ABSTRACT

Nippostrongylus brasiliensis is a rodent intestinal nematode with an important pulmonary migrating stage. Previous studies have observed a lack of TNF-α production and minimal recruitment of neutrophils, which led us to the belief that anti-inflammatory mechanisms could be active in the lung stage. In this study, lipopolysaccharide (LPS) stimulated alveolar macrophages (NR8383) or rat lungs were used as *in vitro* or *in vivo* inflammation models respectively. Both live *N.brasiliensis* larvae and NES significantly reduced the production of LPS-induced pro-inflammatory mediators, TNF-α and NO, but not IL-1β, in NR8383 cells. The inhibition of TNF-α production was related to the heat-labile and trypsin-sensitive fraction of NES concentrate. 1-D protein gel of NES concentrate revealed that the molecular weights of proteins are between 6kDa and 100kDa. Glycoproteins were found abundant in NES concentrate. The inflammatory processes, including NF-κB translocation and TNF-α gene transcription were significantly inhibited by NES and/or NES concentrate. *In vivo*, we observed a significant reduction of neutrophil recruitment (≈ 40%) by NES on a background of LPS (100ng/ml) induced inflammation. This reduction was associated with the significant inhibition in gene transcriptions of proinflammatory mediators TNF-α, IL-1β, iNOS, ICAM-1 and MIP-2 in bronchoalveolar lavage (BAL) cells. The down-regulation of pro-inflammatory mediators and inflammatory processes observed in this study suggests that *N.brasiliensis* larvae and/or NES are capable of modifying the normally potent LPS inflammatory response, both *in vitro* and *in vivo*. This study and planned future studies could be fundamental in developing anti-inflammatory agents with immune-active molecules in *N.brasiliensis*-derived products.