Could tomatoes protect brain cells? Scientists uncover an unexpected clue in a familiar kitchen staple

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Credit: Pixabay/CC0 Public Domain

Envision a world where an ordinary part of your daily diet acts as a robust protector against one of humanity's most devastating neurological

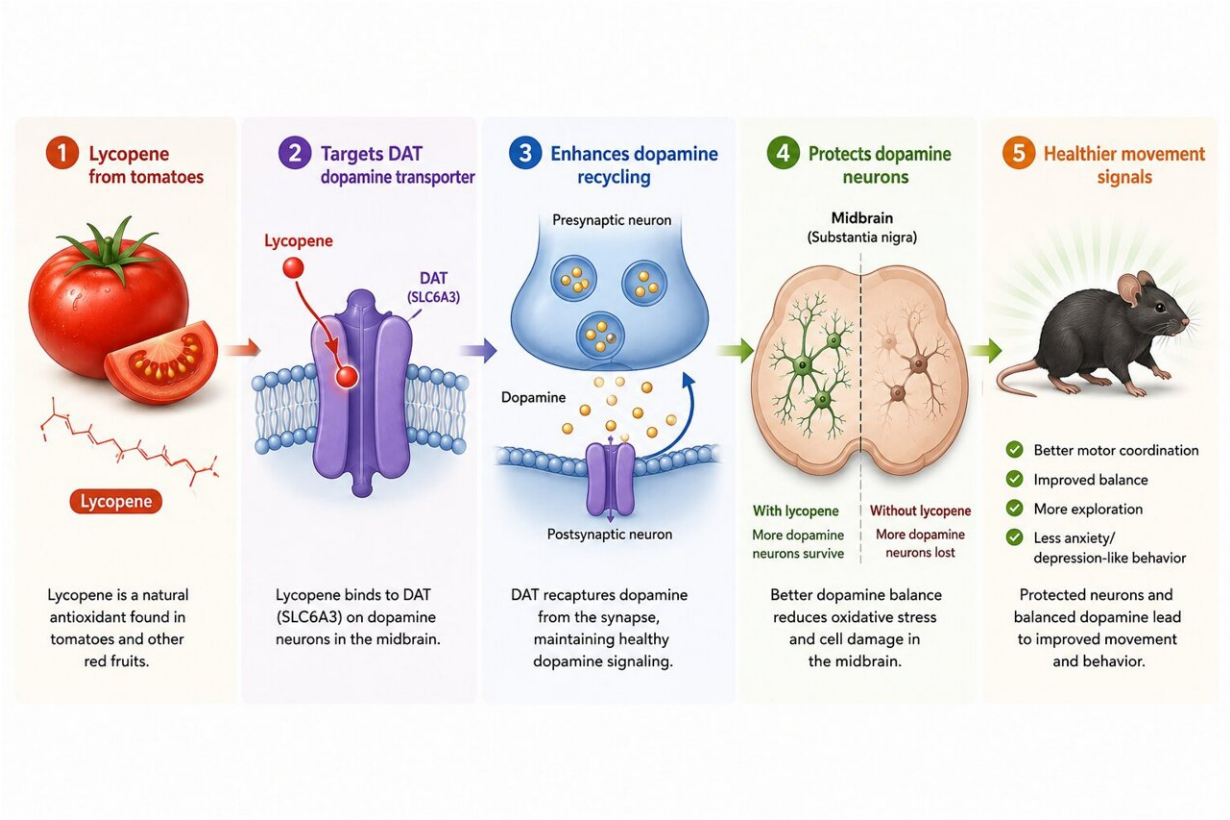
diseases. The search for effective treatments for conditions including Parkinson's disease has been a long journey, with scientists exploring several intricate directions for many years. However, breakthroughs can emerge in the most ordinary places, pointing to nature's own pharmacies.

In a recent publication in the journal *Nutrients*, scientists report a remarkable discovery: Lycopene—a red pigment that gives tomatoes, watermelons and even pink grapefruits their typical color—is a potent neuroprotector.

