INTRODUCTION

Otitis media with effusion (OME) is caused by multiple factors and complex interactions of biochemical, immunological and inflammatory mediators in the middle ear cavity *(Juhn, 1982)*. Among these factors, abnormal function of the eustachian tube (ET) mucosal changes, presence of micro-organisms and the effect of inflammatory mediators seem to have the most influence on the etiology and pathogenesis of OME *(Paparella and Koutroupas, 1982)*.

Platelet activating factor has been identified in human middle ear effusion and has been suggested to participate in developing otitis media with effusion and maintaining middle ear effusion by stimulating vascular permeability and chemotaxis *(Cauwenberge et al., 1987)*. However, the exact role of platelet activating factor as an inflammatory mediator in the pathogenesis of otitis media with effusion is not known. Previous studies have implicated that platelet activating factor is a potent inflammatory mediator. A biologically active phospholipid, platelet activating factor (1-0-alkyl-2-acetyl-sn-glyceryl-3-phosphoryl -choline) is now known to have wide-range of effects on a number of tissues and inflammatory cells *(Barnes et al., 1988)*. Since the early 1970, many studies have demonstrated that platelet activating factor may play an important role in acute inflammation and allergy *(McManus,*

1986). It is released from human neutrophils, platelets eosinophils, macrophages, mast cells, and vascular endothelial cells *(Barnes et al., 1988).* Its activities include induction of microvascular leakage, chemotaxis, bronchoconstriction, increased bronchial hyperresponsiveness, increased airway mucus secretion, and induction of hypotension shock and death *(McManus, 1986).*

It is now recognized that eosinophils may play a significant role in the pathogenesis of several inflammatory and allergic lung disorders including bronchial asthma and certain pulmonary eosinophilic syndromes *(Frigas et al., 1986).*

Eosinophils elaborate a wide range of inflammatory mediators and intracellularly stored granule constituents that may act in concert to induce the pathology that characterizes these disease states *(Barnes et al., 1988 and Davis et al., 1984).*

Platelet activating factor (1-0-alkyl-2-acetyl-sn-glycero-3-phospho-choline) represents one of the most potent chemoattractants for eosinophils thus far identified *(Wardlaw et al., 1986)* and increasing evidence suggests that this lipid mediator may be released immunologically from macrophages and other cells resident in pulmonary tissue, thereby attracting eosinophils into the lung *(Barnes et al., 1988).*