Introduction:
Soldiers from the Iraq and Afghanistan conflicts are commonly exposed to blast-induced Traumatic Brain Injuries (TBI) from improvised explosive devices (IEDs). The mechanism producing brain dysfunction from blast (exposure to wave-induced changes in atmospheric pressure) may be different from civilian injuries (motor vehicle accidents, sports injuries) that result primarily from mechanical forces. Little is known about pathological differences between blast-induced TBI and non-blast-induced TBI, although there exist some evidence that these TBI subtypes may be differentiated[1]. fMRI has shown reduced cortical motor and cerebellar activation to motor tasks in TBI compared to controls and suggests that the well-characterized motor network may be useful for detecting diffuse abnormalities[2]. Resting state functional connectivity MRI (fcMRI) is just beginning to be applied to the study of TBI. Increases in moderate/severe TBI[3] and decreases in mild TBI[4] in within-network default mode connectivity have been observed and increased thalamocortical FC that correlates with neurocognitive dysfunction[5]. We investigated motor cortex functional connectivity in participants with blast versus non-blast mild to moderate TBI participants and controls with orthopedic injuries. In comparing combined TBI groups versus controls, we observed local cortical motor cortex connectivity to be preserved, but more distant (e.g. visual) motor connectivity to be reduced. Furthermore, we found that connectivity between medial thalamic and primary motor cortex is specifically increased in blast-TBI compared to non blast-TBI participants. This finding supports the hypothesis that blast produces a distinct subtype of mild-moderate TBI.

Methods:
22 mild to moderate TBI participants and 19 neurologically healthy, age-matched controls with an orthopedic injury (OI) were recruited for an fMRI and resting connectivity MRI scan. The TBI group was subdivided in two groups: 9 military participants with TBI caused by a blast injury (blast-TBI) and 13 civilians with non-blast TBI (non blast-TBI). Subjects were scanned in an IRB-approved protocol at 3T using a bitebar to reduce head motion, in a 12-ch receive head coil. Scans included T1-MPRAGE, a bilateral complex finger tapping fMRI EPI scan and a resting connectivity fcMRI scan. fMRI and fcMRI EPI protocol: 2x2x4mm voxels, 1954 Hz/pix BW, 31 axial slices, TR/TE/FA=2800/29/80. fMRI and fcMRI data were corrected as described in [6] and the fMRI student’s t-map was coregistered to the connectivity scan. ROIs were drawn for left primary hand motor cortex and were coregistered to connectivity image. A whole-brain seed-based correlation connectivity map was generated using fMRI activation and the left motor ROI and converted to tscores as described in [6]. The correlation map was corrected for global bias by performing an approximate z-transform, by normalizing the mean and standard deviation of the whole-brain tscore distribution. The zscore maps were transformed into standard stereotaxic (Talairach) space and compared between OI and TBI groups, and between the TBI blast and non-blast subgroups by ANOVA.

Results and Discussion:
Functional connectivity to left primary motor cortex in TBI participants (Figure 1a) and controls (Figure 1b) is preserved in cortical primary motor, premotor, supplementary motor areas, but connectivity to the visual cortex, an area with strong synchronous BOLD fluctuations with primary motor cortex at rest, is greatly reduced in TBI compared to controls. Figure 1c shows the difference between the blast-TBI and non blast-TBI groups, showing a distinct increase in medial thalamic connectivity in blast-TBI versus non blast-TBI. This suggests that blast-induced TBI has characteristic differences in functional connectivity from non-blast-induced TBI.

References: