# A small cut with a big impact

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Diseases and injuries trigger warning signals in our cells. As a result, genes are expressed and proteins produced, modified or degraded to adapt to the external danger and to protect the organism. In order to be able to produce a particular protein, the corresponding DNA segment, the gene, needs to be expressed and translated. The DNA is localized in the cell nucleus, and exists as a long string that is coiled and bound by proteins. ARTD1 is one such protein, and therefore has the potential to regulate the expression level of genes through its interaction with DNA.

If cells detect warning signals or foreign bodies like bacteria and viruses in their surroundings, the expression profile of genes changes and an inflammatory response is triggered. To induce changes in gene expression, ARTD1 is removed from particular sites of the DNA. The process by which this is brought about has, until now, remained elusive. The team headed by Professor Michael O. Hottiger from the Institute of Veterinary Biochemistry and Molecular Biology at the University of Zurich has now discovered how ARTD1's DNA recruitment is regulated during inflammation, thereby influencing gene expression and subsequently inflammation.

## Molecular scissors

As the researchers demonstrate in *Molecular Cell*, ARTD1 is cut into two pieces by molecular scissors, the protein caspase 7. Upon cleavage, these pieces can no longer bind to the DNA, thus allowing for more efficient gene expression.

The cleavage of proteins by caspase 7 was so far mainly associated with cell death. "The cleavage of ARTD1 by caspase 7 during inflammation constitutes a new biological function. It permits a new understanding of inflammatory responses and, in the longer term, may lead to the development of new anti-inflammatory drugs," explains Professor Hottiger. The results are of considerable importance because inflammation underlies most diseases, including cancer, immune disorders or metabolic syndrome.

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