

Supplementary Figures:

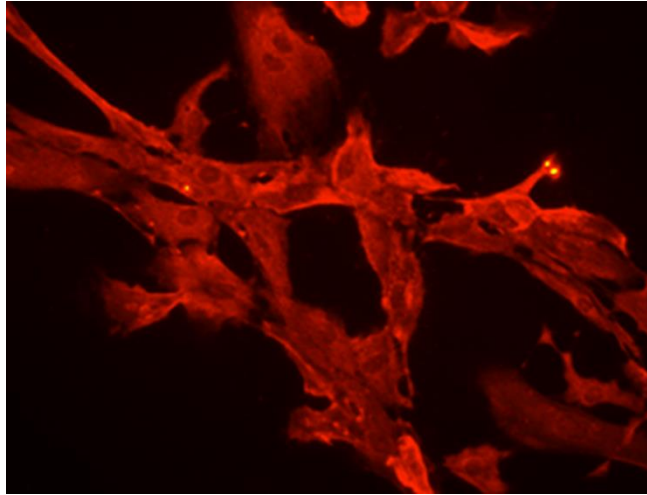


Figure S1: Primary Astrocyte Monocultures Uniformly Express the Astrocyte Marker GFAP. Monocultures were subjected to immunocytochemistry as described in *Methods*. Greater than 90% of cells positively stained for GFAP.

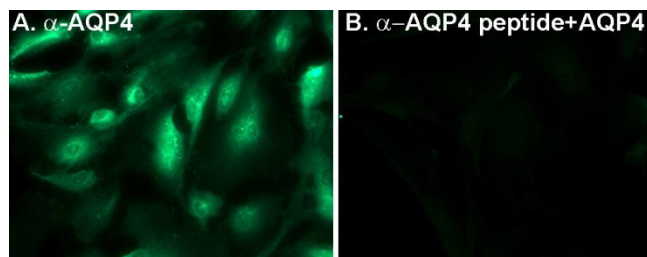


Figure S2: AQP4 is Specifically Labeled in Primary Astrocytes. Fixed astrocytes were pre-incubated with **A)** PBS or **B)** AQP4 blocking peptide before AQP4 immunocytochemistry was performed. Pre-incubation with blocking peptide ablated specific immunolabeling of AQP4 within astrocytes.

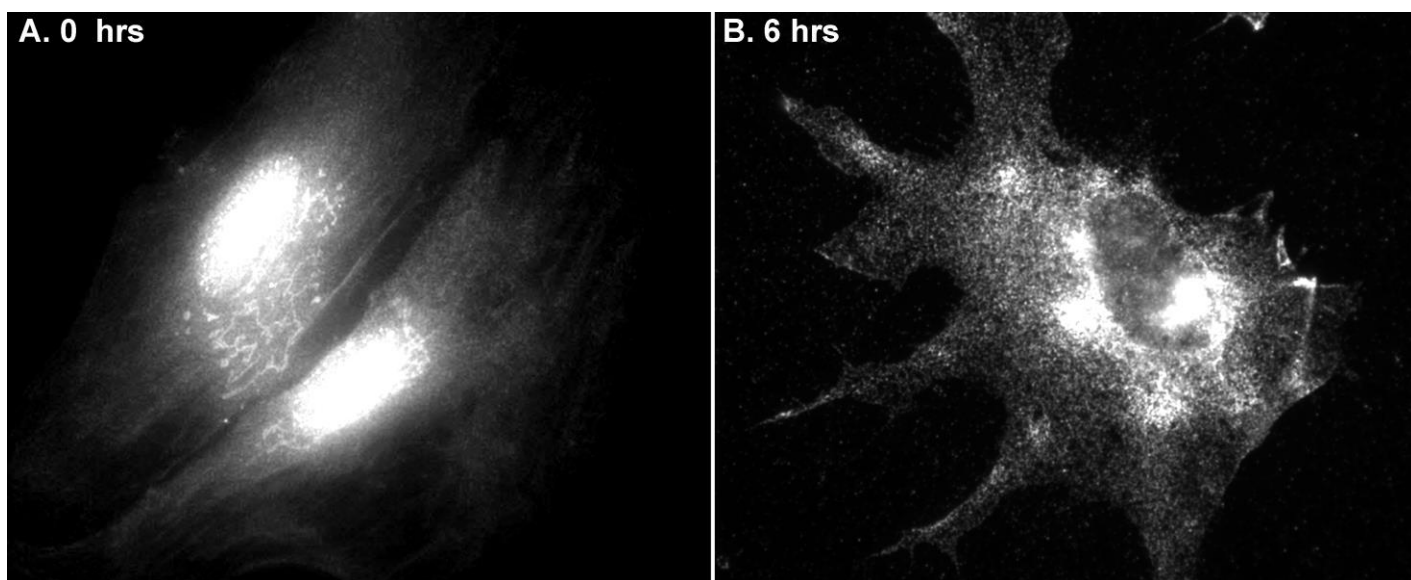


Figure S3: A closer view of AQP4 reorganization in astrocytes after OGD. AQP4 distribution was measured via immunocytochemistry in primary rat astrocytes subjected to **A)** sheer stress alone or **B)** sheer stress plus 6 hrs OGD.

