Turning back the clock for Schwann cells

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Myelin-making Schwann cells have an ability every aging Hollywood star would envy: they can become young again. According to a study appearing in the May 19 issue of the *Journal of Cell Biology*, David B. Parkinson (University College London, London, UK) and collogues have pinned down a protein that returns the cells to their youth, a finding that might help researchers understand why myelin production falters in some diseases.

Wrapped around neurons in the peripheral nervous system, Schwann cells can “dedifferentiate” into a state in which they can’t manufacture myelin. Reverting to an immature type of cell speeds healing of injured nerves. Researchers knew that the protein Krox-20 pushes immature Schwann cells to specialize and form myelin, but they didn’t know what prompts the reversal. One suspect was a protein called c-Jun, which youthful Schwann cells make but Krox-20 blocks.

Parkinson et al. cultured neurons with Schwann cells whose c-Jun gene they could activate. Turning on the gene curbed myelination, suggesting that c-Jun prevents young Schwann cells from growing up. c-Jun also prodded mature Schwann cells to become youthful again, the researchers discovered. Schwann cells that are separated from neurons normally dedifferentiate, but the team found that the cells remained specialized if c-Jun was missing. They suspect that c-Jun works in part by activating Sox-2, as this protein also inhibits myelination.

The researchers now want to investigate whether c-Jun is involved in illnesses where myelin dwindles, such as Charcot-Marie Tooth disease

and Guillain-Barre syndrome. The results might also provide clues about multiple sclerosis, in which immune attacks destroy myelin in the central nervous system. Unlike Schwann cells, oligodendrocytes, the myelin makers in the central nervous system, can't revert to an immature state. Whether c-Jun affects oligodendrocyte differentiation isn't known.

Source: Rockefeller University

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