Conclusions: Serum caspase-3, caspase-9 and caspase-14 levels were not statistically significant different between BD and HC groups. Serum pannexin-1 levels were statistically significant lower in the BD group.

REFERENCES:

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Abstract AB0050 – Figure 1. SAg system profile of ETN_1 and ETN_2 samples at 1 µg/mL.

Conclusions: The BioMAP phenotypic signatures of the ETN_1 and ETN_2 samples profiled in independent experiments using different primary cell pools remained comparable, which was consistent with conserved ETN mechanism of action. The BioMAP platform represents a useful orthogonal approach for assessing ETN activity.


Abstract AB0051 – Figure 1. SAg system profile of ETN_1 and ETN_2 samples at 1 µg/mL.

Conclusions: The BioMAP phenotypic signatures of the ETN_1 and ETN_2 samples profiled in independent experiments using different primary cell pools remained comparable, which was consistent with conserved ETN mechanism of action. The BioMAP platform represents a useful orthogonal approach for assessing ETN activity.


Background: Psoriatic arthritis (PsA) is affecting up to 40% of the patients with psoriasis. The pathogenesis of PsA is not completely understood. One of the hypothesis suggest that repeated minor injuries and trauma and lack of proper inhibition of inflammation lead to chronic inflammation spreading to surrounding tissue and other joints and ligaments. One of the mediators which lead to inhibition of inflammation in healthy conditions are derivatives of arachidonic acid – lipoxins.

Objectives: The aim of the study was to assess if the inhibition of lipoxin A4 on inhibition of synthesis of pro-inflammatory cytokines by peripheral blood mononuclear cells (PBMCs) of patients with psoriatic arthritis.

Methods: The study group consisted of 10 patients with psoriatic arthrits and 5 healthy controls. The peripheral blood mononuclear cells from patients with PsA and healthy controls were isolated and were stimulated with lipopolisacharide (LPS) with or without 200 nM of lipoxin A4 for 24 hours. The supernatants were collected after 24 hour stimulation. The levels of IL-1β, INF-gamma, TNF α, MCP-1, IL-6, IL-8 and IL-33 were assessed by cytometric bead array system.

Results: Incubation of cells with LPS, increased production of all cytokines assessed either in patients with psoriatic arthrits or in healthy controls. In PBMCs from healthy controls incubation of cells with lipoxine A4 decrease production of proinflammatory cytokines (IL-1β, MCP-1, IL-6, IL-8, IL-33 and TNF-α; p<0.05). However in patients with psoriatic arthrits addition of lipoxine A4 did not inhibit LPS – induced proinflammatory cytokines release (IL-1β, MCP-1, IL-6, IL-8, IL-33 and TNF-α; p<0.05).

Conclusions: Our study demonstrated that modulation of Inflammation by lipid mediators in patients with psoriatic arthritis is dysregulated.

Disclosure of Interest: None declared