

# Immune system maintains a memory of past infections by priming genes for future encounters

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Our ability to fight off recurrent infections, such as colds or flu, may lie in the 'immunological memory' found in a newly discovered class of gene regulatory elements, according to research from the University of Birmingham, supported by the BBSRC and Bloodwise.

The research, published in *The EMBO Journal*, identifies one way in which the [immune system](#) is able to provide a quick and successful response to infections that the body has previously encountered, ensuring that long term immunity is built up.

The team, led by Professor Peter Cockerill, demonstrated that a single cycle of activation of the T cells within the immune system leaves behind imprints in the chromosomes within these [immune cells](#). This imprinting occurs at the genes that need to be switched back on as soon as immune cells are reactivated. They propose that this forms the basis of a long-term memory which allows for an immediate response when the body encounters an [infection](#) and T cells are activated for a second time.

Rather than immune cells remaining 'switched on' permanently to [fight infection](#) continuously, they return to a dormant state but are altered by the initial infection and remain in a partially active state primed to combat any recurrence.

Professor Cockerill explained, "The initial immune response switches on certain regions within chromosomes of previously inactive T cells to leave them in a more open structure so that they can then sit poised, ready to respond much faster when activated again in the future."

Being able to silence the immune system until it is required to fight infection is also vitally important, else there would be a risk of damaging cells that are part of the host. The team identified a mechanism that allows cells to remain poised without producing the molecules associated with inflammation that are used to fight infection. If this tight control breaks down then it can be the cause of a number of inflammatory or autoimmune disorders, when [healthy cells](#) are targeted as if they were foreign.

**More information:** *EMBO*

*Journal*, [dx.doi.org/10.15252/emboj.201592534](https://doi.org/10.15252/emboj.201592534)

Provided by University of Birmingham

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