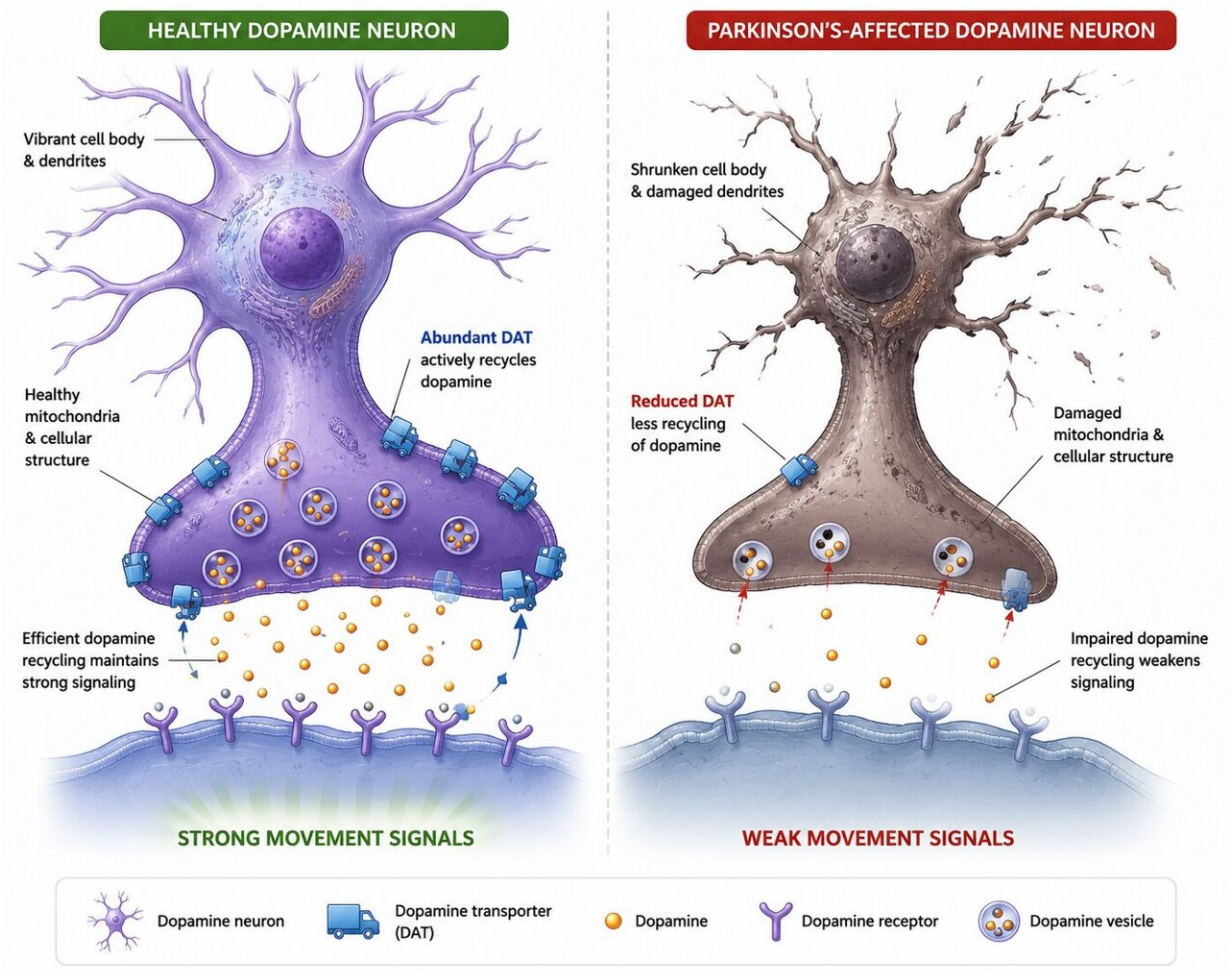
In a study of mice with induced Parkinson's disease, animals fed lycopene daily displayed enhanced motor skills compared with those that had no lycopene in their diet. Specifically, they showed steadier steps and better balance, as well as preserved dopamine-secreting neurons in the midbrain.

The mice got better—but what changed inside the brain?

