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Title: The antimicrobial peptide CRAMP protects against emphysema

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Body: Rationale: COPD is one of the most prevalent causes of death worldwide and is associated with an ongoing destruction of pulmonary structures that finally lead to the development of emphysema. Antimicrobial peptides (AMP) are a part of the innate immune system. The AMP CRAMP is the murine homologue to the human cathelicidin antimicrobial peptide (hCAP-18/LL-37). It plays an important role in angiogenesis, cancer and chemotaxis. This work will show that CRAMP is important for tissue regeneration in an elastase induced model of lung destruction. Methods: Lung destruction was initiated by two times intratracheal administration of elastase. Ten days after the last administration lung function was measured. Cytokines in BAL were measured by ELISA. The lungs were resected and fixed for stereological analysis. Lung tissue was also used for immunohistological staining and RNA-extraction. Results: Elastase treated CRAMP-ko animals had significantly higher neutrophil influx, more IL1- β and TNF- α in their BAL as the controls. 30 days after the first elastase treatment CRAMP-ko had a higher mean linear intercept and a significantly decreased pulmonary system resistance and elastance. MMP-9 expression and activity was increased and the concentration of VEGF was decreased in the elastase treated CRAMP-ko as compared to the elastase treated wildtype mice. Conclusion: This work shows that the antimicrobial peptide CRAMP has a protective function in a model of elastase induced lung destruction. CRAMP-ko animals showed more inflammation, a higher degree of lung destruction as well as a higher expression and activity of MMP-9. The concentration of VEGF was significantly lower in the elastase treated ko animals.