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Research Article

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Exploring the Causal Relationship Between Plasma Lipidome and Allergic Diseases: A Mendelian Randomization Study

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Abstract

Background

Allergic diseases, including allergic rhinitis (AR), allergic asthma (AA), allergic conjunctivitis (AC), and allergic contact dermatitis (ACD), affect a significant portion of the global population. Recent advances in lipidomics have highlighted the role of plasma lipids in immune regulation and inflammation. This study aims to investigate the causal relationship between the plasma lipidome and various allergic diseases.

Methods

We conducted a bidirectional two-sample Mendelian randomization (MR) analysis, integrating data from multiple genome-wide association studies (GWAS). Various MR methods,including Inverse Variance Weighted (IVW), MR Egger, Weighted Median, Simple Mode, Weighted Mode, and Bayesian Weighted Mendelian Randomization (BWMR), were employed. Sensitivity analyses were performed to ensure robustness.

Results

Several plasma lipid subtypes were identified as either risk or protective factors for allergic diseases. Sterol ester (27:1/20:5), phosphatidylcholine (20:4_0:0), phosphatidylcholine (18:0_20:4), and phosphatidylcholine (18:0_22:5) were risk factors for AR. Phosphatidylcholine (20:4_0:0), phosphatidylcholine (18:0_20:5), and phosphatidylcholine (O-16:0_20:4) were risk factors for AA, while phosphatidylcholine (18:1_18:2) was a protective factor. For AC, phosphatidylcholine (18:1_18:1), phosphatidylcholine (18:1_18:2), and triacylglycerol (56:3) were protective factors. No significant associations were found for ACD after FDR correction.

Conclusions

This study highlights the significant roles of specific plasma lipid molecular subtypes in allergic diseases, particularly phosphatidylcholines and sterol esters. These findings enhance our understanding of the lipidomic landscape in allergy pathogenesis and suggest potential lipid-based therapeutic targets.

Keywords: Allergic diseases, Plasma Lipids, Phosphatidylcholine, Mendelian Randomization, Genetic Epidemiology, Lipidomics, GWAS