Cardiovascular Innovations and Applications

Vol. 11 (2026) 3
ISSN 2009-8618
DOI 10.15212/CVIA.2025.0031

RESPONSE TO LETTER TO THE EDITOR

Response by Yang et al. to Letter Regarding Article "Type 2 Inflammation: A Potential Clinical Link Between Asthma and Cardiovascular Diseases"

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In Response:
We appreciate the interest of Jawed et al. [1] in our publication [2]. Here, we respond to the two issues raised, incorporating the latest research on inflammation and cardiovascular diseases (CVD), and using terminology and style aligned with our article to the greatest extent possible:

In response to the phenotypic heterogeneity of type 2 asthma and its effects on CVD risk stratification

Type 2 asthma exhibits significant phenotypic heterogeneity. For example, the eosinophilic phenotype is associated with elevated arterial stiffness, which can indicate early atherosclerotic changes. In contrast, IL-17/IL-8-induced neutrophilic inflammation in non-eosinophilic asthma may pose different vascular risks. However, current CVD risk stratification lacks validated biomarkers for these specific asthma phenotypes. As highlighted in the 2025 ACC Scientific Statement, inflammation plays a critical role in CVD. Yet, in most studies, including the most recent literature, type 2 asthma has been treated as a homogeneous entity, thus potentially masking the unique CVD risks associated with specific phenotypes [3]. Future research should focus on integrating asthma phenotypes with CVD risk stratification and developing phenotype-specific biomarkers to enhance risk assessment accuracy.

In response to the application of existing biomarkers in CVD prediction and anti-inflammatory therapies

Although serum periostin and blood eosinophil counts are useful in predicting airway inflammation, their relationship with CVD remains

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unclear. The 2025 ACC Scientific Statement confirms the value of hsCRP in CVD risk assessment but indicates that the roles of other inflammatory biomarkers require further study [3]. Both the ESC and ACC emphasize the emerging role of anti-inflammatory therapies in CVD management. The CANTOS trial has demonstrated that canakinumab, an anti-IL-1β monoclonal antibody, decreases cardiovascular events by 15%–17% without affecting LDL cholesterol levels [4]. Low-dose colchicine has also shown promise in decreasing recurrent cardiovascular events in patients with chronic stable atherosclerosis and has been approved by the FDA for this purpose [5]. However, the CLEAR-SYNERGY trial did not show benefits in acute STEMI, thereby underscoring the importance of timing in inflammatory intervention [6]. Furthermore, in the CIRT trial, low-dose methotrexate did not show benefits in chronic atherosclerosis [7]. Several large trials are underway to evaluate novel IL-6 ligand inhibitors such as ziltivekimab in various CVD settings, and their results are expected in 2026–2027 [8].

In conclusion, addressing the phenotypic heterogeneity of type 2 asthma and the limitations of current biomarkers in CVD prediction is crucial. Both the ACC and ESC highlight the potential of anti-inflammatory therapies in CVD management. Future studies should focus on developing phenotype-specific biomarkers for CVD risk stratification and exploring anti-inflammatory treatments for CVD prevention and treatment.

Conflict of Interest

The authors have no conflict of interest to declare.

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