Microscopic worms could provide key to repairing injured nerves

30 April 2019

The ability to spontaneously reconnect nerves in the nervous system following an injury may sound like science fiction. But it is an ability that does exist – just not in humans. A number of invertebrate species, like the microscopic roundworm *Caenorhabditis elegans*, are able to re-fuse and restore the function of neurons (/brain-basics/brain/brain-physiology/how-do-neurons-work) that have been cut.

It's an ability that researchers, including Professor Massimo Hilliard (https://qbi.uq.edu.au/hilliardgroup), Dr Rosina Giordano-Santini (/profile/826/rosina-giordano-santini), and Dr Casey Linton (/profile/334/casey-linton) from QBI and Dr Brent Neumann from Monash University are exploring in the hope of one day being able to treat nerve injuries, such as paralysis, in people.

Now, they have uncovered key information (http://www.jneurosci.org/content/39/15/2823) on how this process is regulated in a discovery that represents another step forward towards translating this ability from worms to humans.

Reconnecting severed neurons

Neurons communicate using lengthy, rope-like structures called axons (/brain/brain-anatomy/axons-cable-transmission-neurons). In 2015, Prof Hilliard led the team that discovered the ability of *C. elegans* to carry out a process called axonal fusion, where two separated axons reconnect.

"It's a very efficient process," Prof Hilliard said. "Instead of injured nerves having to regrow the full length to the target, they are basically just bridging a gap to rejoin the nerve and allow it to function again."

To undergo axonal fusion, the axon still attached to the cell must first regrow, then position itself in close proximity to its separated axonal fragment. Once the two axons have reconnected, they fuse their membranes to form a cohesive whole with an outer membrane and the inner material of the cell.

It's a process that offers promise as a potential treatment for people with nerve injuries, which can cause life-long disabilities.

Dr Neumann said, "But to harness this fascinating process, we must first understand how it works through investigating the molecular mechanisms that underlie axonal fusion."

A tale of two molecules

The 2015 research into axonal fusion focused on the molecule that kicks off the fusion process: EFF-1. This protein must be present at the membrane of the nerve to merge two axon fragments; it is inactive if located in a compartment within the cell. Now, the team have discovered another key molecule in this process.

"We have found that the protein RAB-5 plays a crucial role in axonal fusion through regulating EFF-1," Dr Linton said. "RAB-5 is a trafficking protein, which is a type of protein responsible for delivering other proteins from their site of manufacture in the cell to where they are needed. RAB-5 controls the level of EFF-1 on the nerve membrane, which has consequences for a nerve's ability to repair itself."

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