

# Rare parasitic fungi could have anti-inflammatory benefits

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Scientists at The University of Nottingham have discovered that a rare parasitic fungus that lives on hibernating caterpillars in Tibet could have a role to play in anti-inflammatory drugs for conditions such as asthma.

Caterpillar fungi (*Cordyceps*) are rare parasites found on hibernating caterpillars in the mountains of Tibet. For centuries they have been highly prized as a [traditional Chinese medicine](#) - just a small amount can fetch hundreds of pounds.

Scientists at The University of Nottingham have been studying how this fungus could work by studying cordycepin, one of the drugs found in these mushrooms. They have already discovered that cordycepin has potential as a cancer drug. Their new work indicates that it could also have anti-inflammatory characteristics with the potential to help sufferers of asthma, [rheumatoid arthritis](#), renal failure and [stroke damage](#).

The research, published today in the academic journal *RNA*, was led by Dr Cornelia de Moor in the School of Pharmacy. It shows that cordycepin reduces inflammatory gene products in airway smooth muscle cells – the cells that contract during an [asthma attack](#).

Several studies have suggested that cordycepin could be an effective drug for a variety of conditions, including cancer, stroke, [kidney damage](#) and inflammatory lung disease but until now it was unclear how cordycepin could bring about so many different beneficial effects at the

cellular level.

Dr de Moor said: "We have shown that cordycepin reduces the expression of inflammatory genes in airway [smooth muscle cells](#) by acting on the final step in the synthesis of their messenger RNAs (mRNAs) which carry the chemical blueprint for the synthesis of proteins. This process is called polyadenylation. Commonly used anti-inflammatory drugs either work much earlier in the activation of inflammatory genes, such as prednisone, or work on one of the final products of the inflammatory reaction (e.g. ibuprofen). These findings indicate that cordycepin acts by a completely different mechanism than currently used anti-inflammatory drugs, making it a potential drug for patients in which these drugs don't work well.

"However, it is a surprise that cordycepin does not affect the synthesis of mRNAs from other genes, because nearly all mRNAs require polyadenylation."

Dr de Moor's research suggests that this is because inflammatory genes can be very rapidly induced and that cordycepin has its many and varied effects by altering the synthesis of other classes of rapidly induced genes as well. If this is true it could be said that cordycepin slows down the rapid cellular responses to tissue damage and may work by preventing the over-activation of these responses which are associated with conditions such as asthma, rheumatoid arthritis, renal failure, cancer and stroke damage.

However, it also indicates that cordycepin could have adverse effects on normal wound healing and on the natural defences against infectious diseases.

Dr de Moor said: "We are hoping to further investigate which genes are more dependent on polyadenylation than others and why this is the case,

as well as test the effect of cordycepin on animal models of disease. Clinical testing of cordycepin is not in our immediate plans, as we think we first have to understand this drug in more detail before we can risk treating patients with it."

**More information:** A copy of the paper is available online at [majournal.cshlp.org/content/e ... 91.112.full.pdf+html](http://majournal.cshlp.org/content/e...91.112.full.pdf+html)

Provided by University of Nottingham

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