Researchers used a common mouse model of Parkinson's disease in which animals are treated with MPTP—a toxin that selectively injures dopamine neurons to mimic the disease. Over three weeks, one group of mice ate lycopene mixed into their food, while a control group did not.



Conceptual illustration of the study's proposed pathway linking tomato-derived lycopene to DAT, dopamine recycling, and healthier movement signals in a mouse model of Parkinson's disease. Credit: Generated using AI tools for illustrative purposes



Side-by-side illustration of healthy and Parkinson's-affected dopamine neurons, emphasizing reduced DAT levels and disrupted dopamine recycling in the diseased state. Credit: Generated using AI tools for illustrative purposes

When the team tested the mice on balance beams and open-field mazes, the lycopene group outperformed the controls. They walked more steadily, explored more and showed fewer signs of anxiety- or low-mood-related behavior that Parkinson's can cause.

In the brains of these mice, biochemical tests confirmed that lycopene had preserved many more dopamine neurons in the midbrain. In effect,

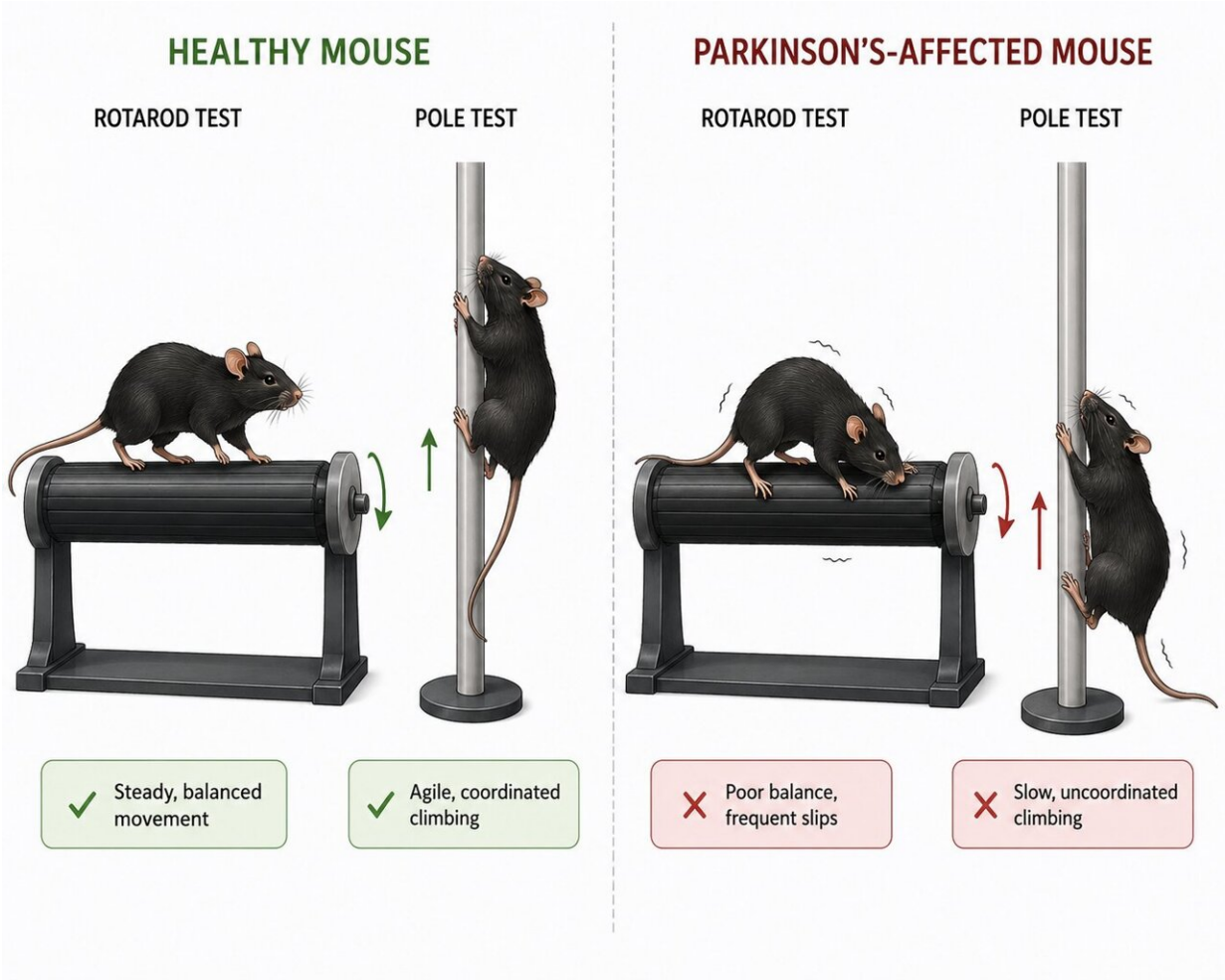
the pigment appeared to slow the disease process: Treated mice had higher dopamine levels and a more normal balance of related chemicals in brain tissue than untreated mice.

