7. SUMMARY AND CONCLUSION

At present about 300 million people are suffering from bronchial asthma globally. As per the data available with Control of Disease Centres, U.S, every year the country spends about $56 billion on asthma. The United Kingdom spends about £1 billion on various Asthma control programmes. In United States, in the year 2010, 8.7 million adults and 7 million children had asthma. This is equal to 1 in 12 adults and 1 in 11 children.(CDC2012)

In the early period of twentieth century, asthma epidemics were reported in different parts of the world. But, when detailed survey was taken in different population, the spatial distribution of asthma showed uniformity within many developed countries. Although the levels of Ozone (O3) and nitrogen dioxide (NO2) in the atmosphere varied in urban and rural areas, there was little difference in the occurrence of asthma and morbidity in major urban and rural areas. The study conducted by Gaur et al (2006) among urban and rural adult population of Delhi to find out prevalence of bronchial asthma and allergic rhinitis found no significant difference in asthma prevalence between urban and rural population. Likewise, the studies conducted by Xu X (1993) in Beijing, China, Wilkie (1995) in New Zealand, Alameldin(2012) in Assiut District, Egypt also confirmed the fact that the occurrence rate of bronchial asthma and allergic rhinitis did not vary between rural and urban areas. Hence it has become evident that allergens like outdoor and indoor allergens, antenatal and postnatal infections, worm infestation, diet, chemicals etc., are only precipitating or exaggerating the existing asthmatic conditions and they are ‘not causative factors’.

Since the occurrence rate of bronchial asthma is 10-15%, why the remaining 85 - 90% of the people is not affected although they are also exposed to the same allergens? Hence the real causative factors must be within the allergic person only. Parrot et al (1953) showed that the serum of normal subjects possessed 20-30% more histamine binding capacity as compared to allergic person in whom it was 0-5% only. This low level of serum histamine binding capacity (SHBC) in allergic patients must be the cause for occurrence of allergic diseases. A modified ELISA test has been
developed by us to estimate the serum histamine binding capacity in normal and allergic persons.

In order to assess and prove that SHBC level will be low in allergic persons as compared to normal persons, a study was conducted with 50 persons, 25 normal and 25 allergic persons. The total immunoglobulin E level was estimated using ELISA test. Based on the IgE level, patients were separated into normal (Ig E level < 100 IU/ml) and allergic persons (IgE level > 200 IU/ml). The anti-histamine antibody was separated from the serum using Affinity Chromatography method. The anti-histamine antibody level was estimated using Bradford protein estimation method. With the normal serum (ID NR) having anti-histamine antibody level of 3.59 µg/200 µl, its serum protein in different dilutions of 1:5000, 1:10000, 1:25000, 1:50000, 1:75000 and 1:100000 were estimated. The standard curve was plotted using the O.D values of different dilutions. The serum anti-histamine antibody level of 25 normal and 25 allergic persons was estimated by ELISA test using histamine coated micro wells. The antihistamine antibody level (SHBC) for 25 normal persons was found to be 35.44 µg/ml and for allergic patients it was 21.61 µg/ml. Hence the serum histamine binding capacity was found to be 39% less in allergic patients. In normal people, all the liberated histamine will be nullified by the anti-histamine antibodies whereas in allergic people, all the histamine liberated, will not be nullified by the antihistamine antibodies, because of its low histamine binding capacity. This free histamine present in the blood is available to act upon the H1 receptors of target cells thereby causing allergic and asthmatic symptoms.

The results from our study conducted with 25 normal and 25 allergic patients proved that the serum histamine binding capacity of normal people was 39% more as compared to allergic people. This low level of SHBC should be the cause for the occurrence of bronchial asthma and allergic rhinitis. The anti-histamine antibody was identified to be immunoglobulin ‘G’ type by MALDI-MS.