also question the claim that previous psychophysical and imaging studies have indicated an involvement of V1 in TDT learning (Yotsumoto, 2012). The psychophysical studies do provide evidence for learning within V1 (or at least retinotopic cortex), but previous imaging studies are ambiguous for the reasons we have outlined. The insights from the psychophysical studies do not allow researchers to draw further conclusions from BOLD signal (e.g., that the number of synapses in V1 is upscaled/downscaled as a function of practice).

Rauss and Schwartz (2012) note that the BOLD signal is closely tied to synaptic input and synaptic activity within a given brain region. Some caution is needed here given that this hypothesis has been criticized, and there appears to be no association between BOLD signal and synaptic activity in some brain regions (e.g., Conner et al., 2011). But even if we accept this premise, we are unclear why Rauss and Schwartz conclude that models c–f are implausible. Imagine learning takes place in V2 (via changes in synaptic strength) resulting in more activation in V1 via top-down feedback. This will lead to more activity in the synapses in V1, and a corresponding change in the BOLD signal in V1, without any learning in V1. In addition, as noted in the target article, learning can lead to an increase or decrease in the number of synapses in a given region, and accordingly, there is no strong reason to assume that BOLD signal will go up or down in response to learning, even ignoring the impact that on-line top-down feedback can have on BOLD signal.

Rauss and Schwartz (2012) are also concerned that we have taken a too narrow perspective on learning, restricting ourselves to the locus of learning. All we can say is that it was the focus of our commentary. We were quite clear that functional magnetic resonance imaging (fMRI) might provide data useful for constraining theories of learning; it is just not well suited for some purposes.

In sum, we stand by our basic argument that a change in the BOLD signal before and after learning is ambiguous with regards to the locus of learning. Still, as emphasized in the comments, BOLD signals may provide relevant constraints for theories of learning. As noted by Karsten and Schwartz, the BOLD signal does provide information regarding the on-line processing consequences of learning (e.g., that V1 is more active following some forms of perceptual learning), and as noted by Yotsumoto, BOLD signals can be used to generate hypotheses regarding the locus of learning. These hypotheses can be usefully combined with other techniques (e.g., magnetic resonance spectroscopy) in order to make stronger conclusions. We hope this exchange clarifies the strengths and weakness of fMRI research when applied to questions of learning.

* *Corresponding author*. School of Psychology, Bangor University, LL57 2AS Bangor, Gwynedd, UK.
E-mail address: d.dorjee@bangor.ac.uk (D. Dorjee).

doi:10.1016/j.cortex.2011.11.004

REFERENCES

Conner CR, Ellmore TM, Pieters TA, DiSano MA, and Tandon N. Variability of the relationship between electrophysiology and BOLD-fMRI across cortical regions in humans. *The Journal of Neuroscience*, 31(36): 12855–12865, 2011.

Dorjee D and Bowers JS. What can fMRI tell us about the locus of learning? *Cortex*, 48(4): 509–514, 2012.

Logothetis NK, Pauls J, Augath M, Trinath T, and Oeltermann A. Neurophysiological investigation of the basis of the fMRI signal. *Nature*, 412(6843): 150–157, 2001.

Rauss K and Schwartz S. Dissociating learning-induced changes in fMRI signal from structural modifications: A comment on Dorjee and Bowers. *Cortex*, 48(4): 515–516, 2012.

Viswanathan A and Freeman RD. Neurometabolic coupling in cerebral cortex reflects synaptic more than spiking activity. *Nature Neuroscience*, 10(10): 1308–1312, 2007.

Yotsumoto Y. Why we should integrate behavioral and neuroimaging studies to examine neural plasticity in perceptual learning. *Cortex*, 48(4): 517–518, 2012.

Yotsumoto Y, Watanabe T, and Sasaki Y. Different dynamics of performance and brain activation in the time course of perceptual learning. *Neuron*, 57(6): 827–833, 2008.