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Brainstorm: Head Injuries and the NFL, Part 10: The Tau of CTE, Continued

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In our last installment, I wrote about a protein called tau, which necessitated talking about salt. I said that when neurons suffer the types of injury associated with CTE, part of the damage occurs because of a change in salt distribution between the inside of a neuron and its immediate outer exterior.

Put simply, blunt force messes with the cell’s normal briny exchange, eventually causing the disorientation that players experience on the gridiron after head injuries. At the level of the cell, the brine is part of a flooding problem, where salts that are supposed to stay outside the neuron come rushing in — and vice versa.

Happily, the cell has a few tricks up its sleeve to try to keep things stable, especially when it senses that something like this flooding is occurring. Like a tiny seafaring vessel, the nerves turn on tiny pumps in an effort to reverse the flow. These pumps require a great deal of energy, potentially depleting the neuron’s supply, debilitating its normal function for a period of time.

But the pumps don’t always work well, especially if the injury is severe. As the salt balance continues to fluctuate, the growing imbalance triggers a series of molecular events inside the nerve that disturbs its cytoskeleton. As you recall from Part 7 of this series, the cytoskeleton participates in giving the neuron its shape. I mentioned that you can think of the cytoskeleton like the interior scaffolding of a building.

Obviously, if you destroy the integrity of the scaffolding, you will destroy the integrity of the building. Nerve cells are no different. If you destroy their scaffolding — what we call their morphology — you can destroy the cells. That’s exactly what happens when the salt concentrations become altered inside nerves during injury. The cytoskeleton starts to loosen up. Eventually, the neuron loses its shape, then it loses its function.

With all of that salty background in mind, I am ready to talk about tau. The reason is that tau protein is a member in good standing of the cytoskeletal
structure.

For reasons that are not well known, a particularly vulnerable salt-influx target in that cytoskeleton is tau. Why is that a big deal? One of the jobs of tau is to act like a “glue,” helping to hold the nerve’s cytoskeleton together, stabilizing the structure of the cell. When the “foreign salt” floods the neurons, tau is modified in such fashion that it loses its ability to help hold the scaffolding together. That is truly bad news. Eventually the cell collapses in a disorganized molecular heap, leaving piles of modified tau scattered throughout the brain. We call these piles neurofibrillary tangles. These tangles are a hallmark of CTE and can be seen under the microscope. One of the items researchers look for in the tissues of athletes who have donated their brains to science is the presence of these tangles.

Know where else we see tau-laden neurofibrillary tangles? In the brains of Alzheimer’s patients. Some researchers believe that repeated closed-head injuries trigger molecular events in the brain akin to processes observed in Alzheimer’s patients.

As if that weren’t bad enough, we have a third mechanism to discuss, one that involves those microglial cells we discussed earlier. It may go a long way toward explaining why repeated head injuries are so toxic. We will take that subject up next time.

Comments