IMMUNOGLOBULIN (IgE) TOTAL SERUM LEVEL IN WORKERS EXPOSED TO WOOD DUST

THESIS

Submitted in Partial Fulfilment for Master Degree in Chest Diseases

By

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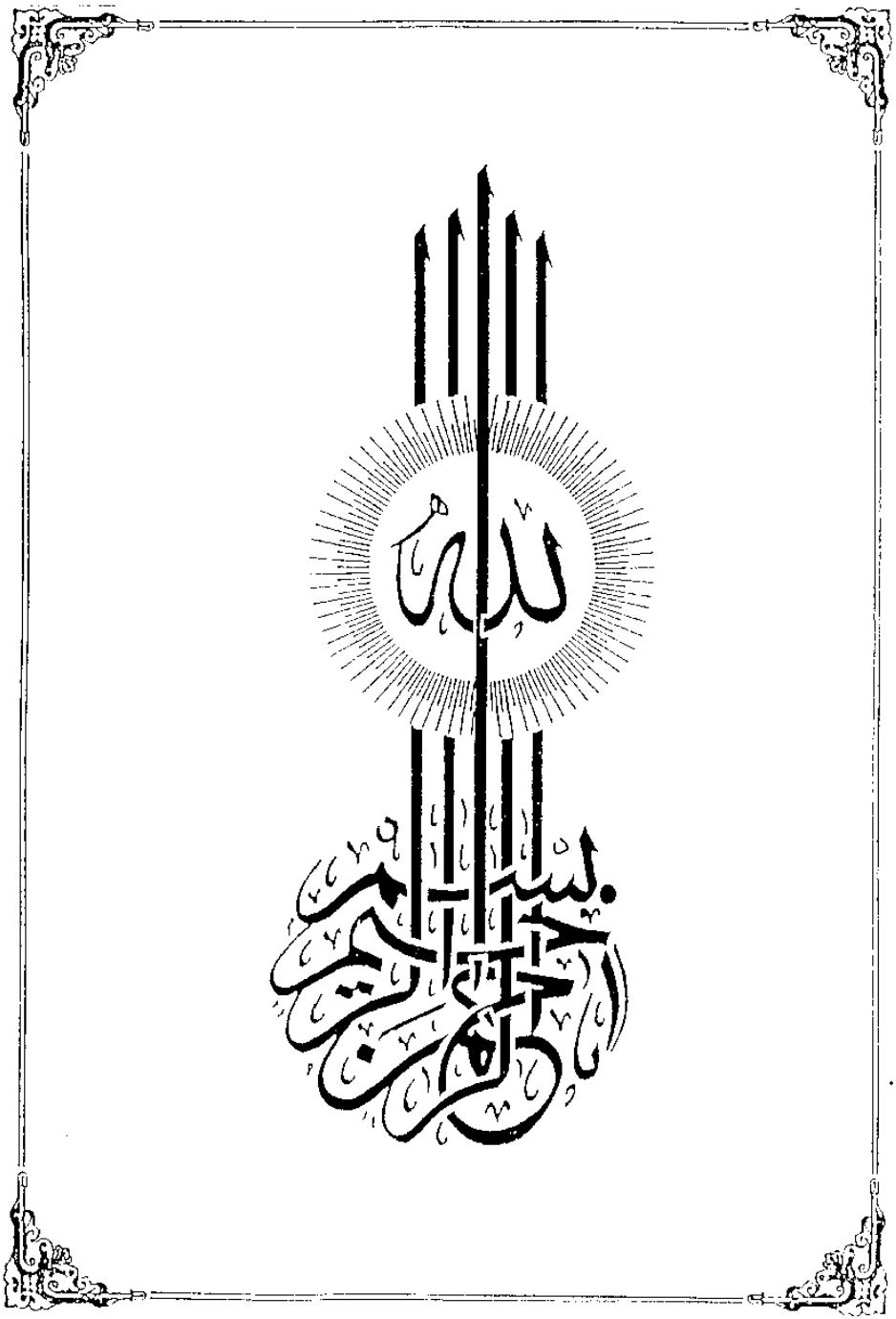

