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EFFECTS OF TAXIFOLIN ON INNATE INFLAMATORY RESPONSE

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INTRODUCTION

Inflammation is a normal physiological response of our body to an injury created, aiming to restore homeostasis and eliminate causative agents. Macrophages and neutrophils play an essential role in the completion of inflammation, they are recruited to the inflamed site and trigger a cascade of events in order to resolve it. today and that the use of anti-inflammatories can lead to undesired ends despite being of wide therapeutic assistance. Taxifolin has already been shown to have antioxidant, anti-inflammatory and anticancer effects. In this context, the present study will address its effect on macrophage and neutrophil activity.

MATERIAL AND METHODS

The anti-inflammatory activity was *in vitro* investigated using macrophages (RAW 264.7) and neutrophils previously treated with Taxifolin (0,1, 1 or 10 µM) and activated with lipopolysaccharide (LPS). Nitrite, IL-1β, IL-6 and TNF levels were measured in the macrophage and neutrophil culture supernatant. Inflammation resolution was measured by the *in vitro* efferocytosis assay. fMLP-induced chemotaxis was *in vitro* quantified using neutrophils previous treated with Taxifolin (0,1 µM).

RESULTS

In vitro, Taxifolin impaired the neutrophil chemotaxis and its ability to produce and/or release cytokines (TNF, IL-1β, and IL-6) and NO upon LPS stimuli. An enhanced efferocytosis of apoptotic neutrophils by macrophages was observed and accompanied by higher IL-10 secretion ($p < 0.01$).

CONCLUSIONS

Together, the data obtained shows that Taxifolin presents anti-inflammatory activity by modulating neutrophil migration/activation as well macrophage-dependent efferocytosis and inflammatory mediator release.

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