Audubon High School SMART Team

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Calbindin: A Marker for Synaptic Integration Following Spinal Injury

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Description:

The World Health Organization reports that 250,000 to 500,000 people suffer a spinal cord injury annually. The injury interferes with presynaptic to postsynaptic communication necessary for proper motor function. Neurons do not typically regenerate, rendering damage permanent. To work around this damage, a virus that expresses an axon growth-promoting protein is injected into the motor cortex. This induces regeneration of injured axons and synaptogenesis. However, newly sprouted axon-to-cell connections can be mistargeted. The integration of newly regenerated axons with postsynaptic cells can be seen by studying the expression of postsynaptic cell markers, such as the calcium-binding protein calbindin. Calbindin contains twelve alpha helices, four calcium binding sites, and six EF-hands. Calbindin connects EF-hands by making hydrophobic contacts to amino acids on each arm. Ile19, trp20, and phe23 from the first arm make contact with leu36, leu39, and ala46 on the second hand while an EF-hand loop is made by contacts between asp24 and ala46. The two cystine residues, cys100 and cys219 play an important role in calcium binding in the protein. Calbindin is more heavily expressed in classes of cells found in the ventral portion of the spinal cord, suggesting a role in motor function. The Audubon High School SMART (Students Modeling a Research Topic) Team has designed a model of calbindin using 3-D printing technology to investigate structure-function relationships. Using calbindin as a marker to identify the location of synaptogenesis in regenerating axons can lead to therapies for regaining motor function in individuals suffering from spinal cord injuries.