BU CTE Center

MAY COGNITIVE KIT WORD SEARCH PUZZLE

N B J W H D N F D A T E G L F M E U H K
W E Q I V W U N X O Q M W D Y E P O I H
L P U D P K H G Z X Q R O T D J B N L Z
L N G R C S U Q C A D R O Z Z R E B R Q
H A C H O N G U H X S A N A M M E F K U
A L R G B T I E Y O U X T T A C B Z N Y
U Z A O L K R N L N V M V T I I V G Y P
K E M D I D E A M A U Y I W N N K F P O
D D L Y R V T U U L F C J R T E F L S T
I G L W J E A S E M A O R E E G J G L X
I A J W R K D H P Y A K H S R O U R E W
A S P A C E O Z E I O B I R F H B I J F
A Z L Y M V X N U B A U J F A T P N A W
I U O I M N Z P S J O E U E C A E P M V
P A L T B U M P O F G R Z F E P M F T I
A S T R O G L I O S I S U P M D S S J L
N G P E Q D K G G Q U B N E T M A T X S
V E T E R A N S T E O R F J N L Z V C R
N O I T A R E L E C C A E P B L T A S I
O X W H D Y E T M E X P F T O I A A W J

Note: The word bank is on the following page

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WORD BANK:

BLAST	KINEMATIC	ASTROGLIOSIS
VETERANS	ACCELERATION	EXPOSURE
NEUROTRAUMA	NEUROBEHAVIORAL	INTERFACE
AXONAL	PATHOGENIC	DORSOLATERAL

This month, the word search clues are from our featured research studies:

Chronic traumatic encephalopathy in blast-exposed military veterans and a blast neurotrauma mouse model

"**Blast** exposure is associated with traumatic brain injury (TBI), neuropsychiatric symptoms, and long-term cognitive disability. We examined a case series of postmortem brains from U.S. military **veterans** exposed to blast and/or concussive injury. We found evidence of chronic traumatic encephalopathy (CTE), a tau protein–linked neurodegenerative disease, that was similar to the CTE neuropathology observed in young amateur American football players and a professional wrestler with histories of concussive injuries. We developed a blast **neurotrauma** mouse model that recapitulated CTE-linked neuropathology in wild-type C57BL/6 mice 2 weeks after exposure to a single blast. Blast-exposed mice demonstrated phosphorylated tauopathy, myelinated axonopathy, microvasculopathy, chronic neuroinflammation, and neurodegeneration in the absence of macroscopic tissue damage or hemorrhage. Blast exposure induced persistent hippocampal-dependent learning and memory deficits that persisted for at least 1 month and correlated with impaired **axonal** conduction and defective activity-dependent long-term potentiation of synaptic transmission. Intracerebral pressure recordings demonstrated that shock waves traversed the mouse brain with minimal change and without thoracic contributions. **Kinematic** analysis revealed blast-induced head oscillation at accelerations sufficient to cause brain injury. Head immobilization during blast exposure prevented blast-induced learning and memory deficits. The contribution of blast wind to injurious head **acceleration** may be a primary injury mechanism leading to blast-related TBI and CTE. These results identify common pathogenic determinants leading to CTE in blast-exposed military veterans and head-injured athletes and additionally provide mechanistic evidence linking blast exposure to persistent impairments in neurophysiological function, learning, and memory."

Interface astrogliosis in contact sport head impacts and military blast exposure

"Exposure to military blast and repetitive head impacts (RHI) in contact sports is associated with increased risk of long-term **neurobehavioral** sequelae and cognitive deficits, and the neurodegenerative disease chronic traumatic encephalopathy (CTE). At present, the exact **pathogenic** mechanisms of RHI and CTE are unknown, and no targeted therapies are available. Astrocytes have recently emerged as key mediators of the multicellular response to head trauma. Here, we investigated interface **astrogliosis** in blast and impact neurotrauma, specifically in the context of RHI and early stage CTE. We compared postmortem brain tissue from former military veterans with a history of blast **exposure** with and without a neuropathological diagnosis of CTE, former American football players with a history of RHI with and without a neuropathological diagnosis of CTE, and control donors without a history of blast, RHI exposure or CTE diagnosis. Using quantitative immunofluorescence, we found that astrogliosis was higher at the grey-white matter **interface** in the **dorsolateral** frontal cortex, with mixed effects at the subpial surface and underlying cortex, in both blast and RHI donors with and without CTE, compared to controls. These results indicate that certain astrocytic alterations are associated with both impact and blast neurotrauma, and that different astroglial responses take place in distinct brain regions."