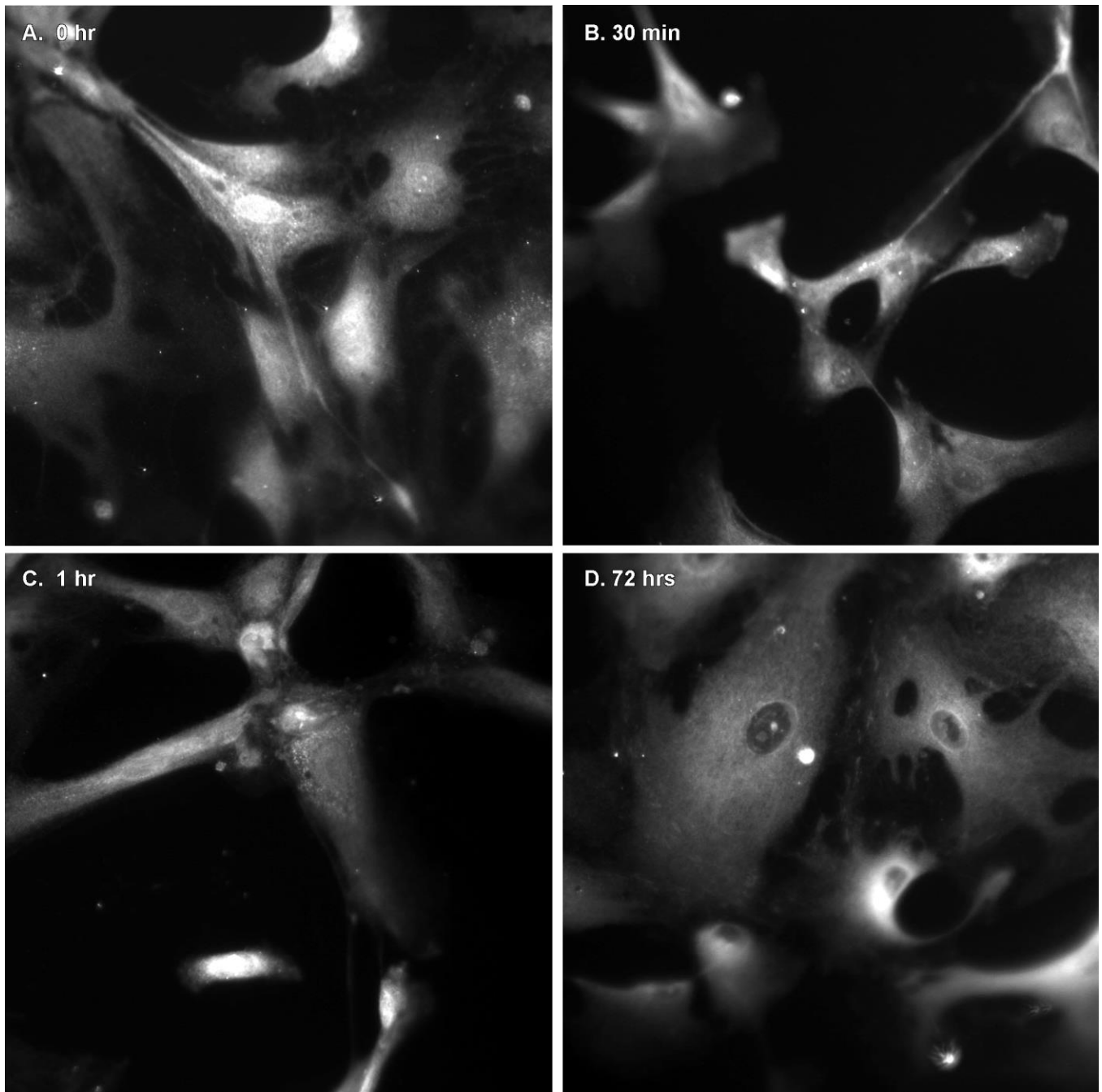


Figure S4: AQP4 Localization Does Not Change in Response to Astrocyte Stretching. Primary astrocytes were subjected to a 5 second stretch to mimic a primary insult after TBI, then were fixed and subjected to immunocytochemistry for AQP4 after **A)** no stretching, **B)** 30 minutes post-stretch, **C)** 1 hour post-stretch, and **D)** 72 hours post stretch.

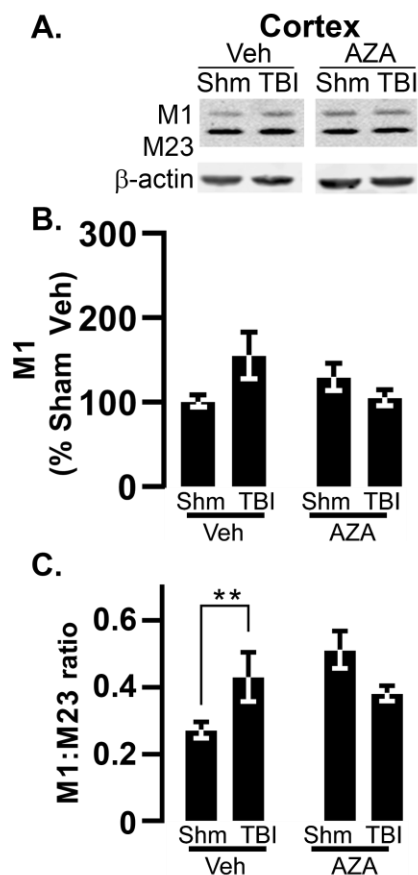


Figure S5: Cortical aquaporin 4 isoform expression at 24hrs after TBI. Mice were subjected to a mild closed-cortical impact, and 30 minutes later were treated with either DMSO (veh) or Acetazolamide (AZA). Tissue microdissection occurred at 24hrs post-TBI. Expression of M1 and M23 were analyzed via SDS-PAGE and western blotting. Results are expressed as means with SEM. ** $p < 0.01$ via ANOVA and Tukey's HSD, $n = 5$ mice/group.

