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Brainstorm: Head Injuries and the NFL, Part 7: What Mops Have to Do With the NFL

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In the last few installments, we discussed some vulnerable regions of neurological real estate that suffer damage in afflicted athletes, and their association with changes in outwardly observable behavior. Unfortunately, this is only a partial list. Enough is now known that I could create three or four more installments of this blog and still not cover everything that is being researched.

We then discussed the mostly tragic behavioral consequences that manifest once the damage shows up. But we left off last time by asking, what exactly does “damage” mean? To understand the answer to that question, we must delve into the microscopic world of the nerve cell. Eventually we will become even more intimate, getting down to the various molecules within individual cells that may mediate some of the behavioral damage I mentioned last time. Only then will we discover the true nature of CTE, and conclude our discussion.

In this installment we will talk about some basic facts about the brain’s cellular biology. In the next, we will talk about how that biology suffers when someone experiences closed-head injuries.

As you may recall from an earlier blog in a different series, I mentioned that nerve cells come in a wide variety of shapes and sizes. Yet they all have features in common, which in that series I likened to a mop. The hairy “business end” of the mop is called the cell body. The extensions that protrude from that cell body are called dendrites. Many nerve cells have a single long extension looking something like a mop’s handle. This longish extension is called the axon. Both axon and cell body are surrounded by an oily membrane called the axolemma, clinging to the neuron like an Olympic swimsuit.

The interior of a nerve cell is a complex broth of molecules embedded in a salty soup. This soup is called the cytosol and is populated not only by small
molecules, but also by large, foreboding structures. Easily the most dominant structure in the cytosol is the cell’s nucleus, which looks like a central dot right in the middle of the cell body. The nucleus contains most of the cell’s command and control functions, mediated through its interior DNA. Smaller, nearly beanlike structures surround the nucleus — and also pepper the entire cytosol. These are called mitochondria. They function like tiny batteries for the cell, generating most of the energy needed for its interior functions.

The nucleus and mitochondria do not simply float around the inside cell, bobbing up and down in a disorganized, random fashion. There is a complex interior “skeleton” inside nerve cells, which not only places these interior structures inside compartmentalized “microenvironments,” but also give the cells their overall size and shape. Logically called the cytoskeleton, this interior scaffolding is itself composed of many molecules. One of the most important of these molecules is a BB-like protein called tubulin. These BB-like proteins join together end to end, forming long lines we call microtubules. Other molecules latch onto these long lines, helping to keep them from falling apart, or, in some cases, disassembling them in a controlled fashion.

These cytoskeletal molecules will play a very important part in our understanding the biological basis of CTE. In the next installment, we will discuss what happens to injured sports stars at the level of brain cell and molecule, with a specific focus on those BB-like proteins and their attendants.

Comments