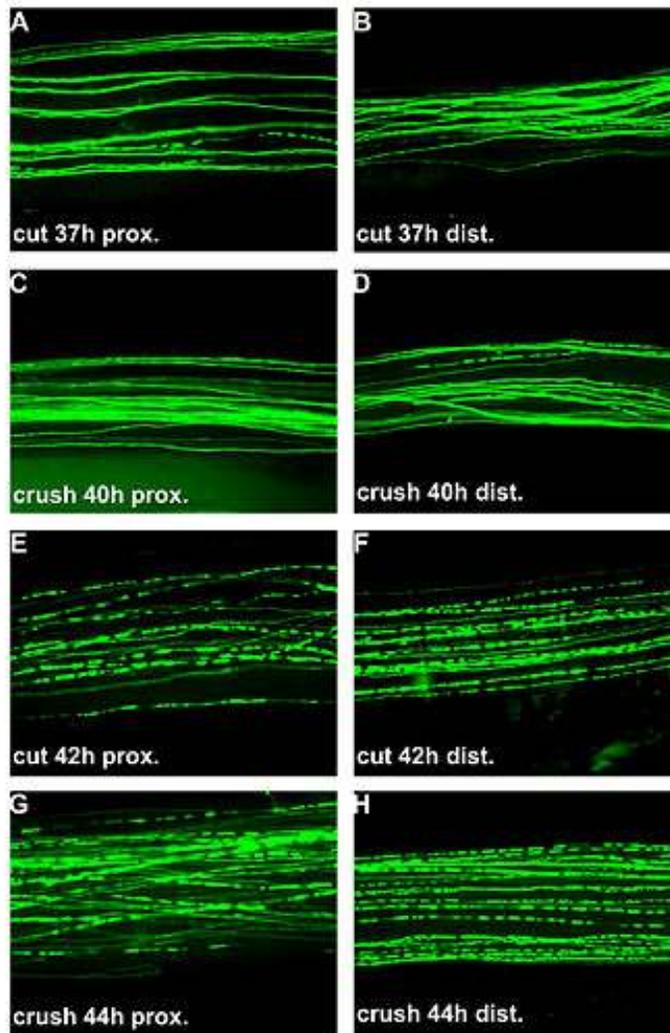


BIO303
The Saylor Foundation's "Wallerian Degeneration"

When a nerve axon is cut, crushed, or frozen, a process called Wallerian degeneration occurs. Wallerian degeneration is also called anterograde degeneration, named after Augustus Waller, an English neurophysiologist (1816–1870), who was the first to describe the degeneration of severed nerve fibers.



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When an axon is cut, the neuron cell body is separated from the distal part of the axon. Wallerian degeneration is the process that results when the axon separated from the neuron's cell body degenerates distal to the injury. Degeneration usually occurs 24–36 hours after a lesion, and prior to degeneration, the distal axon stump may remain electrically excitable.

Wallerian degeneration occurs after axonal injury in both the peripheral nervous system (PNS) and Central Nervous System (CNS). However, there are significant differences in Wallerian degeneration between the two nervous systems.

In the PNS, degeneration initiates by macrophages entering the transected area to remove the myelin and axonal debris. The axonal skeleton disintegrates, and the axonal membrane breaks apart.

The axon's neurolemma (the outermost layer of the neuron made of Schwann cells) does not degenerate and remains as a hollow tube. Within 96 hours after the injury, the Schwann cells that make up the hollow tube synthesize growth factors which attract axonal sprouts to the distal end of the severed axon. These axonal sprouts originate at the proximal end of the severed axon.

If an axon sprout reaches the tube, it grows into it and advances about 1 mm per day (3 cm/month). The tubes provide pathways for the sprouting (regenerating) axons to follow to muscles and skin. The Schwann cells then remyelinate the newly formed axons which eventually reach and innervate the target tissue.

In the CNS, axonal regeneration is much slower. The crucial difference is that in the CNS, including in the spinal cord, myelin sheaths are produced by oligodendrocytes and not by Schwann cells. Myelin debris is more slowly removed in the CNS, because it takes 2–4 weeks post-injury for the macrophages to remove the debris. The myelin debris contains inhibitory factors which slow the regeneration process. Later, astrocytes proliferate into the transected area and form astrocytic scars which block the pathways for axonal growth.

