

# How a rabbit virus learned to infect hares

## New research findings offer clues to how large DNA viruses adapt, evolve and sometimes inspire new therapies

By Richard Harth, ASU News  
October 6, 2025

Myxoma virus, or MYXV, is a type of poxvirus — a family of large DNA viruses that include smallpox and monkeypox.

Myxoma virus does not infect humans, and in its original American rabbit hosts, it causes only mild skin growths. But when introduced into European rabbits in the 1950s, it triggered a deadly disease, killing millions of rabbits.

Recently, scientists were surprised to see a new form of the virus jump species to infect Iberian hares, causing high death rates. Understanding how MYXV overcame species barriers could shed light on how poxviruses in general adapt and emerge as new threats.

The new study shows that MYXV gained a handful of new genes that act like molecular keys — unlocking the cells of a previously resistant species. This rare glimpse into viral evolution reveals how a pathogen can adapt to a new host by subtly reshaping its genetic toolkit.

"We still need to uncover the functions of many MYXV immune modulators and host range factors, not only to understand the biology of this virus, but also to develop better (cancer-fighting) oncolytic viruses," according to Rahman Masmudur, corresponding author of the study, which appears in the journal [Viruses](#).

Masmudur is a researcher with the [Virginia G. Piper Center for Personalized Diagnostics](#) and the [School of Life Sciences](#) at Arizona State University.

### Dodging defenses

Poxviruses, including MYXV, are master manipulators of the immune system. About half of their genes are devoted to thwarting the host's immune defenses. This genetic arsenal lets them replicate efficiently and sometimes cross into new hosts. Because MYXV can infect a wide variety of cancer cells in the lab, it is also being explored as a potential cancer treatment in humans.

Poxviruses such as MYXV attach to molecules on cell surfaces that are common across many animal species. This makes the initial stages of infection — binding and entry — relatively easy across species. The real barrier lies inside the cell, where the virus must evade immune defenses

such as interferons and programmed cell death to replicate successfully.

The myxoma virus carries dozens of protein tools to disarm the immune system. Some block the signals that summon immune cells; others dampen inflammation, and still others keep infected cells alive long enough for the virus to reproduce. Together, these tricks hold the body's defenses at bay just long enough for the virus to spread.

Many of these proteins act as host-range factors, determining which animals the virus can infect. In rabbits, MYXV has evolved a full toolkit to slip past their defenses. In hares, new or altered proteins appear to open access to cells that were previously resistant. This combined strategy helps explain why MYXV is deadly in rabbits but far weaker or harmless in other animals.

## **A virus' expanding reach**

Scientists have long known that poxviruses adapt through “genomic accordions” — cycles of gene duplication and contraction that let them test mutations until a fitter version emerges. What's new in this study is how that process played out in real time.

In 2018, when MYXV leapt from rabbits to Iberian hares, researchers found the virus had picked up four new genes. One of them, M159, acts like a key that unlocks hare cells. This gave MYXV the new ability to replicate in hares while still retaining its long-known lethality in rabbits.

This natural experiment provides rare, direct evidence of how acquiring or duplicating genes can give a virus the power to invade a new host species.

## **Implications and risks**

The authors highlight that a species' immune system is uniquely shaped by its evolutionary history of infections. This explains why most viruses are host specific, but also why spillovers into new species do sometimes occur — especially between closely related species with similar immune landscapes.

MYXV's jump to hares underscores the need for ongoing wildlife surveillance to detect and understand these shifts early.

Beyond wildlife health, understanding MYXV's immune-modulating proteins could help scientists design new treatments. For example, viral proteins that evolved to evade immune defenses might inspire new anti-inflammatory drugs or cancer therapies.

Studying how MYXV interacts with the innate immune system can help us better understand human immunity and point to vulnerabilities in other poxviruses, such as mpox.

The study presents MYXV as a model system for studying how poxviruses evolve, evade immune defenses and occasionally jump species. While researchers have identified many key viral proteins, much remains unknown about their precise mechanisms. Filling these gaps could not only protect wildlife but also help researchers develop new biomedical tools.

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This story originally appeared on [ASU News](#).

## Main image



A new study reveals how the myxoma virus, once deadly only to rabbits, evolved the genetic tools to infect Iberian hares. By acquiring just a few key genes, the virus learned to dodge a new host's immune defenses — offering rare insight into how viruses jump species and adapt. The research, led by scientists at Arizona State University, could help us better understand viral evolution and even inspire new approaches to treating disease. Stock photo

## Text image(s)



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