

Discovery could help stem smoking-related diseases

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(Medical Xpress) -- Sufferers of smoking related lung diseases could have their debilitating symptoms reduced following the discovery of a potential new treatment.

The discovery, by researchers at the University of Melbourne, Royal Melbourne Hospital, Australia, and the Brigham and Women's Hospital, Harvard Medical School, US, could dramatically improve treatments and slow the progression of COPD (Chronic Obstructive Pulmonary Disease) which includes the incurable condition emphysema.

COPD is a progressive disease that makes it hard to breathe and is mostly caused by excessive smoking. Approximately 2.1 million Australians have some form of COPD. By 2050, this figure is expected to more than double to 4.5 million.

The international team identified that the protein SAA plays a key role in chronic inflammation and [lung](#) damage in COPD and also inhibits the natural effort of the lung to repair itself after smoking has stopped.

The findings have been published in *The Proceedings of National Academy of Sciences*.

Professor Gary Anderson from the University of Melbourne said the discovery could become a dual treatment to improve lung function at any stage of COPD.

“It has the potential to dramatically improve the lives of many people suffering these conditions and reduce the huge burden of health and hospital costs associated with their treatment,” he said.

Lead author Associate Professor Steven Bozinovski from the University of Melbourne said the findings were significant because SAA was normally made in the liver, but they found that very high levels were made in the lungs of COPD patients. “It was a breakthrough for us to confirm that SAA played such a key role in the lung,” he said.

The team confirmed that SAA not only caused inflammation but hindered [natural healing](#) in the lung.

Harvard’s Associate Professor Bruce Levy said, they found that as the SAA interacted with its receptor it not only triggered lung inflammation, it also stopped a natural healing molecule which helped to turn off inflammation and heal the lung.

“This mechanism appears to explain one of the reasons that inflammation in COPD just never resolves despite stopping smoking,” he said.

The discovery could lead to the development of a dual treatment by firstly, targeting SAA to switch off its function in the lung and secondly, adding a synthetic form of the natural healing agent to boost lung healing. Clinical development for the synthetic agent is currently under way in the US.

The proposed combined treatment could also improve the effectiveness of steroid treatment for COPD, which is effective in treating other [lung diseases](#) such as asthma.

“Steroid treatments work in conditions like asthma by turning off the

production of inflammatory substances; however, our latest finding reveals that steroids actually fail to block the production of SAA and hence inflammation in the lung,” Professor Anderson said.

“We believe SAA plays a critical role in why steroids are much less effective than they should be in treating COPD,” he said.

It is hoped the new treatment will go to clinical trial within the next seven years.

“This is not a golden ticket to smoke,” he said. “We are hopeful the combined [treatment](#) will assist patients of all stages of COPD, particularly those in stage four with constant hospital visits, to improve their quality of life, but it would not cure disease,” he said.

He said the only way to prevent COPD is not to smoke. “If you are currently smoking the best thing to do is to quit as this will prevent the worsening of COPD,” he said.

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Provided by University of Melbourne

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