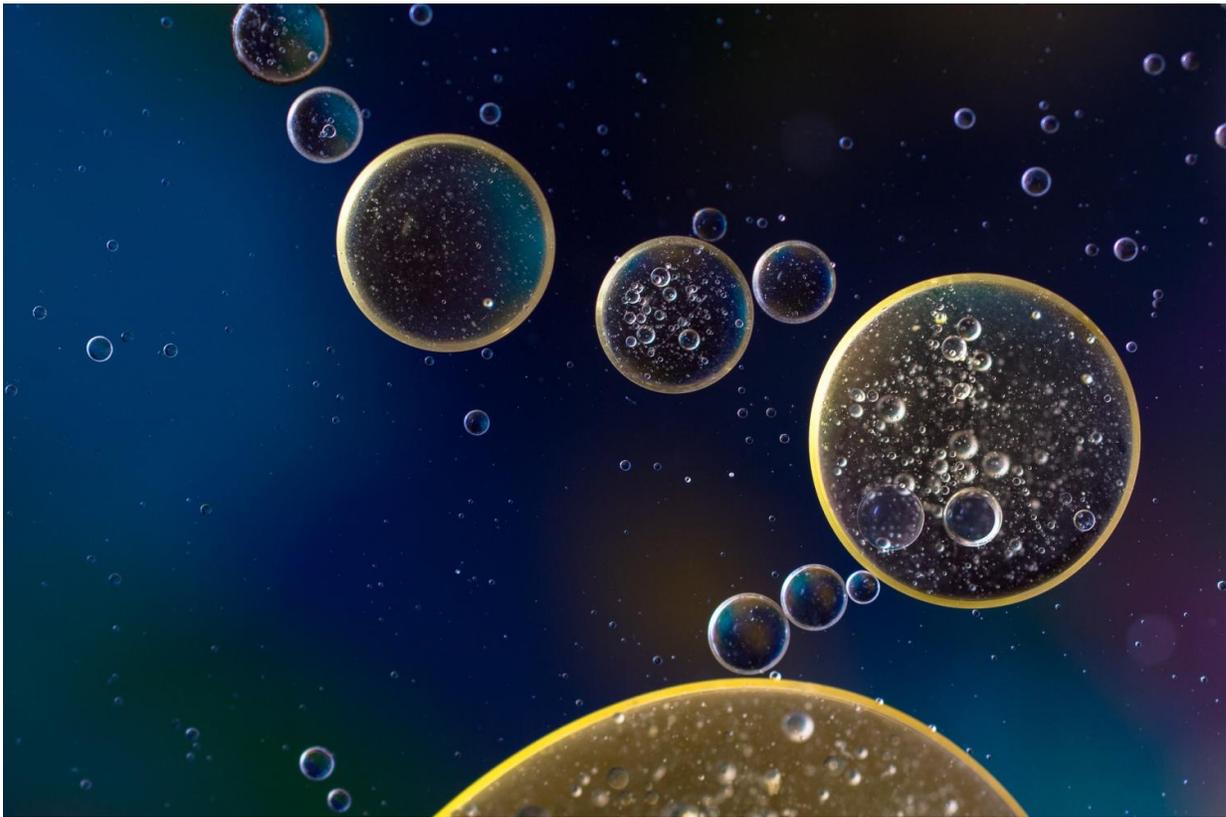


# Immune find could aid stem cell therapy quest

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A discovery of how stem cells are protected from viruses could inform the development of therapies for use in medicine, research suggests.

The finding could help research aimed at boosting the [immune response](#) of [stem cells](#)—early stage cells with the potential to become specialised tissues—for use in treating disease or damaged tissues.

The research identified ways to switch on a key part of the immune system that protects against viruses in stem cells, known as the interferon response.

Researchers from the University of Edinburgh studied stem cells from mouse embryos to understand how stem cells can develop resistance to viruses, before they become specialised cells.

The team discovered a protein—known as mitochondrial antiviral signalling protein (MAVS) - that switches on this immune response in stem cells.

A [small molecule](#)—known as miR-673—was found to regulate when the MAVS protein is turned on and off.

When miR-673 was removed from in stem cells in the lab, production of the MAVS protein was restored, switching on the anti-viral response.

The same mechanism is likely to operate in humans, researchers say. This [antiviral response](#) may be absent from [embryonic stem cells](#) as it can disrupt development.

Researchers hope that their findings will make the use of stem cells more efficient, to one day be given to patients to replace cells lost or damaged by degenerative diseases such as Parkinson's or diabetes. The study, published in *eLife*, was funded by Wellcome.

Jeroen Witteveldt, of the University of Edinburgh's School of Biological Sciences, who took part in the study, said: "Unveiling how this crucial

antiviral mechanism is switched off, and methods to switch this back on in a controlled manner, could make stem cell therapies much more efficient."

**More information:** Jeroen Witteveldt et al, MicroRNA-deficient mouse embryonic stem cells acquire a functional interferon response, *eLife* (2019). [DOI: 10.7554/eLife.44171](https://doi.org/10.7554/eLife.44171)

Provided by University of Edinburgh

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