**ABSTRACT**

Traumatic brain injury was delivered to the right dorsolateral frontaloparietal cortex of adult male rats (n=8) using the fluid percussion method. Five days later animals were scanned using a 7.0 Tesla MR scanner for T1 and T2 relaxation values. Imaging was acquired to study long term changes in emotion and cognition associated with the trauma. There were no significant changes in T1 relaxation.

**MAIN RESULTS**

Shown are the average (n=8) significant increase in BOLD signal intensity (yellow/red) following 5% CO2 challenge over the rostral/caudal boundaries of the TBI lesion. There were no significant differences in vascular responsivity between lesioned and control sides. The T2 relaxivity maps show a change in transverse relaxation in the rostral/caudal boundaries of the TBI lesion (arrows) in a representative rat. This change reflects an increase in water due to edema associated with the trauma. There were no significant changes in T1 relaxivity.

**SUMMARY AND CONCLUSIONS**

Magnetic resonance imaging was used to characterize the extent of brain injury in response to a unilateral insult to the frontaloparietal cortex. As expected, high resolution anatomical images clearly show the rostral/caudal limits of tissue damage in the cortical mantle on the side of the trauma. While there were no significant changes in T1 relaxivity, T2 measures clearly showed the extent of the trauma as reflected by the increase of water with edema. When challenged with CO2 there was no difference between the ipsilateral and contralateral halves of the brain. This indicates no change in vascular responsivity and would predict normal coupling between metabolically active brain regions and cerebral blood flow.

DTI with tractography showed disruption in myelinated fiber tracts on the side of the brain trauma (see white circles in DTI panel). Quantitative anisotropy when combined with co-registration into a 3D, segmented, annotated MRI rat atlas detected many areas that showed alterations in water diffusion. Interestingly, many of these area were subcortical, particularly the hypothalamus, amygdala and hippocampus. These data indicate that trauma directed to the cortex can alter the microarchitecture of deeper brain areas. This finding my help to explain the changes in emotion and cognition associated with TBI.