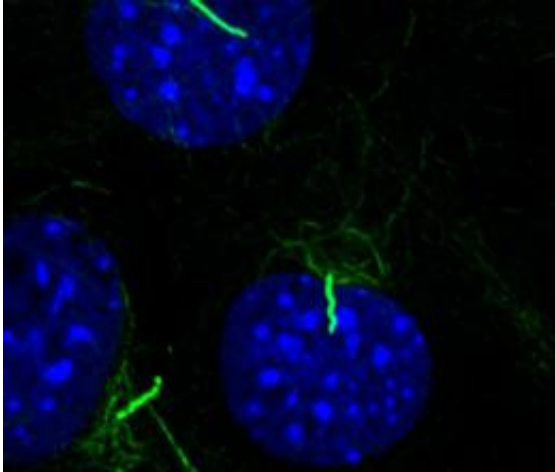


Scientists discover new inflammatory target

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Primary cilia

(Medical Xpress) -- Scientists from Queen Mary, University of London have found a new therapeutic target to combat inflammation.

The research, published in the journal Cellular and Molecular Life Sciences, revealed tiny organelles called [primary cilia](#) are important for regulating [inflammation](#). The findings could lead to potential therapies for millions of people who suffer from [arthritis](#).

Dr Martin Knight who led the research at Queen Mary's School of Engineering and Materials Science said: "Although primary cilia were discovered more than a century ago, we're only beginning to realise the importance they play in different diseases and conditions, and the

potential therapeutic benefits that could be developed from manipulating cilia structure and function.”

The researchers investigated the role of primary cilia in inflammation. They took cartilage cells and exposed them to a group of inflammatory proteins called cytokines, specifically interleukin-1 (IL-1), to see whether there were any changes to the primary cilia.

“When we exposed the cells to IL-1, in just three hours the primary cilia showed a 50 per cent increase in length,” he said.

“But what was most interesting was when we treated cells to prevent this elongation of the cilium. The cartilage [cells](#) had a greatly reduced response to the inflammatory proteins and were therefore not as inflamed. This suggests a brand new [therapeutic target](#) for inflammation.”

Co-author Dr Angus Wann, said this is the first time primary cilia have been suggested as a target for novel therapies to reduce the effects of inflammation.

“If we can work out how to better manipulate the primary cilium, we could potentially attenuate or even prevent inflammation,” he said.

More information: ‘Primary cilia elongation in response to interleukin-1 mediates the inflammatory response’ is published in the journal *Cellular and Molecular Life Sciences*.

[DOI:10.1007/s00018-012-0980-y](https://doi.org/10.1007/s00018-012-0980-y)

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