Azithromycin, clarithromycin and telithromycin inhibit MUC5AC induction by Chlamydia pneumoniae in airway epithelial cells.


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Abstract

BACKGROUND:
Airway mucus hypersecretion is an important problem in chronic respiratory diseases including bronchial asthma. Chlamydia pneumoniae is recently confirmed to be a pathogen in bronchial asthma, but the relationship between C. pneumoniae and mucus hypersecretion is uncertain. In this study, we examined whether C. pneumoniae induces MUC5AC mucin in airway epithelial cells. We also examined the effects of macrolide and ketolide antibiotics on the C. pneumoniae-induced mucus production.

METHODS:
MUC5AC production in bronchial epithelial cells after stimulation with C. pneumoniae was analyzed by ELISA and quantitative RT-PCR. NF-kappaB and phosphorylated ERK were also analyzed. For inhibition study, cells were pretreated with azithromycin, clarithromycin and telithromycin before stimulation.

RESULTS:
C. pneumoniae dose-dependently induced MUC5AC production and gene expression. The ERK-NF-kappaB pathway was involved in C. pneumoniae-induced MUC5AC production. Macrolides and ketolides dose-dependently reduced C. pneumoniae-induced MUC5AC production. However, azithromycin was apparently less effective than the other antibiotics. Clarithromycin and telithromycin, but not azithromycin, reduced NF-kappaB activation.

CONCLUSIONS:
Clarithromycin and telithromycin were thought to interfere with the signal pathways between ERK and NF-kappaB. These results suggest that airway mucus hypersecretion is one of the mechanisms of C. pneumoniae-induced bronchial asthma, and that macrolide and ketolide antibiotics represent a novel therapeutic intervention in these patients.

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