

# Dual-Echo Magnetic Resonance Spectroscopy Imaging: application to Traumatic Brain Injury

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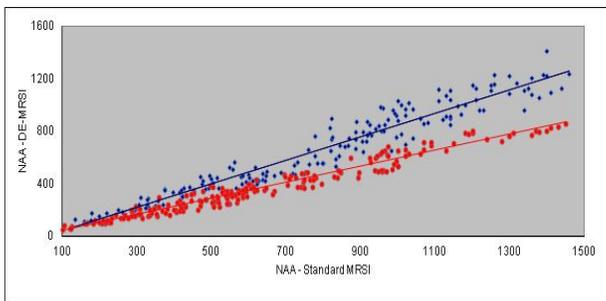
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## Introduction

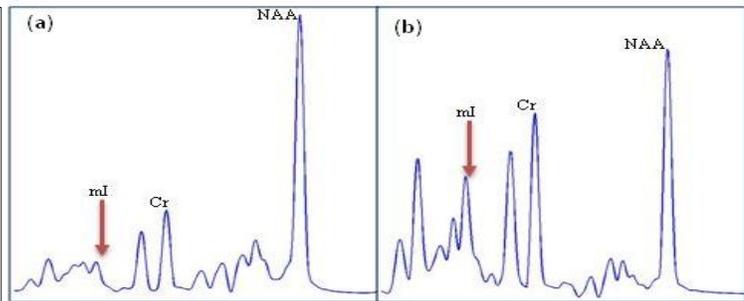
Magnetic Resonance Spectroscopy Imaging (MRSI) allows localized measurement of *in vivo* metabolic composition from a multidimensional array of spatial locations. In MRSI studies of the human brain, the echo time (TE) of acquisition plays a huge role in the determining which metabolite signals are present in the spectrum as well as how well the baseline for signal quantification can be defined. Short TE acquisitions enable the measurement of signal from short T2 metabolites such as *myo*-inositol (mi), glutamate, GABA and macromolecules<sup>1</sup>. However short TE spectra are commonly contaminated by broad, overlapping lipid resonances that inherently complicate baseline determination and metabolite quantification. Therefore several investigators have opted for obtaining spectra acquired at long TE which allows a better-defined baseline but does not include signals from short T2 metabolites. Given that the scan times are long for MRSI, seldom both the long and short TE data is obtained on the same subject within the same session, resulting in an incomplete understanding of the underlying biochemical changes. Herein we describe a dual-echo (DE) MRSI acquisition that is capable of simultaneously acquiring both long and short TE data sets with the same scan time as a standard MRSI method.

## Methods

DE-MRSI sequence was developed by modification of a vendor supplied standard MRSI sequence. In order to accommodate for the dual acquisition scheme, the short TE echo was sampled with a dwell-time of 200 $\mu$ s (5 times less than the standard acquisition). Because short T2 metabolites such as *myo*-inositol are of interest for characterizing TBI, we sacrificed some spectral resolution during the first echo (TE = 30ms). The sequence was calibrated and compared with the standard vendor supplied sequence for the spectra obtained from the short echo time using an in-house <sup>1</sup>H-MRS phantom containing all the normal brain metabolites. All scans were implemented on a Siemens Tim-Trio 3T MRI scanner using a 12-channel receive only head coil. A linear regression was used to compare measurements obtained from the <sup>1</sup>H-MRS phantom using the DE-MRSI sequence to measurements obtained from the <sup>1</sup>H-MRS phantom using the standard MRSI sequence. The DE-MRSI sequence was also tested on human subjects to determine how well pathology related changes could be detected. Nine Traumatic Brain Injury (TBI) patients and 7 healthy volunteers were scanned using the DE-MRSI sequence with scan parameters as follows: TE1 30ms, TE2 270ms, TR 1320ms, BW1 5kHz, BW2 1kHz, FOV = 160 x 160 x 106, VOI = 106 x 106 x 48, acquired resolution 12x12x8, total acquisition time 7min 40 secs. Metabolite quantification was performed using LCMoDel<sup>2</sup>. The regions analyzed for metabolic alterations due to TBI included *Right Putamen* (RPUT), *Left Putamen* (LPUT), *Right Periventricular White Matter* (RPWM), *Left Periventricular White Matter* (LPWM) and the *Thalamus* (THAL). A two-tailed independent samples *t*-Test was used to determine group differences in metabolite-ratio values between TBI patients and the healthy volunteer group.



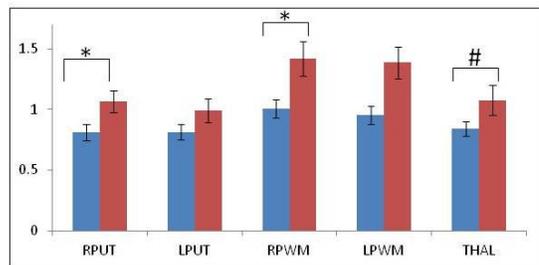
**Figure 1.** Linear correlation of NAA signal measured in different voxels of a <sup>1</sup>H-MRS phantom by the DE-MRSI sequence with NAA signal measured in the corresponding voxels by the standard MRSI sequence at TE = 30ms (blue diamonds) and TE = 270ms (red circles)



**Figure 2.** Short TE <sup>1</sup>H-MRS spectrum of RPWM acquired using DE-MRSI sequence for (a) healthy control subject (b) Traumatic Brain Injury subject. Note the increase in *myo*-inositol and a decrease in NAA in the TBI patient

## Results

Figure 1 shows a strong linear relationship between metabolite values measured by the standard MRSI sequence and metabolite values measured by the DE-MRSI



**Figure 3.** Comparison of mi/Cr levels of various anatomical regions between a healthy volunteer group (blue bars) and TBI group (red bars) using short TE DE-MRSI data. Error bars indicate standard error. #*p*<0.1 ; \**p*<0.05

sequence in a <sup>1</sup>H-MRS phantom for both short TE ( $r^2 = 0.9356$ ) and long TE ( $r^2 = 0.9561$ ) data. The quality of the spectra with the reduced spectral width is tolerable and is able to easily discern short T2 metabolites as shown in Fig 2 in the case of a control subject and a TBI patient. Figure 3 shows increases in mi/Cr levels in various anatomical regions of the brain parenchyma of TBI patients when compared to healthy controls.

## Discussion

The preservation of a strong linear correlation between metabolite values measured by the standard MRSI and DE-MRSI sequences indicates that measurements from the DE-MRSI sequence agree with the vendor supplied standard MRSI sequence despite the decreased spectral resolution. Figure 1 serves as a calibration curve to correct for losses in SNR and to arrive at absolute concentration values of the individual metabolites when necessary. Using the DE-MRSI technique we found increases in mi/Cr levels in TBI patients when compared to healthy controls. Increases in mi/Cr levels in the brain resulting from TBI has been previously reported<sup>3,4</sup>.

## Conclusion

DE-MRSI technique allows us to obtain a complete picture of all the metabolites that are possible with <sup>1</sup>H-MRSI and can provide complete information of the metabolic patterns of change in various pathophysiologies in the same time as obtaining data from a single echo.

**References:** [1] Ross B, et al *Anat Rec.* 2001;265:54-84. [2] Provencher SW. *NMR Biomed* 2001;14:260-264. [3] Garnett MR, et al *Brain* 2000; 123:2046-2054 [4] Brooks WM, et al *J Neurotrauma* 2000;8:829-840