VI. SUMMARY

This study was focused on the allergic rhinitis patients who had no others allergic condition, asthma or atopic dermatitis. The nasal scraping was collected to clarify whether BMC was mast cell and used to demonstrate the predominant bacteria colonized in nasal mucosa. The serum was collected and tested for anti house dust mite IgE antibody. The collection of predominate colonized bacteria and anti-house dust mite IgE with the patient severity were investigated. The 69 AR were registered who 15 were mild AR, 24 were moderate AR and 30 were severe AR. The 26 nonallergic healthy subjects were used as control. The age and sex distribution was similar in both groups.

The predominate cells found in nasal scraping of AR and correlated with symptom, namely BMC cells, was proved to be mast cell which was an important cell in allergy. The granules of this cell stained purple-blue with Wright-Giemsa and also positive stained with specific dye of mast cell, Toluidine blue and Alcian blue/Safranin dyes. All of these stainings could demonstrate the numerous granules in cytoplasm and unique morphology of degranulation. The number of BMC and degree of degranulation were similar in all staining methods and correlated with severity, in severity dependent pattern. These results suggested that BMC was mast cell.

Moreover, this study showed some of the mast cell recruited into the nasal mucosa during allergic inflammation was positive stained with Alcian blue/Safranin, which is the specific dye of connective tissue mast cell (CTMC). This means that CTMC moved from submucosa layer into the mucosal layer, especially in severe cases.

Determination of type and number of bacteria colonized in nasal cavity of AR and nonallergic healthy subjects were performed by culturing nasal mucosal specimens and identification of predominant bacteria. *Staphylococci* species were the most common isolates in AR. The predominant bacteria in AR and nonallergic group were different in type and number. The mean number of colonized bacteria showed

that *S. aureus* and *K. pneumoniae* were gradually increased in mild, moderate, and severe AR, respectively, and were also significantly higher in the number in AR than nonallergic subjects ($p < 0.01$). This data indicated that some of predominant bacteria colonized at nasal mucosa of patients might be involved in the pathogenesis of AR.

In order to evaluate the specificity of the indirect ELISA test for antibody to house dust antigens, the recombinant Der p 1 (r Der p 1), r Der f 1 and r Blo t 1 were used as antigen. The standard curve of IgE concentration was performed along with the test system. The same anti human IgE-biotin conjugate and streptavidine-HRP conjugate were used in the test system and IgE standard curve setting. If the equal amount and concentration of anti-human IgE-biotin conjugate was used in the test system and the standard curve setting and gave the same optical density value result, the amount of IgE anti-house dust mite and the standard human IgE should be equal. Therefore, with the standard human IgE in various known concentration, the OD value obtained could be used to construct the standard curve for determining the concentration of anti-house dust mite IgE specific to the house dust mite antigen in the test system. The 55 AR and 29 nonallergic subjects were tested for IgE anti house dust mite. Anti-Der p 1 was found the most (45.45 %) in AR than anti-Der f 1 (25.45 %) and anti-Blo t 1 (5.45 %). With the concentration, the average amount of anti-Der p 1 was also found the highest (~100 ng/ml) in AR than anti-Der f 1 (~81 ng/ml) and anti-Blo t 1 (0.21 ng/ml). Even all of these AR has severity score range mild to severe but the amount of every anti house dust mite IgE antibodies were not correlated with the symptom severity scores.

In summary, this study clearly showed that BMC in nasal scraping of AR was mast cell by their morphology and specific staining characteristics. CTMC contributed at the site of inflammation in mucosal layer indicated that CTMC migrated from submucosal layer outward to mucosal layer. CTMC accompanied with MMC responded to the stimuli in nasal cavity, leading to allergic inflammation. The total number of CTMC and extensive degranulated type was found higher in severe AR than moderate and mild AR. These mean that CTMC not only migrated but also has functions, since the mediator contents in connective tissue type is more harmful than mucosal type.

The colonization of bacteria in nasal mucosa was correlated with the severity, both in numbers and types of bacteria. This showed that colonized bacteria might partially involve in the pathogenesis of AR. The use of antimicrobial agents should be considered in addition to traditional treatment as report in atopic dermatitis.

Detection of anti house dust mite IgE from patient sera not only explain the pathogenesis of AR as type I hypersensitivity but also indicate the epidemiology of *Dermatophagoides spp*. but not *Blomia tropicalis* in this area. Therefore, the immunotherapy of AR should include Der p 1 and Der f 1 which as known in asthma.