Physiological noise covariance across receiver channels explains time-series SNR model for RF coil array fMRI data

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Introduction: The temporal SNR is the relevant figure of merit in fMRI time series data for assessing the detection of neuronal activations in the presence of noise fluctuations. The original model for relating image SNR to temporal SNR demonstrated that physiological noise scales linearly with signal level, and in so doing provided a quantitative relationship between image SNR and temporal SNR [1]. Recently it has been demonstrated that fMRI data acquired with multi-channel array coils deviates from the standard Kruger model [2,3] and the deviation appears to increase with element count [2]. An additional parameter in the model yielded a better fit to the data (at the cost of an additional degree of freedom), however its origin and meaning is unclear. We note that the Kruger model does not consider potential correlations of the physiological noise across the array coil channels. Here we revisit this extended model and consider the effects of these correlations. We demonstrate that extending the physiological noise model to include a physiological covariance matrix (with non-zero off-diagonal entries) can explain the observed deviation of the data from the Kruger model and provides an interpretation of the recently proposed models. In this work we show that multi-channel data only obeys the Kruger model for a specific relationship of the physiological noise correlations between channels, and that this relationship is violated in experimental data.

Theory: Temporal SNR of a particular voxel in the image data acquired with an *N*-channel array coil is given by $\mathrm{tSNR} = \left|\mathbf{w}^{\mathrm{H}}\mathbf{s}\right| / \sqrt{\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}\mathbf{w}}$, where $\mathbf{s}$ is the signal level vector across all channels, $\boldsymbol{\Psi}$ is the total channel noise covariance matrix, and $\mathbf{w}$ is the (complex-valued) combination weight vector. Assuming that thermal noise and physiological noise are statistically independent, $\boldsymbol{\Psi} = \boldsymbol{\Psi}_0 + \boldsymbol{\Psi}_{\mathrm{p}}$ where $\boldsymbol{\Psi}_0$ represents the thermal noise covariance and $\boldsymbol{\Psi}_{\mathrm{p}}$ represents the physiological noise covariance across channels. The tSNR can be expressed in terms of the thermal SNR, SNR_0, as

$$\mathrm{tSNR} = \frac{\left|\mathbf{w}^{\mathrm{H}}\mathbf{s}\right|}{\sqrt{\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_0\mathbf{w} + \mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_{\mathrm{p}}\mathbf{w}}} = \frac{\mathrm{SNR}_0}{\sqrt{1 + \dfrac{\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_{\mathrm{p}}\mathbf{w}}{\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_0\mathbf{w}}}} \qquad (1)$$

Where $\mathrm{SNR}_0 = \left|\mathbf{w}^{\mathrm{H}}\mathbf{s}\right| / \sqrt{\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_0\mathbf{w}}$. In the standard Kruger model, the physiological noise standard deviation σ_{p} in a single channel is proportional to the signal level s in that channel, or $\sigma_{\mathrm{p}} = \lambda s$. This implies that the physiological noise variance can be predicted from the signal level alone—assuming a fixed physiological SNR within a particular tissue class [4]. In order for Eq. (1) to reduce to the Kruger model for the multi-channel case, then $\boldsymbol{\Psi}_{\mathrm{p}}$ must be proportional to an outer product of the signal vectors; explicitly, $\boldsymbol{\Psi}_{\mathrm{p}} = \lambda^2\mathbf{s}\mathbf{s}^{\mathrm{H}}$. Then $\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_{\mathrm{p}}\mathbf{w} = \left|\lambda\mathbf{w}^{\mathrm{H}}\mathbf{s}\right|^2$ and therefore

$$\mathrm{tSNR} = \frac{\mathrm{SNR}_0}{\sqrt{1 + \dfrac{\lambda^2\left|\mathbf{w}^{\mathrm{H}}\mathbf{s}\right|^2}{\mathbf{w}^{\mathrm{H}}\boldsymbol{\Psi}_0\mathbf{w}}}} = \frac{\mathrm{SNR}_0}{\sqrt{1 + \lambda^2\mathrm{SNR}_0^2}} \quad \text{if } \boldsymbol{\Psi}_{\mathrm{p}} = \lambda^2\mathbf{s}\mathbf{s}^{\mathrm{H}}.$$