A surprising clue points to dopamine's recycling system

Scientists have identified something essential about lycopene and its mechanisms of action. One of the main components of dopamine-producing neurons is the dopamine transporter (DAT). DAT can be thought of as a miniature recycling truck located on the outer surface of neurons. It recycles dopamine by taking it back inside the neuron to be reused. As the number of dopamine-producing neurons decreases in patients with Parkinson's disease, the amount of DAT also decreases.

Remarkably, the lycopene-treated mice showed significantly higher levels of DAT in their midbrain cells than untreated mice. This means lycopene appears to help maintain the brain's natural dopamine recycling system. Further experiments, including computer simulations and lab tests, suggested that lycopene can directly bind to the DAT protein.

As one of the study's authors explained, "We found that LYC increased the expression of DAT/SLC6A3 and formed direct physical binding with the DAT/SLC6A3 protein." This suggests that lycopene might stabilize dopamine signaling by helping these crucial recycling trucks do their job more effectively.



Side-by-side illustration showing how Parkinson's disease impairs movement and balance in mice during behavioral tests commonly used in neuroscience research. Credit: Generated using AI tools for illustrative purposes

At the cellular level, lycopene acted as a protective agent, "relieving motor impairment and reducing midbrain pathological injury" in the Parkinson's mice. This suggests the antioxidant pigment doesn't just mask symptoms but actively shields delicate brain cells.

If a compound can be identified that reliably stimulates this natural recycling system—or similarly protects dopamine neurons—it could

represent a significant stride toward halting the progression of Parkinson's disease.

Why aren't scientists calling it a treatment yet?

But experts caution that these lab findings are just a first step. Mice treated with MPTP develop symptoms much faster than humans do, and a single animal study does not guarantee the same effect in humans. The researchers tested only one dose of lycopene for a few weeks; it's unknown whether long-term intake or different amounts would help or hurt.

Importantly, it's not yet clear how much dietary lycopene actually reaches human brains after a meal. And while this work links lycopene, more DAT and better mouse outcomes, it doesn't prove that DAT is the sole cause of those gains. In fact, the authors admit, "further functional experiments are still required to clarify the precise regulatory mechanism."

Where could this discovery lead next?

Nevertheless, the research opens new possibilities. It will be interesting to conduct additional research to see whether inhibiting the DAT protein would stop lycopene from protecting neurons. Additionally, researchers are looking forward to finding out how lycopene stimulates DAT production and whether these effects are long-lasting. Finally, tests are needed to determine what doses of lycopene would be beneficial to humans.

For now, these findings should be viewed with cautious optimism. It's fascinating that a familiar food pigment might hold neuroprotective potential—a concrete example of how diet-derived antioxidants

sometimes show effects in animals. But this is not a green light to swap your medication for tomato juice. There's a long road from a mouse experiment to a human therapy. For now, lycopene remains a promising lead in the lab, not a proven remedy.

More information: Jun Xia et al, Neuroprotective Effects of Lycopene in Parkinson's Disease Mice: Potential Modulation of DAT/SLC6A3-Mediated Dopaminergic Pathway, *Nutrients* (2026). DOI: 10.3390/nu18142234

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