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Astrocytes found to transfer mitochondria to neurons after stroke

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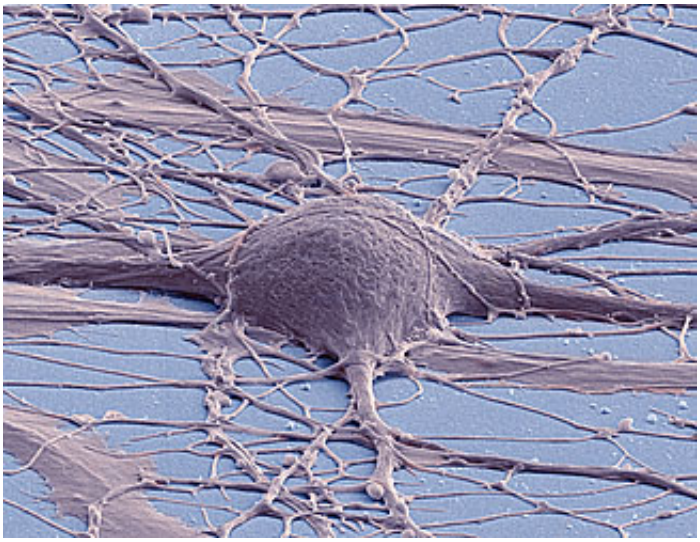

This is a scanning electron micrograph (false color) of a human induced pluripotent stem cell-derived neuron. Credit: Thomas Deerinck, UC San Diego

(Medical Xpress)—A combined team of researchers from Massachusetts General Hospital/Harvard Medical School in the U.S. and Xuanwu Hospital, Capital Medical University in China has found that when neurons in the mouse brain suffer mitochondrial damage astrocytes donate some of their own to help repair them. In their paper published in the journal *Nature*, the team describes how they conducted a series of tests designed to find out whether astrocytes donate mitochondria material and if so, whether it helps to restore health to damaged neurons.

Astrocytes are star-shaped glial cells that surround neurons, providing insulation and support—prior studies have shown that they are involved in carrying out removal of dead material. In this new effort, the researchers started with the results of experiments conducted by a team at Columbia University four years ago that showed that bone marrow stem cells provided mitochondria to damaged lung cells to help them recover—they wanted to know if the same might be true for astrocytes and neurons.

To find out, the researchers engineered mice to produce extra amounts of a signaling enzyme called CD38. They then found that when rodent astrocytes were mixed with them, they expelled some degree of mitochondrial material—neurons added to the mix were then found to absorb some of the mitochondrial material.

The next step was to find out if the same process actually happened in a living animal. They found that it did by causing brain injuries to mice and then injecting the sites with mitochondria they had retrieved from astrocytes—microscopic analysis showed the neurons had, indeed, absorbed the material and that as a result, the neurons were healthier than were injured cells that had not received injections.

The researchers also wanted to know if CD38 signaling was necessary for the process to work—to find out, they injected material that interfered with its function into test mice—those with such injections were found to have less astrocyte-donated mitochondrial material in their neurons than did those that did not receive such injections, which suggest that it is a necessary part of the process.

The overall results by the team suggest that human stroke patients might benefit from CD38 injections or drugs that cause the body to produce it, but the researchers are quick to point out that the protein is very active throughout the body, which means such therapies could cause a large

number of unknown side effects. More study is needed, but the findings do offer hope for treating such injuries and perhaps maladies such as Parkinson's disease.

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