

Diffusion Weighted Imaging, ADC mapping, and Sodium MR imaging of Operable Breast Cancer after Neoadjuvant Therapy: Preliminary Results

M. A. Jacobs^{1,2}, R. Ouwekerk¹, V. Stearns², K. Macura¹, A. C. Wolff², R. El Khouri¹, I. Kamel¹, and D. Bluemke^{1,3}

¹The Russell H. Morgan Department of Radiology and Radiological Science, The Johns Hopkins University School of Medicine, Baltimore, MD, United States, ²Sidney Kimmel Comprehensive Cancer Center, The Johns Hopkins University School of Medicine, Baltimore, MD, United States, ³Radiology, NIH Clinical Center, Bethesda, MD

Purpose: To prospectively investigate the feasibility of using magnetic resonance imaging (MRI) Diffusion Weighted imaging (DWI) with Apparent Diffusion Coefficient (ADC) mapping and sodium imaging (²³Na) before (baseline) and after preoperative systemic treatment (PST) in breast cancer.

Methods: Eleven patients with suspicious breast lesions were prospectively enrolled into this study. Six patients were classified as BIRADS 4 or 5 after conventional imaging and did not receive PST. Five patients (BIRADS 5) with large masses underwent PST. Fat suppressed ¹H MRI T₂ spin echo (SE), T₁ fast spoiled gradient echo (FSPGR), fat-suppressed 3D T₁-FSPGR pre- and post-contrast images were obtained after injection of GdDTPA contrast agent (0.1mmol/kg), DWI was acquired using SE-EPI(TR/TE=5000/90ms,128x128,b=0,500, 750,1000s/mm²,NEX=1) and ²³Na MR images were obtained with Twisted Projection Imaging which allows ultra short TE (TE/TR=0.4/100ms)[1]. Total data acquisition time was about 45 min. Trace ADC maps were constructed and ADC values (mean±SD) and ratios of glandular and lesion tissue were obtained. Quantitative estimates of total sodium concentration (TSC) mmol/L (mM) were made using an external reference technique [1]. Breast lesions were categorized by histopathology, all patients receiving PST were determined as responders based on pathology. Statistical significance was set a p<0.05.

