INTRODUCTION

Pepsin is a causal agent of inflammation during Nonacidic Reflux

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ABSTRACT

Common clinical manifestations and endoscopic inflammation and other immune responses initiated within the airway mucosa are a fundamental determinant of tissue damage during reflux and are predicted to give rise to the diverse phenotypes characteristic of reflux-attributed disease.

The aim of this study was to investigate the potential of pepsin to contribute to mucosal inflammation, and thereby mucosal damage, via change in cytokine and receptor gene expression during nonacidic extra-esophageal reflux.

Materials & Methods

Expression of 7 genes was increased by >1.5-fold (p < 0.05) relative to control: CCR6 and CXCL14 (see Table 2).

Increased expression of IL1 and TNF and IL1 are elevated in the esophageal epithelium in combined-type esophagitis in rats: a transcriptome analysis. Int J Mol Med 2006; 18:821-8.

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Table 1. Cytokine and Receptor Expression of Control and Pepsimtreated Human Hypopharyngeal Cells

MATERIALS & METHODS

Table 2. Change in Gene Expression of Human Hypopharyngeal Cells Exposed to Pepsin at Neutral pH

RESULTS

Conclusions

Increased expression of IL8 and other proinflammatory cytokines was observed in the esophageal epithelium in combined-type esophagitis in rats, supporting a role for inflammation in the pathogenesis of combined-type reflux disease.

REFERENCES

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