**Glial role in blood brain barrier disruption and pathology of blast induced traumatic brain injury**

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Blast-induced traumatic brain injury (bTBI) is a major concern for Warfighters exposed to improvised explosive devices (IEDs), breaching charges, and heavy weapon systems during combat and training. However, the injury risk criteria and mechanisms that define brain vulnerability remain unclear. Blood-brain barrier (BBB) disruption is emerging as one of the main pathological outcomes of blast exposure and may initiate secondary injury processes, including vascular damage, neuroinflammation, and neurodegeneration.

Glial cells are central to this pathology, which was shown both in post-mortem brain pathology form the individuals with the history of blast exposure. Astrocytes regulate BBB integrity and respond to injury through reactive astrogliosis, commonly marked by increased GFAP expression, while activated microglia, marked by Iba1, may amplify inflammation through cytokine and oxidative signaling. Pre-clinical blast studies in gyrencephalic models show BBB disruption, microbleeds, GFAP-positive astrogliosis, and microglial activation, which are consistent with findings in blast-exposed human brains showing astroglial scarring, neuroinflammation, and pathology near vessels and gray-white matter interfaces. Interestingly, these outcomes were not consistently observed in lissencephalic animals such as rodents. These findings suggest that astrocytic and microglial responses may link blast-mediated BBB breakdown to persistent neuropathology and may help refine brain-specific injury risk criteria with the appropriate animal model.