A stochastic linear model of neurovascular dynamics in the BOLD signal

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Introduction: The mapping from neuronal activation to measured blood oxygenation level dependent (BOLD) signal in fMRI involves a complex interplay between physiological and physical processes that is yet to fully elucidated. Methods that account for the variability of the hemodynamic response function (HRF) have, with few exceptions (eg.[1] and subsequent works), focused on parameterised, deterministic models of the HRF (eg. [2]). We propose a stochastic linear model (SLM) of the measured BOLD response, in which noise drives the neurovascular dynamics directly. The key difference between the SLM and the 'bilinear dynamical systems' approach [3] is that the SLM employs a fully stochastic model of the neurovascular response rather than attempting to separate stochastic neuronal activity from deterministic HRF's.

Methods: The SLM models the BOLD signal as the summation of a stochastic neurovascular signal, autoregressive (AR), moving average noises and Gaussian measurement noise. The stochastic neurovascular signal is modelled by an AR(1) model with parameter *a*, with the stimulus sequence an exogenous input modulated by weighting parameter *b*. An iterative coordinate descent algorithm [4] estimates both the unknown neurovascular signal and unknown signal and noise parameters. The SLM activation weight is defined as $\beta^{SLM} = b/(1-a)$, derived from the limit of the signal model autoregression.

Three healthy controls were scanned (3T Siemens TRIO) while performing a simple motor task (222 EPI images, in-plane: 3.125x3.125mm^2, TR=1.6s, FA=90°, TE=20ms, 24 axial slices of 5mm with 0.5mm gap). The visuomotor task was presented as a block design of alternating periods (30s each). Non-normalised images were motion corrected and spatially smoothed (6mm isotropic Gaussian kernel). Three ROIs were delineated (3mm radius spheres) for each subject using anatomical landmarks over primary motor cortex in both hemispheres (M1L and M1R) and over the supplementary motor area (SMA). For comparison with the SLM, activation weights based on a general linear model, β^{GLM}, were calculated.

Results: The activation weights estimated with the SLM are stronger in the motor cortical regions than the GLM and appropriately suppressed elsewhere in the brain (Fig.1). The SLM estimated neurovascular signal better models the observed BOLD signal compared to the GLM estimate (Fig.2), without overfitting as evidenced by flat auto- and partial auto-correlation functions. The SLM has similarly been shown to be more robust and consistent than the GLM, demonstrated on a seven-subject motor task fMRI dataset (data not shown).

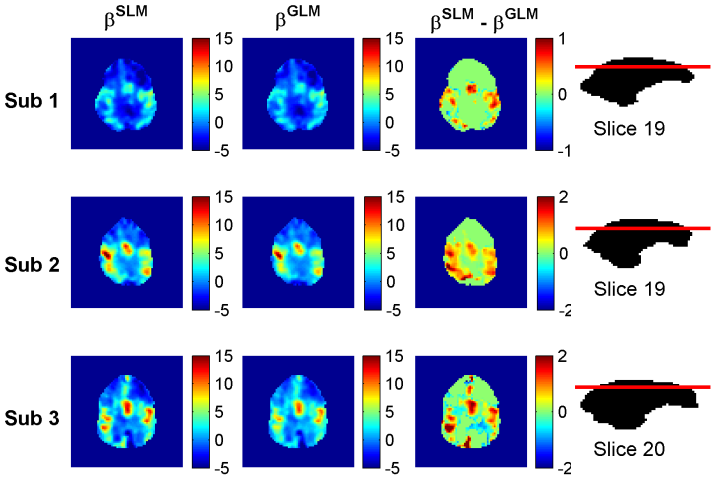

Figure 1: Activation evident in M1L, M1R and SMA of 3 subjects, is enhanced by SLM: β^{SLM} (column 1), β^{GLM} (column 2), β^{SLM} - β^{GLM} (column 3).

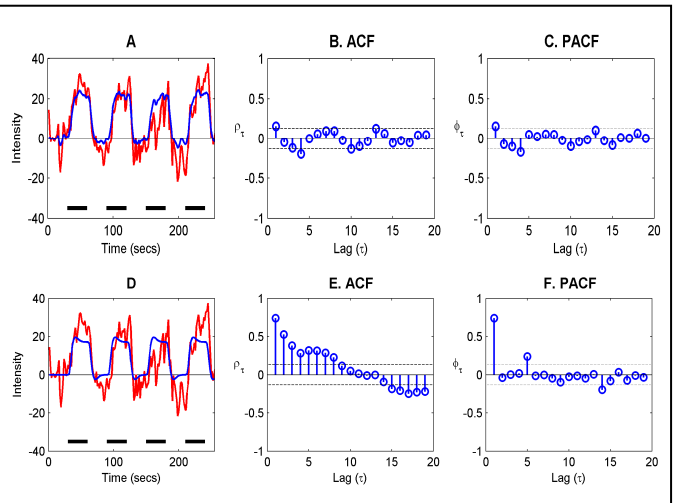

Figure 2: Subject 3. (A-C): SLM. (D-F): GLM. (A,C) SMA time-series (red), estimated neurovascular signal (blue), (B,E) ACF, (C,F) PACF.

Discussion: The use of state noise to drive the system dynamics is a fundamentally different approach than the inclusion of AR noise in the observation equation, as is common in the GLM. The ARX model proposed in [5] is in the observation equation and thus reduces to a deterministic model in high SNR. What has previously been described as the nonlinearity of the HRF [6] is now placed in the domain of deterministic versus stochastic. It is of interest to compare in future the SLM with methods in which parameterised, deterministic HRF's are estimated [2], or those in which the HRF is constrained to be a smoothly varying function [1]. Similarly, in future work we will compare the stochastic linear model with recent nonlinear extended balloon models of the BOLD signal [7] and their estimation strategies.

References: [1] Ciuciu et al. (2003) IEEE Trans.Med.Im. 22:1235-1251. [2] Woolrich et al. (2004) IEEE Trans.Med.Im. 23:213-231. [3] Penny et al. (2005) Phil.T.Roy.Soc.B. 360:983-993. [4] Luenberger (1984) *Linear and Nonlinear Programming*, Addison-Wesley. [5] Baraldi et al. (2007) NeuroImage 37:189-201. [6] Birn et al. (2001) NeuroImage 14:817-826. [7] Deneux and Faugeras (2006) NeuroImage 32:1669-1689.