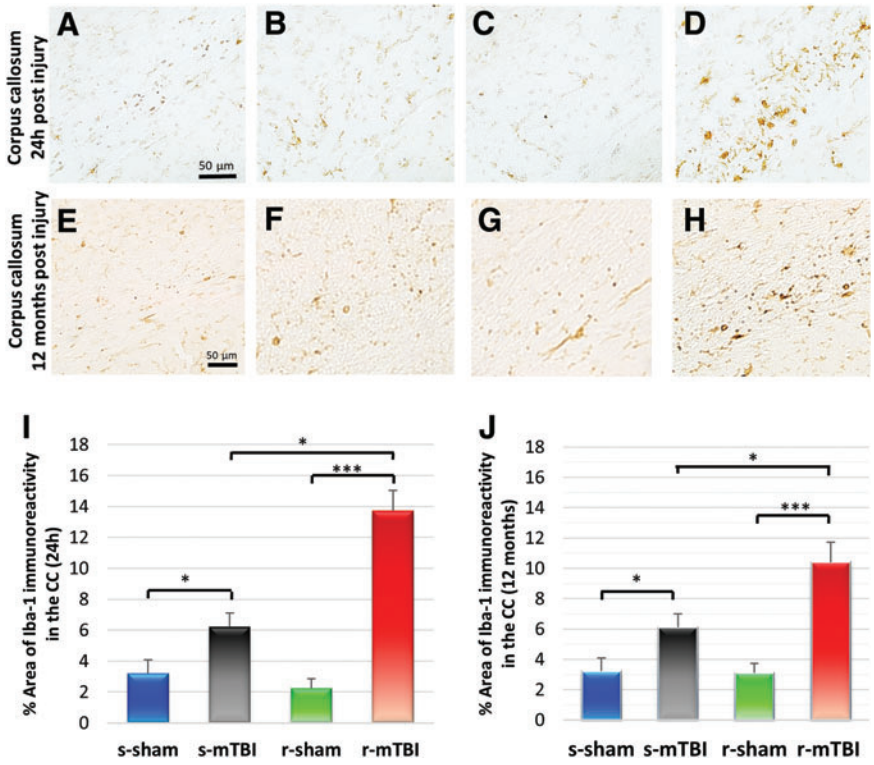

SUPPLEMENTARY FIG. S2. Immunohistochemical labeling for microglia with0 anti-Iba1. Sagittal section of the mouse brain at ±0.4 mm lateral to midline in the corpus callosum (CC) at 24 h post last injury (**A–D**), and 12 months post last injury (**E–H**). There was no evidence of neuroinflammation in the hTau sham animals at any time point (**I,J**). In the single injury animals, both resting and activated mcroglia (with a bushy morphology) were observed at 24 h post-injury in the CC (s-mTBI 6.22 ± 0.9% vs. s-sham 3.22 ± 0.9%; $p < 0.05$; D,**I**). For mice that underwent r-mTBI, immunostaining for Iba-1 revealed clusters of activated microglia in the CC (r-mTBI 13.7 ± 1.3% vs. r-sham 2.21 ± 0.6%; $p < 0.0001$; s-mTBI 6.22 ± 0.9% vs. r-mTBI 13.7% ± 1.3%; $p < 0.05$; D,I). By 12 months post-injury, the single and r-mTBI showed increased microglial activity in the body of the CC when compared with corresponding shams (r-mTBI 10.4 ± 1.3% vs. r-sham 3.01 ± 0.5%; $p < 0.0001$; s-mTBI 6.1 ± 0.9% vs. s-sham 3.4 ± 0.7%; $p < 0.05$; F,H,J).