The relationship between the fMRI BOLD response and beta band neuromagnetic effects

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Introduction: BOLD fMRI is a widely used method for exploring brain activity. However, for BOLD to reach its full potential it is necessary to understand the neural basis of the response. Previous studies have shown a good correlation between fMRI BOLD data and time-locked, non-phase locked oscillatory effects in MEG.[1]. Oscillatory activity in the β-band (15-30Hz) is a well studied effect in the sensorimotor cortex [2] and exhibits a chararcteristic loss in power (Event Related Desynchronisation-ERD) during motor activity followed by an increase in power (Event Related Synchronisation-ERS) on movement cessation [3]. Here, the induced oscillatory response in the β-band is measured by MEG and the haemodynamic response is measured by fMRI for both finger movement of varying durations and visual stimuli of varying contrast. We assess the linearity of the MEG and BOLD responses in order to determine the extent to which the BOLD response may be governed by β activity.

Methods: Four healthy subjects took part in the study. The motor paradigm comprised visually cued abductions of the right index finger (4Hz). A trial contained a 2s pre-stimulus rest period, finger movement of 1, 2, 4, or 6 second duration and a post stimulus rest period, making each trial 12s in total. For fMRI, trial durations were increased to 30s to allow the h.r.f to return to baseline. The visual paradigm comprised a sinusoidal drifting grating, presented in a circular window in the lower left hand quadrant of the visual field with a visual angle of 5°. Five contrasts (0, 0.125, 0.25, 0.5 and 1) were presented pseudo-randomly, with the stimulus presented for 4secs. Trial length was 8secs in MEG and 16 secs in fMRI. Both experiments consisted of 20 trials per duration/contrast in MEG and 8 trials per duration/contrast in fMRI. MEG data were acquired at a sample rate of 600Hz, on a 275-channel CTF system. Co-registration to anatomical MRI was performed using head digitisation (Polhemus Isotrack). MR data consisting of 18 axial slices covering the motor or visual cortex, respectively were acquired on a Philips 3T system running GE-EPI (TR=2000ms, TE=45ms for the motor experiment, TE=40ms for the visual, 3x3x3mm^3 voxels, 192mm FOV).

Data Analysis: MEG data were analysed using synthetic aperture magnetometry (SAM) [4]. Spatial localisation of β ERD was achieved by comparison of oscillatory power in an active contrast window, spanning the stimulus presentation time (pale blue region in inset in top right corner of Figure 1), and a passive time window spanning 2s of the post-stimulus rest period. An active window of 2sec following stimulus cessation (pink region in Figure 1 inset) was used for ERS localisation. Pseudo T-stat images [4] (1mm^3 resolution) were created showing regions of activity within these bands. Virtual sensor traces were located in peaks of activity in the SAM images and time courses of electrical oscillatory power were obtained by applying a Hilbert transform of the virtual sensor data and averaging across trials. Linearity of the β response was assessed by integration of the Hilbert envelope. Areas of significant (p=0.05 corrected) BOLD contrast were identified using SPM5. These regions of interest (T-stat >5.5) were used for spatial localisation. Regions with T-stat>8 were used to obtain average time-courses of the haemodynamic response. Linearity of the BOLD response was assessed by integration of the BOLD time course.

