Effect of Protease Inhibitor in an *in vivo* Lung Edema Model

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Lipopolysaccharide (LPS) induces an endotoxemia, with earliest damage in lungs, characterized by neutrophil infiltration and releasing of neutrophil proteases and reactive oxygen species. *Caesalpinia echinata* (pau-brasil), belonging to the Leguminosae family, contains large amounts of protease inhibitors in its seeds. So, the aim of this work was to verify the effect of a kallikrein inhibitor from *C. echinata* seeds – CeKI, in a lung edema model. Three months Wistar rats received in their tails phosphate buffer, pH 7.4 or CeKI (2.6 or 7.8 mg) in the same buffer. After 20 min, a tracheotomy was performed to inject 1 mg LPS (LPS, CeKI 2.6 and CeKI 7.8 groups) or PBS (control), directly into their lungs. Six hours later, bloods were colleted, with or without EDTA, lungs were extracted and bronchoalveolar lavage fluid (BALF) was obtained. Polymorphonuclear cells (PMN) were analyzed in BALF. After centrifugation of serum, plasma and BALF, neutrophil elastase, myeloperoxidase (MPO), angiotensin converting enzyme (ECA) and kinin were quantified with appropriated buffers, substrates and inhibitors. In the BALF, neither qualitative nor quantitative differences were found in total protein, but there was an increase in PMN and elastase activity in the LPS group, inhibited by CeKI. In the plasma, kinin was detected in the LPS group, 100 folds higher than in the others, but concerning the enzymes there were no difference among the groups. In the serum, no changes were observed in ECA or MPO assays. In conclusion, CeKI was able to control PMN infiltration, elastase activity and kinin releasing probably due to coagulation enzymes inhibition (FAPESP, CAPES and CNPq).