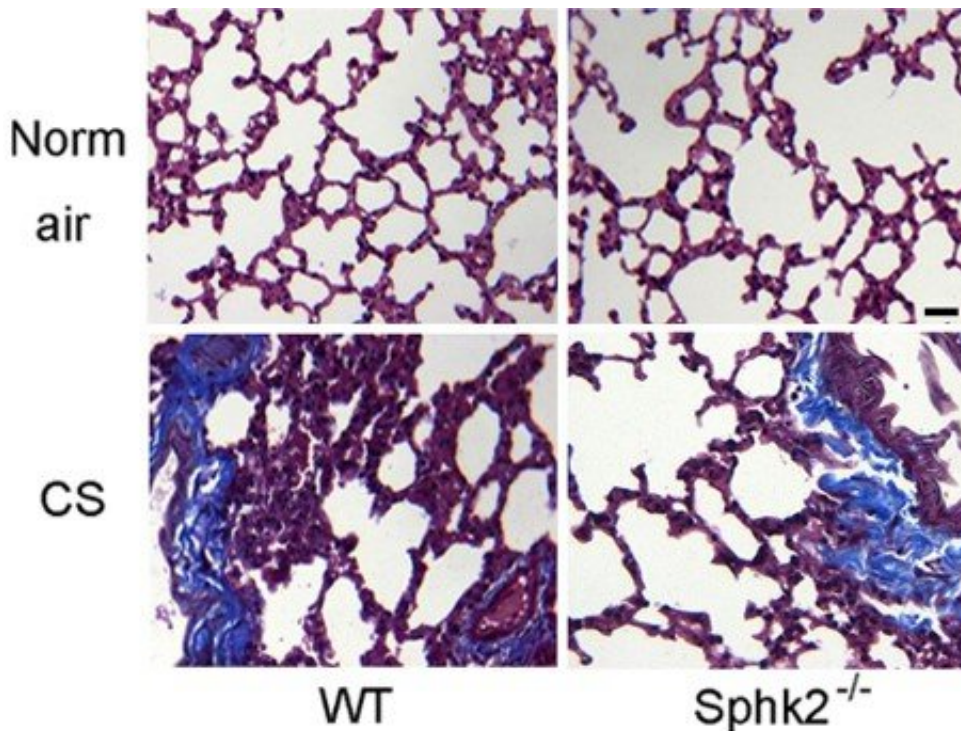


# SphK2 found to have a crucial role in the pathogenesis of COPD

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CS-induced pulmonary fibrosis was alleviated in *SphK2*<sup>-/-</sup> mice. WT and *SphK2*<sup>-/-</sup> mice were both subjected to air or CS exposure 5 days a week for 6 months. Credit: Biomolecules and Biomedicine

Chronic obstructive pulmonary disease (COPD) is a chronic respiratory disorder that progresses slowly and is characterized by cough, asthma, dyspnea and shortness of breath. Previous studies have shown that cigarette smoke (CS) is one of the major causes of COPD. Chronic

bronchial inflammation induced by CS promotes lung injury, fibrosis and remodeling, finally leading to emphysema, a lung condition that causes shortness of breath. However, the potential mechanism of CS-mediated COPD symptoms remains incompletely understood.

Researchers from the Loudi Central Hospital sought to explore the underlying mechanism and relationship between CS-induced inflammation and the development of COPD. Their research is published in the *Bosnian Journal of Basic Medical Sciences*.

Sphingosine-1-phosphate (S1P) is one of the major sphingolipid metabolites synthesized by sphingosine kinases (SphKs). SphK2 is highly expressed in the lungs, but its role in COPD remains unclear. In the present study, researchers found that both mRNA and protein levels of SphK2 were significantly upregulated in mouse lungs after CS exposure for 6 months, which is closely associated with lung inflammation and fibrosis induced by CS.

However, in SphK2 gene knockout mice, accumulation of neutrophils, secretion of inflammatory cytokines, and lung fibrosis induced by CS were markedly reduced. In addition, the deletion of SphK2 also rescued the expression of CFTR, which is an important protein in epithelial surface hydration, barrier function and innate defense.

This study demonstrated the crucial role of SphK2 in the pathogenesis of COPD, and it was proven that SphK2 deficiency would be beneficial for the treatment of lung inflammation, remodeling, and COPD after chronic CS exposure. Therefore, SphK2 could be an effective therapeutic target for COPD-like symptoms and diseases.

**More information:** Yanhui Chen et al, Deletion of sphingosine kinase 2 attenuates cigarette smoke-mediated chronic obstructive pulmonary disease-like symptoms by reducing lung inflammation, *Bosnian Journal*

*of Basic Medical Sciences* (2022). [DOI: 10.17305/bjbms.2022.8034](https://doi.org/10.17305/bjbms.2022.8034)

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