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ABSTRACT N. PO54

RESOLVIN RESPONSE IN BRONCHIAL ASTHMA PATIENTS: ENDOTYPE AND CLINICAL SEVERITY.

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Background and Aims: Asthma is a chronic inflammatory condition characterized by distinct endotypes. The most common is type 2 asthma, which is allergic and/or eosinophilic in nature. In contrast, non-type 2 asthma—typically neutrophilic or paucigranulocytic—often has a later onset and is more strongly associated with risk factors such as smoking, pollution, and obesity. Resolvins (Rv) are lipid mediators derived from omega-3 fatty acids, primarily eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), that can promote the resolution of inflammation in asthma. This study aims to determine resolvin levels in asthma patients in relation to endotype and clinical severity.

Methods: In a observational study, we enrolled thirty-nine patients with varying degrees of bronchial asthma severity (steps 3+4 and 5). Patients were also classified according to inflammatory endotype: type 2 allergic (GT2A, n = 20), type 2 non-allergic (GT2NA, n = 13), and non-type 2 (GNT2, n = 6). A control group (GC, n = 12) was also included. All participants underwent clinical assessment and lipidomic analysis focusing on D-series resolvins.

Results: Step 5 asthma patients showed significantly lower levels of RvD1 (step 3+4 vs. step 5, p = 0.018) and 17(R)-RvD1 (step 3+4 vs. step 5, p = 0.045) (Figure 1). Patients treated with the highest doses of inhaled corticosteroid/-long-acting β2-agonist (ICS/LABA) had lower levels of 17(R)-RvD1. No significant differences were observed for RvD2 and RvD3, **(Figure 1)**. The type 2 non-allergic group exhibited higher levels of RvD2 compared to both the type 2 allergic group (p = 0.040) and the non-type 2 group (p= 0.057), **(Figure 2)**.

Conclusions: Pro-resolving mediators are produced during asthma; however, their biosynthesis appears reduced in severe and uncontrolled disease. The lower levels of RvD1 and 17(R)-RvD1 observed in patients with higher clinical severity and reduced therapy responsiveness suggest that persistent and adaptive airway inflammation may be due, at least in part, to impaired resolution mechanisms. RvD2 is known to regulate type 2 inflammatory responses, promoting resolution in murine models through RvD2–DRV2 receptor interaction. Our findings support the pro-resolving role of RvD2, highlighting its potential as a key mediator in eosinophil recruitment and activation in the type 2 endotype. Large-scale studies are warranted to further elucidate the role of resolvins in asthma pathogenesis and their potential utility as biomarkers.

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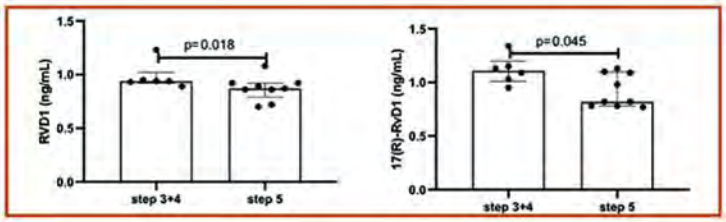
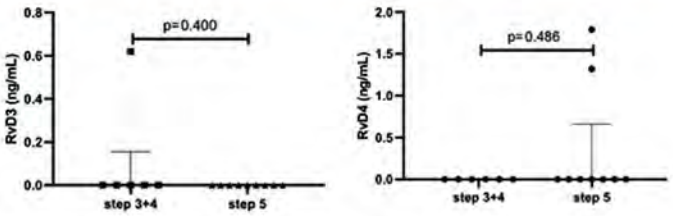
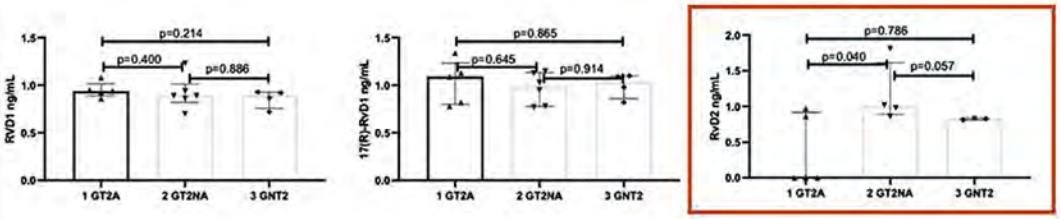
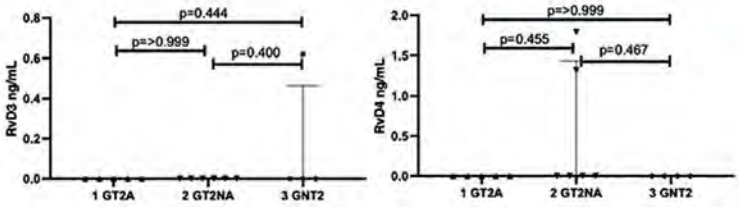

RVD1 (ng/mL)
p=0.018
step 3+4
step 5
17(R)-RvD1 (ng/mL)
p=0.045
step 3+4
step 5


RvD3 (ng/mL)
p=0.400
step 3+4
step 5
RvD4 (ng/mL)
p=0.486
step 3+4
step 5


RVD1 ng/mL
p=0.214
p=0.400
p=0.886
1 GT2A
2 GT2NA
3 GNT2
17(R)-RvD1 ng/mL
p=0.865
p=0.645
p=0.914
1 GT2A
2 GT2NA
3 GNT2
RvD2 ng/mL
p=0.786
p=0.040
p=0.057
1 GT2A
2 GT2NA
3 GNT2


RvD3 ng/mL
p=0.444
p=>0.999
p=0.400
1 GT2A
2 GT2NA
3 GNT2
RvD4 ng/mL
p=>0.999
p=0.455
p=0.467
1 GT2A
2 GT2NA
3 GNT2