A Study on Electrophysiological Properties of Medial and Lateral Axons in *Lumbricus* Earthworm

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**RESULTS**

Figure 2. Threshold voltage (V) for medial and lateral axons of *Lumbricus* at various stimulus durations (msec) with approximate rheobase and chronaxie values. *Lumbricus* was prepared by longitudinal dissection and results were recorded using a powerlab analogue-to-diginal converter and nerve chamber.

**DISCUSSION**

1) *Lumbricus* has a faster conduction velocity than most invertebrates of similar diameter because its neurons are myelinated and often compacted (Hartline 2007). Invertebrate axons are encased by glial cells, but do not have compact myelin (Zalc 2006). This myelination allows the blocking of current leakage and occurs in intervals along the axon, leaving unmyelinated regions known as nodes of Ranvier (Hartline 2007). The blocking of current leakage is due to increased membrane resistance at the nodes, which force the action potential to propagate to the next node (Hills 2012). This phenomenon known as saltatory conduction, is vastly faster than a continuous sweep of action potentials over unmyelinated neuron. Additionally, the layers of myelin increase the capacitor distance that separates the charges between the axon membrane, decreasing the capacitance (Hills 2012). The decreased capacitance and increased membrane resistance in combination allows myelinated axon to conduct a faster impulse.

2) Comparing the lateral and medial axons, the medial axon propagated the action potential at a faster rate. Figure 3 showed that medial axons had a conduction velocity of 0.608 m/s while lateral 0.143 m/s. Higher conduction velocity in the medial axon can be attributed to its larger diameter and lower resistance. Larger diameters speed up the signal conduction because local currents are spatially spread out more along the axon. Spreading of local currents means a longer length constant (Hills 2012). The length constant includes a ratio between the Rm and internal resistance (Ri). With increased surface area and cross-sectional area of the axon, Rm and Ri is lowered (Hartline 2007). All in all, this would make the length constant and conduction velocity to increase at a higher rate than the diameter. As seen in figure 1, lateral axons had a higher threshold (320 mV) than medial (163 mV), indicating a greater resistance to current flow. The more time it takes to reach the threshold, the slower the conduction velocity (Hills 2012).

3) Three ionic mechanisms prevent the axon from immediately being re-excited or “backflowing” once stimulated—this is known as the refractory period. The main driving force for the refractory period is the inactivation of sodium gated-channels. This event occurs right after sodium voltage-gated channels are open, allowing sodium ions to rush into the axon as the action potential travels down. The trailing sodium voltage-gated channels is inactivated but still open, preventing any local depolarizations to arise. At this point, there is a charge reversal (positive inside, negative outside axon), preventing any new propagation of action potentials, meaning that no additional sodium ions can enter in. Eventually the inactivated sodium voltage-gated channels close, which allows a new action potential to propagate through.

The second event is the axon’s increased potassium permeability, which occurs right after depolarization, known as the falling phase. This event lasts the longest of the three events until the refractory period has ended (Hills 2012). Here, potassium ions rush into the axon, slowly restoring the membrane potential to resting. During this event, the axon is unable to be re-stimulated, since the membrane voltage is still resetting and has not reached the resting potential. In some tissues, the potassium voltage-gated channels are open long enough to the point where excessive potassium accumulates in the axon, causing an undershoot in membrane potential—lower than resting potential. Any new impulses must have a voltage strong enough to overcome this deficit below the resting potential (Hills 2012).

The last event is like the second. The increase in potassium permeability causes a decrease in membrane resistance due to high number of leakage channels open. This would dissipate any new impulses (positive charges), therefore more current would be needed to depolarize the membrane to threshold (Hills 2012). Inactivated sodium voltage-gated channels, increased K+ permeability, and decreased membrane resistance all contribute to the axon’s refractory period.

**LITERATURE CITED**

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