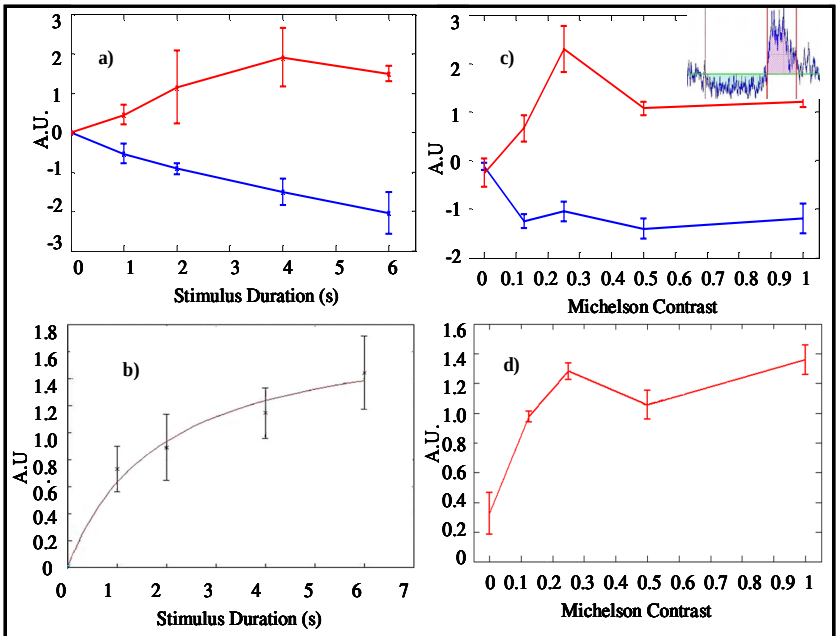

Figure 1: Linearity of a) β ERD (blue), β ERS (red) and b) BOLD response in the motor cortex with respect to stimulus duration and linearity of c) β ERD (blue), β ERS (red) and d) BOLD response in the visual cortex with respect to stimulus contrast. All results are averaged across subjects.

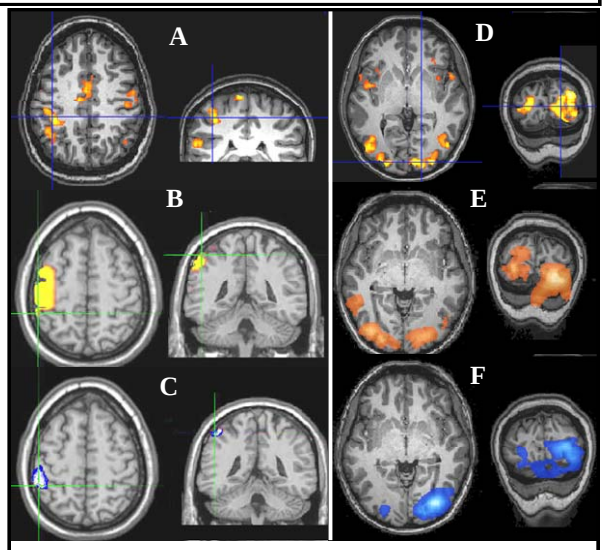

Figure 2: Spatial localisation of MEG and BOLD in a single representative subject for fingertap experiment (**A-C**:- [A] BOLD T>6, [B] β ERS Ŧ>2, [C] β ERD Ŧ>2) and visual experiment (**D-F**:- [D] BOLD T>5.5, [E] β ERS Ŧ>2, [F] β ERD Ŧ>2).

Results and Discussion: Figure 2 shows a compelling agreement in spatial localisation of the β ERD, ERS and BOLD data for both the motor and the visual experiment. Interestingly the rebound β activity appears slightly anterior to the ERD in the motor experiment and shows more bilateral activity than the ERD in the visual experiment. Figure 1a shows a linear trend in the β power loss (shown in blue) with respect to stimulus duration, as one might expect if it is to be thought of as an idling rhythm which is switched off during stimulation. In contrast, the BOLD response shown in Figure 1b is non-linear and tends to saturate at longer stimulus durations suggesting a degree of adaptation. The rebound ERS in Figure 1a has no distinct trend with stimulus duration. The β ERD shown in Figure 1c seems to behave as a threshold effect rather than changing with stimulus contrast. The ERS is again much more variable with no obvious trend. The BOLD derived contrast response curve exhibits a non-linear trend and appears to saturate at higher contrasts.

Conclusion: The excellent co-localisation of BOLD and β activity (both ERD and ERS) strongly suggests that these processes are intimately linked. However, the exact nature of this link is unclear: the dependences of the β-ERS on duration (motor) and contrast (visual) corresponds reasonably well to those of the BOLD response, but the dependences of the β ERD, the more robust response across all subjects does not and it is somewhat counter intuitive that a loss in power in the β band should lead to an increase in energy demand and hence a BOLD effect. Nevertheless, this MEG effect is reflective of a change in state of a large population of neurons and so could still be linked to increases in metabolic activity. Reconciliation of these different measures of brain activity will require improved understanding of how each relates to the underlying activity, and this in turn will shed light on the neural mechanisms, themselves.

References: [1]M.J. Brookes et al., Neuroimage 26(2005) 302-308, [2]G. Pfurtscheller et al.,Clin. Neurophys. 110(1999)1842-1857,[3]C.M. Stevenson et al., Intern. Cong. Series 1300(2007) 325-328, [4]S.E. Robinson et. al., Biomag 98, 11th Int Conf on Biomagnetism, 1998.