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Wound Healing at a Smaller Scale: Study Reveals for the First Time That Membrane Tension Regulates Cellular Repair

Similar to a small cut that heals itself after a short time, individual cells in our body suffer "wounds" in their protective layer, the cell membrane, every day and have to repair them again. Researchers at the University of Münster have now identified the processes that enable the precise closure of such membrane injuries. Their findings were recently published in the journal "Advanced Science".

One moment of carelessness and bang - you have cut your finger. It bleeds, but after a while the wound heals by itself. Every day, individual cells in our body also suffer "wounds" in their protective layer, the cell membrane, and have to repair them again. Researchers at the Faculty of Medicine at the University of Münster have now identified the processes that enable the precise closure of such membrane injuries. Their findings were recently published in the journal "Advanced Science".

For their experiments, the team led by Prof. Volker Gerke from the Institute of Medical Biochemistry studied endothelial cells, which line the blood vessels. "We know that endothelial cells frequently experience membrane damage caused by changes in normal blood flow, and these cells must repair the damage to prevent cell death. To examine this process in detail, we used a laser to create controlled defects in the cells and observed their repair in real-time under the microscope," explains Dr. Nikita Raj, the study's first author.

In an earlier study, the researchers demonstrated that "early endosomes," which primarily function to transport substances into the cell, fuse with the cell membrane during wound closure to seal the damaged area. However, this fusion by itself is insufficient to complete repair, as the added material creates an uneven, scar-like surface on the membrane. "At the site where early endosomes seal the wound, the membrane becomes loose, leading to a decrease in membrane tension. We were able to show for the first time that this change in tension serves as a signal, triggering the reuptake of excess membrane material into the cell," explains Prof. Volker Gerke. This reuptake restores the normal membrane tension which is necessary for maintaining cellular function.

These repair mechanisms are not limited to endothelial cells but are relevant to a wide range of cell types and have potential significant clinical implications. "Impaired plasma membrane repair plays a crucial role in the development of neurodegenerative diseases, muscle degradation, and aging," says Dr. Raj. Looking ahead, she adds, "Our fundamental insights into cellular repair processes are essential for understanding various diseases. In future studies, we aim to investigate how these mechanisms are disrupted in specific disease conditions, such as atherosclerosis."

The project was initiated with a €5,000 grant from the "Pilot Projects" program of the Cells in Motion Interfaculty Centre at the University of Münster, which supports early-stage projects by young scientists. The study was conducted in collaboration with Prof. Timo Betz from the University of Göttingen (formerly at the University of Münster), Prof. Britta Trappmann from TU Dortmund (formerly at the Max Planck Institute for Molecular Biomedicine, Münster), and Prof. Bart Jan Ravoo from the Center for Soft Nanoscience at the University of Münster.

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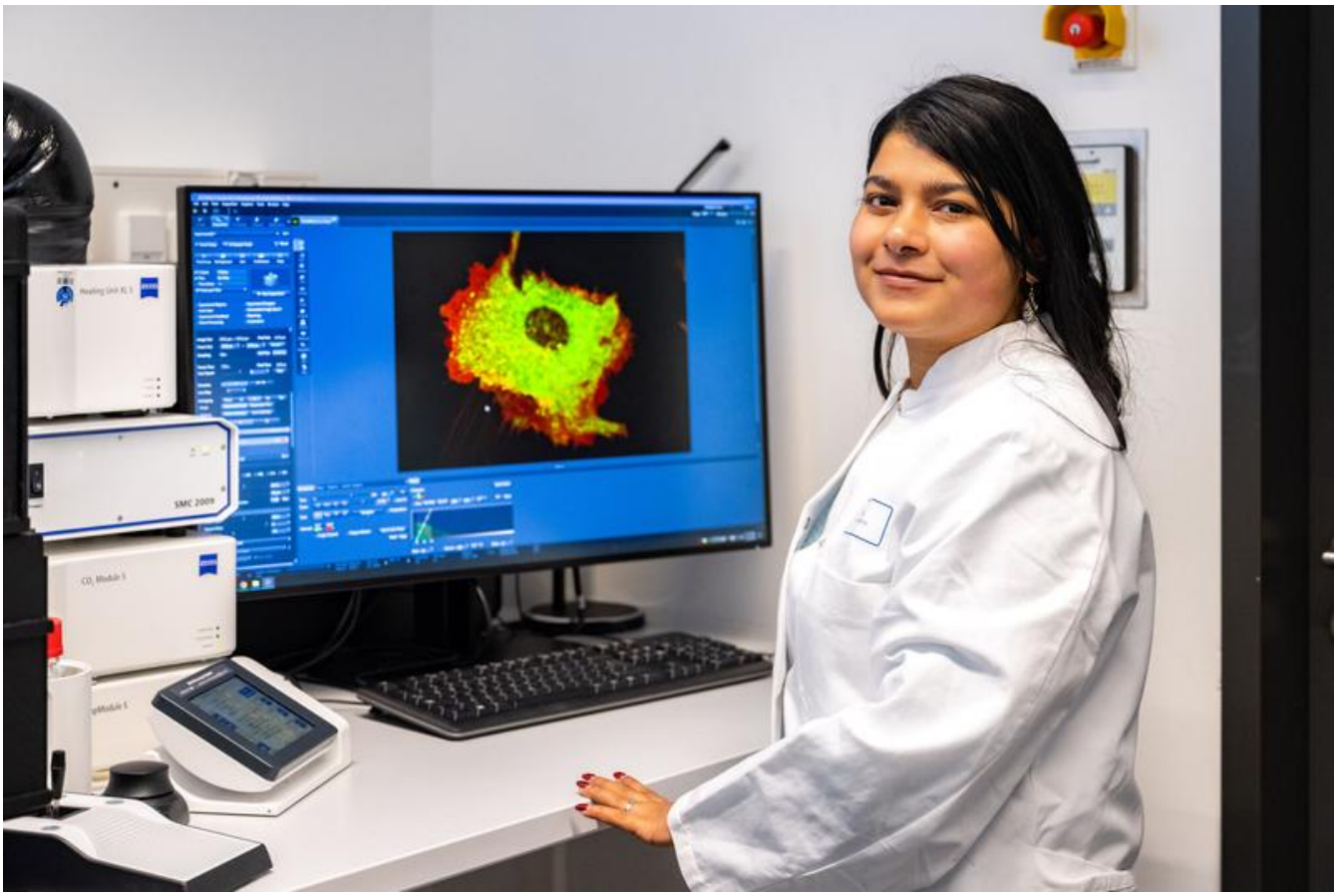

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