


Article

Probiotics' Efficacy in Preventing Asthmatic Allergic Reaction Induced by Air Particles: An Animal Study

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Abstract: Global air pollution and diesel exhaust particles (DEPs) generated by intratracheal instillation aggravate asthma. In this study, we evaluated the effect of probiotics via tracheal- or oral-route administration on allergies or asthma. We continuously perfused rats daily, using the oral and tracheal routes, with approximately 10^6 – 10^8 CFU probiotics, for 4 weeks. During this period, we used OVA-sensitized rats to build the asthma models. We orally or intratracheally administered *Lactobacillus paracasei* 33 (LP33) to the rats, which reduced the number of total inflammatory cells, lymphocytes, and eosinophils in the bronchoalveolar-lavage fluid, the IgE concentration, and the cytokine levels of TH2 cells, but we found no significant difference in the cytokine levels of TH1 cells. LP33 can be used to prevent asthmatic allergic reactions induced by aerosol particles. Nevertheless, the dosage form or use of LP33 needs to be adjusted to reduce the irritation of lung tissues, which may produce lesions of the trachea. We observed that DEP dosage can alleviate emphysema, and that LP33 has a substantial effect on improving or slowing allergic asthma.

Keywords: diesel exhaust particles; asthma; ovalbumin; probiotics; allergic disease



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1. Introduction

Asthma is a chronic pulmonary-inflammatory disease characterized by a large amount of eosinophilic bulb-infiltration and inflammatory reactions in the bronchus, excessive mucus secretion, and obstruction of airflow in the respiratory tract [1]. Various factors aggregate asthma, such as severe air pollution from the diesel exhaust particulates (DEPs) produced by the combustion of diesel fuel [2]. In Taiwan, the main source of suspended particulates is DEPs, which are produced from large vehicles such as buses and trucks. Epidemiological studies have noted an association between exposure to traffic-derived pollutants and lung function in the asthmatic population [3].

DEPs are composed of elemental carbon, polycyclic aromatic hydrocarbons, acidic aerosols, volatile organic compounds, etc. After being inhaled by the respiratory system, DEPs penetrate deep into the alveoli and even into the microvessels of the alveoli, and freely penetrate the cells and tissues of the human body, causing systemic effects, especially a substantial increase in the number of lung and bronchial diseases [4].

Numerous researchers have assessed the effect of particulate pollutants, and particularly of DEPs, on the respiratory system in animal models. In a study of the effect of house-dust extract (HDE) on BALB/c mice, the results showed that DEPs increased HDE-induced airway inflammation, airway mucus-production, oxidative response, and inflammatory-cell infiltration, as well as CXC chemokines at bronchoalveolar-lavage concentrations and airway hyperresponsiveness (AHR) [5]. The variation in exposure to DEPs (low dose: $100 \mu\text{g}/\text{m}^3$ DEPs, high dose: $3 \text{ mg}/\text{m}^3$ DEPs) for 1 h per day, 5 days per week, for