Horizon
The EU Research & Innovation Magazine

European Commission

Scientists recruit the cell's own waste system to heal diabetic wounds

Diabetic foot ulcers can lead to serious health complications. EU-funded researchers have developed a new therapy to tackle the runaway inflammation that makes these wounds so hard to heal.

14 July 2026 - By MICHAEL ALLEN

Diabetes affects more than 800 million people worldwide and can cause a range of serious complications. Among the most challenging are diabetic foot ulcers – chronic wounds that resist healing and can lead to amputation or death if not treated effectively.

"Diabetes is a very complex disease, causing a number of serious complications that affect patients' lives in many ways," said Juan Ruiz-Constantino, CEO of Spanish biotechnology company Lincbiotech. "One of the most complex is diabetic foot ulcers."

Ruiz-Constantino is leading an EU-funded research project called APTADEGRAD to address the biological reason these wounds resist healing. The project is due to run until the end of 2027.

The researchers from Belgium, Portugal, Spain and the UK have developed a new type of therapy designed to improve the way diabetic wounds are handled. Instead of simply cleaning and dressing the ulcers, which is the current standard of care, this new therapy targets the runaway inflammation that can prevent wounds from closing.

New treatments needed

A key challenge with diabetes is peripheral nerve damage and impaired blood circulation. Damaged nerves mean that wounds on the feet can initially go unnoticed, while poor circulation hinders healing.

Around a quarter of people with diabetes will develop a foot ulcer at some point, according to APTADEGRAD's project data. A significant proportion will result in some form of amputation.

The mortality rate is also striking. The project data indicates that around 5 % of patients die within a year of developing a diabetic foot ulcer, rising to 42 % after five years.

The economic burden is substantial. Germany alone carries out an estimated 34 500 diabetes-related amputations every year, while diabetic foot ulcers and amputations cost the UK's National Health Service the equivalent of EUR 1.1 billion annually.

Yet despite the scale of the problem, there is still no approved therapy that directly addresses the disease mechanisms driving diabetic foot ulcers.

"You are treating the symptoms, but you don't have an approved drug for a disease-modifying approach," explained Ruiz-Constantino.

Why diabetic wounds don't heal

Inflammation normally helps fight infection and coordinate tissue repair. In diabetic wounds, however, inflammatory molecules can build up to excessive levels, preventing the wound from progressing through the normal healing process.

The researchers have identified several important drivers of this response, including proteins known as IL-1α, its receptor IL-1R1 and MMP-9.

Rather than blocking them altogether, Ruiz-Constantino and his colleagues want to remove excess amounts of them. Their solution relies on lysosomes – tiny structures inside cells that act as the body's recycling system.

"The lysosomes are part of the quality control system we have inside our cells," Ruiz-Constantino explained.

The idea is to capture proteins outside the cell, transport them inside and send them to this cellular recycling system, restoring healthier protein levels and a more balanced inflammatory response.

Chimeras: capture and destroy

To activate this waste removal process, the team has developed a new class of compounds called lysosome-targeting chimeras, or LYTACs.

These molecules work like miniature delivery systems. One end captures an inflammatory protein, while the other binds to receptors on the cell surface. This prompts the cell to pull the entire complex inside and send it to the lysosome for destruction.

This allows researchers to reduce protein levels in a controlled way rather than eliminate them completely. The aim is to maintain enough of the proteins to support healing while preventing the excessive inflammatory response that keeps wounds open.

The idea behind LYTACs is still relatively new. When the researchers first proposed the concept a few years ago, they had a strong scientific rationale, but limited experimental evidence. Since then, the APTADEGRAD researchers have made substantial progress.

The research team has designed and synthesised the compounds, demonstrated how they work in laboratory studies and scaled up production for further testing.

Dr Holly Wilkinson, a wound healing specialist at the University of Hull, UK, has been helping to evaluate the technology.

In diseases such as rheumatoid arthritis, psoriasis and Crohn's disease, monoclonal antibody therapies have transformed treatment by blocking inflammatory proteins. Similar approaches have been explored for diabetic wounds, but with limited success.

"Antibodies bind to and block a protein, but they don't remove it from the environment. The APTADEGRAD approach does actually remove that protein from the environment, and this is what we think will be more beneficial for tackling the wound inflammation," explained Wilkinson.

Once the chimera has done its job, the whole complex – drug and protein together – is broken down inside the cell, which should also reduce the risk of side effects. This is particularly important in patients with complex illnesses such as diabetes.

Promising early results

The results so far are encouraging. In diabetic wound models, the compounds reduce inflammation and accelerate healing. In many cases, they perform as well as or better than antibody-based approaches targeting the same proteins.

"We see that the treatments accelerate wound healing and dampen inflammation," Wilkinson said. "A lot of the time we see effects that are as good as or better than antibody treatments."

The trials have so far focused on injections administered under the skin near ulcers, but the team has also developed a hydrogel formulation that can be applied directly to wounds, allowing the medicine to be released gradually over time.

Both the hydrogel and injection-based approaches are designed to maximise local effects while minimising unwanted side effects elsewhere in the body.

Wider implications

Although diabetic foot ulcers remain the immediate focus, the possibilities could extend much further.

If successful, the technology could eventually be adapted to treat other chronic wounds and inflammatory diseases, while also providing some of the first real-world evidence for the broader medical potential of LYTAC-based therapies.

The next step will be generating enough evidence on safety and effectiveness to support the first human clinical trials, which Wilkinson hopes could begin around 2030.

"If we could get something into the clinic that demonstrates effectiveness at healing diabetic wounds, it would be absolutely game changing," she said.

For millions of people living with diabetes, a treatment that tackles the root causes of these wounds rather than simply managing their consequences could represent a long-awaited breakthrough.

Research in this article was partly funded by the European Innovation Council (EIC). The views of the interviewees don't necessarily reflect those of the European Commission. If you liked this article, please consider sharing it on social media.

More info

- APTADEGRAD (CORDIS)
- APTADEGRAD project website

- EU health research and innovation