Abstract


Cysteine ameliorates allergic inflammatory reactions by suppressing thymic stromal lymphopoietin production in activated human mast cells.

Nam SY, Kim HM, Jeong HJ.

Department of Pharmacology, College of Korean Medicine, Kyung Hee University, Seoul, 02447, Republic of Korea; Department of Food Science & Technology, Hoseo University, Asan, Chungnam, 31499, Republic of Korea.

OBJECTIVE: Thymic stromal lymphopoietin (TSLP) derived by mast cells is recognized as a critical factor in many allergic inflammatory disorders. Cysteine is a well-known amino acid which exhibits anti-inflammatory activities. However, the effect and mechanism of cysteine on TSLP production have not been investigated. Thus, we hypothesized that cysteine may regulate TSLP production from mast cells.

METHODS: To test this hypothesis, the anti-inflammatory effects and signaling pathways of cysteine were investigated in phorbol 12-myristate 13-acetate 4 and calcium ionophore A23187 (PMACI)-stimulated human mast cell line HMC-1.

RESULTS: Cysteine dramatically attenuated the levels of TSLP of both mRNA and protein without cytotoxicity. Moreover, cysteine suppressed caspase-1 activation and nuclear factor-κB translocation. The phosphorylation of p38 and c-Jun N-terminal kinase was downregulated in all cases in PMACI-stimulated HMC-1 cells treated with cysteine. In addition, cysteine decreased PMACI-induced proinflammatory cytokines in terms of both protein and mRNA levels.

CONCLUSION: In conclusion, cysteine regulates TSLP production by blocking caspase-1, nuclear factor-κB, p38, and c-Jun N-terminal kinase-dependent pathways in activated HMC-1 cells, suggesting its potential as a regulator of allergic inflammatory diseases.

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