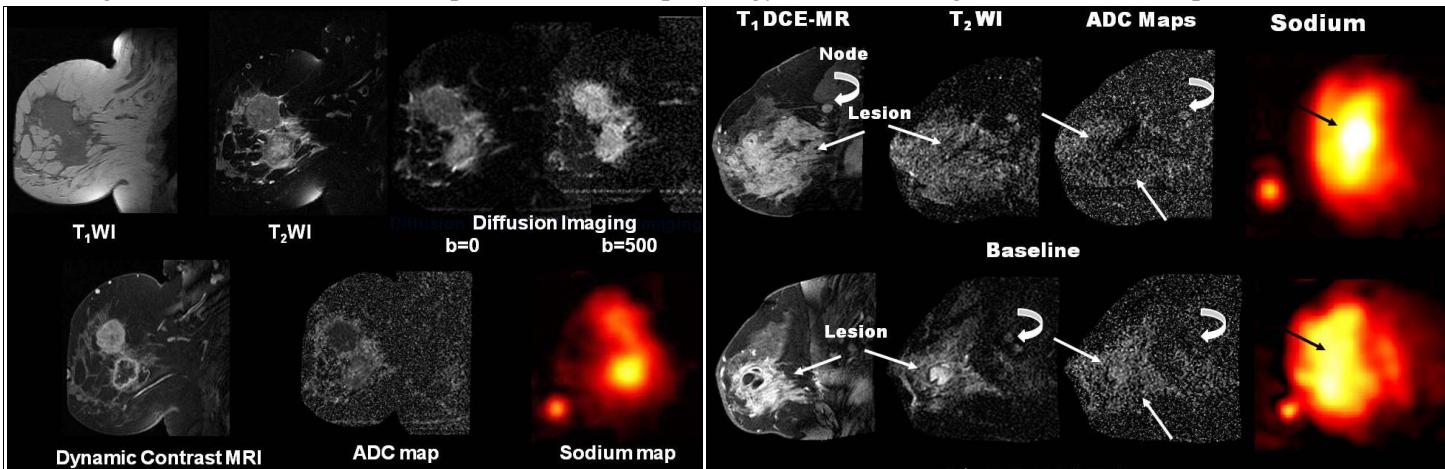


Figure 1. Representative 43y/o patient with a T3N1 lesion. Multiparametric T₁- and T₂-weighted, DCE, DWI/ADC, and Sodium images, demonstrating a heterogeneous breast lesion. The superior (12 o'clock) lesion has a homogenous enhancement with decreased ADC and lower TSC. The deep lower lesion exhibits rim enhancement and increased ADC with higher TSC.

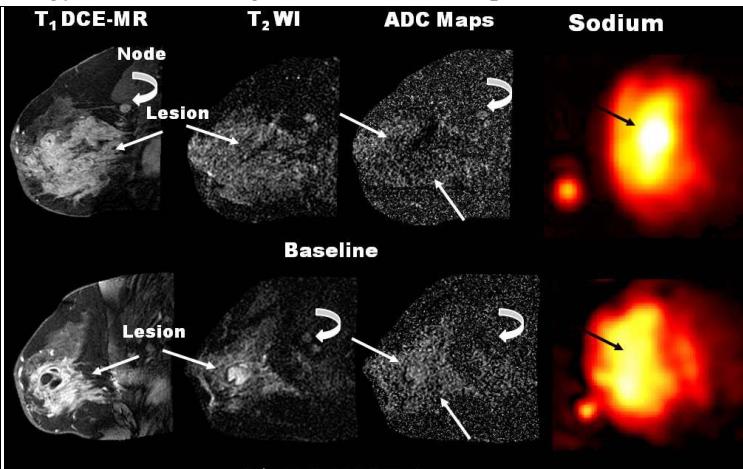


Figure 2. Representative 39 y/o patient with a T4dN0 lesion. Baseline DCE T₁- and T₂-weighted, DWI/ADC, and Sodium images demonstrating a large enhancing left breast lesion with heterogeneous ADC values and increased TSC. After PST, decreased enhancement in the lesion and increased ADC with small focal regions of decreased ADC. There was a decrease in the TSC. This patient was a responder.

Results: In non-therapeutic patients, the mean ADC value in was $1.17 \pm 0.26 \times 10^{-3} \text{ mm}^2/\text{s}$ with a lesion to glandular (L/GT) ratio=0.62. In PST patients, the mean baseline lesion ADC value was $1.21 \pm 0.11 \times 10^{-3} \text{ mm}^2/\text{s}$ with L/GT=0.70, mean ²³NA was 69mM and lesion volume was 162mm³. After the first PST cycle (7days), there was increase (46%; p=0.02) in the mean ADC value, $1.78 \pm 0.11 \times 10^{-3} \text{ mm}^2/\text{s}$, L/GI=1.18, with a decrease in ²³NA concentration (26%;49mM;p=0.06) and tumor volume (37%;102mm³). **Discussion:** The ADC metric increases after treatment with concurrent decreases in the TSC concentration were indicative of a therapeutic response as determined by pathological correlation. ADC mapping is a measure of the movement of water [3,4], whereas, ²³NA adds a measure of the sodium concentration and the characteristics of cellular environment within breast tissue [1,2]. Thus, combined clinical proton (¹H) imaging, DWI/ADC mapping, and sodium (²³Na) imaging provides radiological biomarkers of molecular and metabolic environment for the monitoring of therapeutic intervention in breast cancer.

References: [1] Ouwekerk R, BCR T 2007. [2] Jacobs MA, TCRT 2004;3(6):543-550. [3] Chenevert T. L. *JNCI*;2029-2036. 2000. [4] Sharma U; *NMR Biomed* 2008 **Acknowledgement:** NIH grants: 1R01CA100184; P50 CA103175; 5P30CA006973(IRAT)