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Research Article

Antioxidant Effect of MnTE-2-PyP on Lung in Asthma Mice Model

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Abstract

Aim. To investigate the effects of MnTE-2-PyP on some markers of antioxidant defence system in asthma mice model. **Material and Methods.** The animals were divided into four groups: group 1, controls; group 2, injected with ovalbumin, group 3, treated with MnTE-2-PyP, and group 4, treated with ovalbumin and MnTE-2-PyP. The activities of superoxide dismutase, catalase, glutathione peroxidase and nonprotein sulfhydryl groups content (NPSH) were determined in lung homogenate. **Results.** The activities of superoxide dismutase and catalase in group 2 decreased significantly as compared to control group. The decrease of the same enzymes in group 4 was lower and significant as compared to group 2. Changes in the glutathione peroxidase activity showed a similar dynamics. The NPSH groups content decreased in group 2. In group 4 this decrease was relatively lower as compared to group 2. **Conclusions.** The application of MnTE-2-PyP mitigated the effects of oxidative stress in asthma mice model.

1. Introduction

Asthma is a major, worldwide health concern, affecting children and adults. It affects 7% of the US population and 300 million worldwide [1]. Currently in the USA, some 22 million people are reported to have asthma, six million of whom are children [2]. It is among the commonest chronic conditions in Western countries affecting 1 in 7 children and 1 in 12 adults (equivalent to 5.1 million people in the UK) and is responsible each year for 1500 avoidable deaths, as well as 20 million lost working days. The annual UK healthcare cost is estimated to be £2.5 billion [3]. Oxidative stress is believed to play a role in the development of number of human diseases such as cardiovascular disorders, immunologic diseases, cancer, and asthma. A large amount of epidemiological and clinical evidence exists to support the relationship between increased reactive oxygen species (ROS) and the pathogenesis of bronchial asthma [4–8]. Oxidative stress is a deleterious process that leads to lung damage and consequently to various lung diseases. To protect against exposure to oxidants the lungs have a powerful antioxidant system, including nonenzymatic and enzymatic antioxidants, which may delay or prevent oxidation, but also eliminate reactive oxygen species [5]. At high levels of oxidative stress, however, antioxidants become depleted, and an imbalance between oxidants and antioxidants occurs, which causes pathological damage, or a variety of cellular responses through formation of secondary ROS [9]. All the major varieties of inflammatory lung diseases, asthma, chronic obstructive pulmonary disease, idiopathic pulmonary fibrosis, acute respiratory distress syndrome, interstitial lung diseases, and bronchopulmonary dysplasia share a common feature of impaired oxidant/antioxidant ratio [10]. Therefore, the supplementation of antioxidants to boost the endogenous antioxidants or scavenge excessive ROS production could be utilized to prevent the inflammatory response in asthma by restoring oxidant-antioxidant balance. Current knowledge of the effects of oxidative stress allow the development of new classes of antioxidants in the treatment of asthma and other disorders, associated with the oxidative stress. Considerable progress has been made in the last years, in developing mitochondria-targeted antioxidants such as manganese porphyrins [2]. A number of water-soluble *meso*-substituted manganese porphyrins with a molecular weight above 800 quickly pass through the cell membranes and are distributed into the mitochondria [11, 12]. Therefore we aimed to study the effect of MnTE-2-PyP (Manganese (III) 5,10,15,20-tetrakis (N-ethylpyridinium-2-yl) porphyrin), a manganese-*meso*-porphyrin also known as AEOL-10113, on some markers of lung antioxidant defence system in asthma mice model.

2. Material and Methods

2.1. Chemicals

Ovalbumin, grade V and phosphate-buffered saline (PBS) were purchased from the Sigma-Aldrich Company and Imject Alum was obtained from Pierce Chemical Company (USA).

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