

VOLUME 42 • NUMBER 5 • 2012

TJMS

TURKISH JOURNAL OF MEDICAL SCIENCES



TÜBİTAK

Turk J Med Sci
<http://journals.tubitak.gov.tr/medical/>

ISSN 1300-0144
E-ISSN 1303-6165

Published by the Scientific and Technological Research Council of Turkey

Effects of L-2-oxothiazolidine-4-carboxylic acid on the lung antioxidant defense system in an asthma mouse model

Lyudmil GEORGIEV TERZIEV¹, Veneta LYUBENOVA SHOPOVA², Violeta YORDANOVA DANCHEVA²,
Galya TZVETANOVA STAVREVA³, Angelina MILCHEVA STOYANOVA⁴

Aim: We aimed to study the effect of a glutathione precursor, L-2-oxothiazolidine-4-carboxylic acid (OTCA), on the lung antioxidant defense system in an animal asthma model.

Materials and methods: The study was carried out on 24 female C57BL/6 mice. The mice were divided into 4 treatment groups: group 1 – control group; group 2 – injected with ovalbumin (OVA) and given an OVA inhalant; group 3 – treated with OTCA and phosphate-buffered saline inhalant; and group 4 – injected with OVA and OTCA and given an OVA inhalant. Under sodium pentobarbital anesthesia the animals were killed by exsanguination 48 h after the last inhalation to obtain a lung homogenate. The activities of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GP) and the content of nonprotein sulfhydryl (NPSH) groups in lung homogenate were investigated.

Results: OVA decreased the activities of SOD ($P = 0.007$), CAT ($P = 0.004$), and GP ($P = 0.05$) and the NPSH content ($P = 0.0008$) in the lung homogenate compared with the control animals. Treatment with OVA and OTCA (group 4) resulted in a significant increase in the activities of CAT ($P = 0.01$) and GP ($P = 0.05$) and the NPSH content ($P = 0.002$) compared to the OVA group (group 2).

Conclusion: OTCA (160 mg/kg) restored the activities of basic enzymes in the lung antioxidant defense system in an OVA-induced asthma mouse model 48 h after the last nebulization.

Key words: Asthma, L-2-oxothiazolidine-4-carboxylic acid, lung antioxidant defense system

Introduction

The development and maintenance of allergic airway inflammation depends on the complex interaction of many cytokines and chemokines. T cells involved in the proallergic response produce cytokines associated with induction of classical Th₂ response, in which there is an oversecretion of IL-4, IL-5, and tumor necrosis factor- α (TNF- α) (1). Accompanied by an increase in the mortality rate (2), bronchial asthma incidence has reached more than 29% within the past years in the West European

countries (3). The development of an inflammatory immune response in numerous pulmonary diseases, including asthma, has been reported. It is described as an activation of epithelial cells and macrophages and an influx of activated neutrophils, eosinophils, monocytes, and lymphocytes into the airways. These cells can modulate the inflammatory response known to form a large amount of reactive oxygen species (ROS) and reactive nitrogen species (RNS) in the airways (4,5). Enzymatic and nonenzymatic antioxidants neutralize the toxic oxygen products formed under physiological conditions. However,

Received: 30.03.2011 – Accepted: 17.11.2011

¹ Sector of Clinical Immunology and Allergology, Medical University-Pleven, Pleven - BULGARIA

² Sector of Disaster Medicine, Medical University-Pleven, Pleven - BULGARIA

³ Sector of Experimental and Clinical Pharmacology, Medical University-Pleven, Pleven - BULGARIA

⁴ Sector of Chemistry, Medical University-Pleven, Pleven - BULGARIA

Correspondence: Lyudmil GEORGIEV TERZIEV, Sector of Clinical Immunology and Allergology, Medical University-Pleven, Pleven, 5800 - BULGARIA

E-mail: luterzi@mail.bg