Commentary
DOI: http://dx.doi.org/10.53043/2347-3894.acam12009

Commentary: Genetic Association of Circulating C-Reactive Protein Levels With Idiopathic Pulmonary Fibrosis: A Two-Sample Mendelian Randomization Study

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Asian Journal of Complementary and Alternative Medicine. Volume 12 Issue 03

Published on: 20/12/2024

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Cite this article as: Chen M. *Commentary: Genetic Association of Circulating C-Reactive Protein Levels With Idiopathic Pulmonary Fibrosis: A Two-Sample Mendelian Randomization Study.* Asian Journal of Complementary and Alternative Medicine, Vol 12(3), 68-68:2024.

Zhang et al. [1] employed a two-sample Mendelian randomization approach to explore the causal relationship between circulating C-reactive protein (CRP) levels and idiopathic pulmonary fibrosis (IPF) risk, adding to the evidence that chronic inflammation plays a pivotal role in IPF pathogenesis. Their findings indicate that elevated CRP levels may drive IPF development, suggesting a potential therapeutic target.

CRP, a recognized inflammatory biomarker elevated in chronic diseases and cancers [2, 3], aligns with the evolving perspective of IPF as a disease driven by inflammation and fibrogenesis. This finding contradicts a previous MR study by Si et al. [4], which found a negative association, highlighting the need for further research. Furthermore, a recent study [5] demonstrated that elevated CRP levels are independently associated with earlier mortality in IPF, fibrotic hypersensitivity pneumonitis (fHP), rheumatoid arthritis-associated interstitial lung disease (RA-ILD), and systemic sclerosis-associated interstitial lung disease (SSc-ILD), suggesting a role of systemic inflammation in the prognosis of these interstitial lung diseases (ILDs).

Strengths of this study include robust methodology, large sample sizes from GWAS data, and minimal confounding influence in causal estimation. Limitations include a focus on European ancestry, reliance on GWAS summary data, and potential bias form unknown SNP functions.

If a causal relationship between elevated CRP levels and IPF is confirmed, this could pave the way for anti-inflammatory interventions targeting CRP or its associated pathways, potentially improving patient outcomes and enabling earlier diagnosis of IPF. This study significantly advances our understanding of the inflammation-IPF relationship, yet further research is needed to validate these findings across diverse populations and to explore the broader implications for other respiratory diseases.

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