

The Measurement of Spatially-Dependant Tissue Sodium Concentration Increase in a Rodent MCAO Stroke Model

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Purpose and Introduction: The aim of this study was to accurately quantify changes in the Tissue Sodium Concentration (TSC) in the acute phase of a rodent stroke model with high spatio-temporal resolution ($0.38\text{mm}^3/10\text{min}$) at 7T. The ability to accurately quantify subtle changes in TSC could provide a marker for tissue viability in stroke [1]. However, the quantification accuracy is limited by the typical low SNR's arising from the inherently low TSC, fast signal decay and low gyro-magnetic ratio of sodium.

Subjects and Methods: A $^{23}\text{Na}/^1\text{H}$ birdcage resonator with 72mm inner diameter (i.d.) and a square-shaped receive-only surface coil (26mm^2) were developed to maximize SNR and B_1 -field homogeneity for quantitative ^{23}Na -MRI while also allowing for the acquisition of anatomical ^1H images. A modified 3D GRE sequence on a Bruker BioSpec 70/30 system was used to achieve as short a TE as possible ($800\mu\text{s}$). Stroke was induced using the intra-luminal thread model in the left hemisphere of the rat brain. The contralateral hemisphere, unaffected by the stroke, served as a control during the experiment. ^{23}Na images were measured from 2h to 5h post stroke onset time (SOT) in 10min steps (Figure 1). A representative ADC map acquired 1h post SOT in a previous study is shown in Figure 2a. The histology results (haematoxylin and eosin staining) showed a clear lesion of the left hemisphere (Figure 2b).

Results and Discussion: A linear fit of the TSC change after the stroke onset time (Figure 2c) revealed a slope of 10- 30mM/h across the lesion region, as delineated by histology. Indeed, the effects of variable collateral supply to different regions of the brain were indicated by the different times at which the TSC began to increase at different positions within the lesion, all of which nonetheless increased to a maximum value of approximately 145mM, corresponding to the extra-cellular sodium concentration. A recent study determined the SOT using an identical linear model, although only relative rather than absolute quantitative TSC values were measured, and consequently it was not possible to take into account the TSC limit of approximately 145mM imposed by the buffering of sodium levels from the vascular compartment [2]. As a result, SOT estimation could prove highly inaccurate when linear regression is performed at arbitrary time points. The current study provides improved spatio-temporal resolution coupled with quantitative measures of the TSC, allowing for a more realistic assessment of the stroke evolution. Future studies will focus on the determination of a tissue viability threshold to develop a clinically applicable parameter for stroke diagnosis.

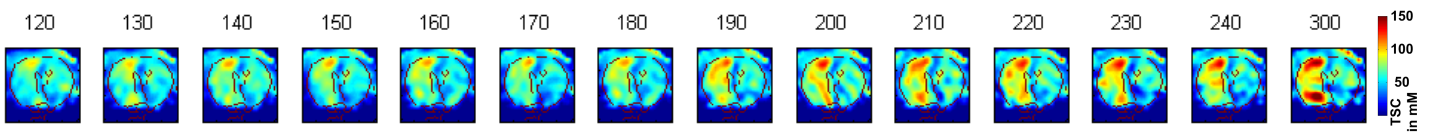


Figure 1: TSC maps with superimposed ^1H edge images at multiple time points. Times are labeled above each map in minutes post SOT.

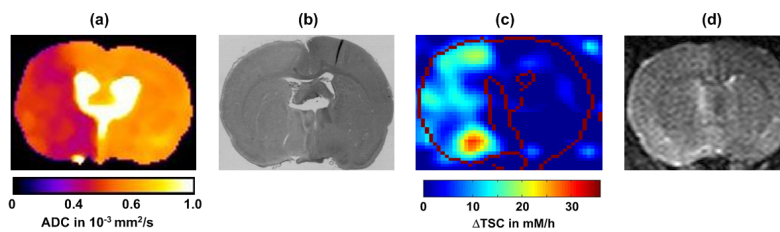


Figure 2: (a) ADC map 1h pSoT, (b) Histology 6h pSoT, (c) TSC slope map, (d) T_2 -weighted ^1H image 5h pSoT.

References: [1] R. Ouwerkerk et al, *Tissue sodium concentration in myocardial infarction in humans: a quantitative ^{23}Na MR imaging study*. Radiology, 2008. 248(1): p. 88-96. [2] S.C. Jones et al, *Stroke onset time using sodium MRI in rat focal cerebral ischemia*. Stroke, 2006. 37(3): p. 883-8.