Turning down gene expression promotes nerve cell maintenance

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Anyone with a sweet tooth knows that too much of a good thing can lead to negative consequences. The same can be said about the signals that help maintain nerve cells, as demonstrated in a new study of myelin, a protein key to efficient neuronal transmission.

Normal nerve cells have a myelin sheath, which, much like the insulation on a cable, allows for rapid and efficient signal conduction. However, in several diseases - the most well-known being multiple sclerosis - demyelination processes cause the breakdown of this "insulation", and lead to deficits in perception, movement, cognition, etc. Thus, in order to help patients of demyelinating disease, researchers are studying the pathways that control myelin formation and maintenance.

A new study by University of California scientists examines the role of a structural protein, called lamin, in maintaining myelin. They found that, while lamin is necessary in the initial stages of myelin formation, too much lamin promotes myelin breakdown. Further investigation led the researchers to the discovery of a signal that fine-tunes lamin expression. This signal, a microRNA called miR-23, can turn down lamin gene expression, and thereby prevent demyelination due to lamin overexpression.

This new work reported in *Disease Models & Mechanisms* (DMM), dmm.biologists.org, adds another piece to the puzzle that is understanding myelin formation and maintenance. Additionally, the identification of miR-23 as a myelin regulator introduces a new potential

drug target in developing treatments for demyelinating illness.

The report was written Shu-Ting Lin and Ying-Hui Fu at the Department of Neurology, University of California San Francisco. The report is published in the March/April issue of *Disease Models & Mechanisms (DMM)*, a research journal published by The Company of Biologists, a non-profit based in Cambridge, UK.

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