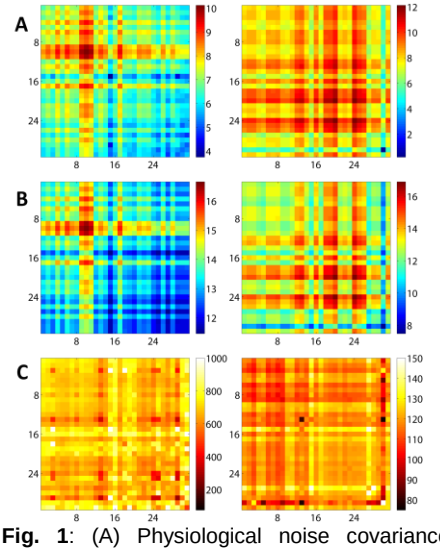

Fig. 1: (A) Physiological noise covariance matrix $\boldsymbol{\Psi}_{\mathrm{p}}$ and (B) signal level matrix $\mathbf{s}\mathbf{s}^{\mathrm{H}}$ calculated from two locations in the cortical gray matter (both displayed on a log scale). (C) Ratio of the two, displayed as percentage.

Intuitively, each entry (*i*,*j*) in the *N*×*N* matrix $\mathbf{s}\mathbf{s}^{\mathrm{H}}$ at a particular voxel captures the product of the signal levels between coils *i* and *j* at that voxel. (Note that $\mathbf{s}\mathbf{s}^{\mathrm{H}}$ is distinct from the signal correlation matrix.) If $\boldsymbol{\Psi}_{\mathrm{p}} = \lambda^2\mathbf{s}\mathbf{s}^{\mathrm{H}}$, then the standard model applies to multichannel data, otherwise the physiological noise covariance may be expressed as $\boldsymbol{\Psi}_{\mathrm{p}} = \mathbf{C}_\kappa + \lambda^2\mathbf{s}\mathbf{s}^{\mathrm{H}}$ for some matrix $\mathbf{C}_\kappa$ since covariance matrices are positive semi-definite.

Methods: Two subjects were studied at 7 T on a Siemens whole-body scanner (Siemens Healthcare, Erlangen, Germany) using acquisition protocols described previously [2,5]. The BOLD-weighted GE-EPI acquisition parameters were: TR/TE/fa/matrix/BW/esp/N_{slc}/N_{rep}=5.4 s/20 ms/90°/128×128/3004 Hz/px/0.41 ms/20/60 with 3 mm isotropic voxels, and a custom-built vendor 32-channel head array coil [6]. All data were reconstructed offline [2], the linear trend was removed from each time series, and $\boldsymbol{\Psi}$ was measured by calculating the correlation between the time-series of each channel. The physiological noise covariance was calculated by subtracting the image noise covariance from $\boldsymbol{\Psi}$.

Results: Fig. 1 demonstrates the physiological noise covariance matrix $\boldsymbol{\Psi}_{\mathrm{p}}$ and the signal level matrix $\mathbf{s}\mathbf{s}^{\mathrm{H}}$ at two distant locations within the cortical gray matter. While some large entries are common to both matrices, the log scaling in Fig. 1 highlights the many differences between the structure of the two matrices, and there is no constant scaling factor λ^2 that can be applied to $\mathbf{s}\mathbf{s}^{\mathrm{H}}$ to match $\boldsymbol{\Psi}_{\mathrm{p}}$. The mismatch between these two matrices demonstrates that the standard model does not fully account for physiological noise covariances.

Discussion: The observation of a direct relationship between signal level and physiological noise in a single channel does not extend to a similar relationship between the physiological noise covariance between a pair of channels and the product of their signal levels. While it may be the case that fMRI data acquired from two nearby channels will have similar signal levels and physiological noise coupling, it is also possible that distant coil elements (e.g., on opposite sides of the head) have similar signal levels yet very little physiological noise correlation. The generalized model can capture the effect of physiological noise covariance, whose influence is expected to increase with larger numbers of coil elements. Because much of the fluctuations observed in fMRI time series data is due to global physiological noise sources [7], successful removal of these nuisance factors will likely impact the physiological noise covariance across channels perhaps increasing the ability of the Kruger model to describe the relationship between image and time-series SNR. Future work will investigate whether the matrix $\mathbf{C}_\kappa$ is dependent on signal levels, noise levels, or both.

References: [1] Krüger *et al.* (2001) *MRM* 46:631. [2] Triantafyllou *et al.* (2011) *ISMRM* 19:3593. [3] Hutton *et al.* (2011) *ISMRM* 19:3630. [4] Bodurka *et al.* (2007) *NeuroImage* 34:542. [5] Triantafyllou *et al.* (2008) *ISMRM* 16:2465. [6] Keil *et al.* (2010) *ISMRM* 18:1493. [7] Bianciardi *et al.* (2009) *MRM* 27:1019. **Acknowledgements:** Supported by NCRR P41 RR14075, NIBIB R01 EB006847, and NIBIB K01 EB011498.