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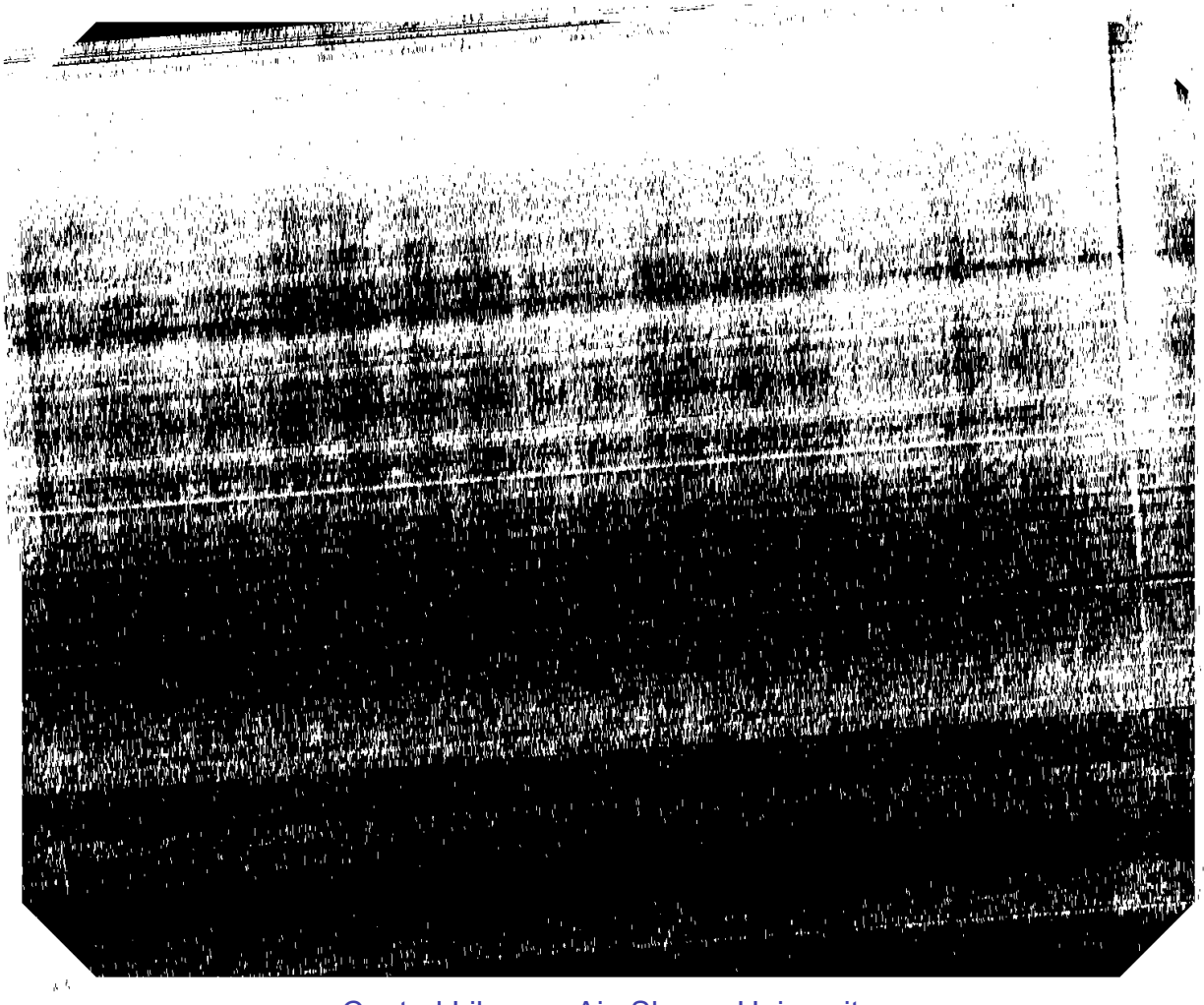
INTRODUCTION

The biological effects of wood give rise to many different morbid symptoms or processes, the nature of which depends on the quantity and composition of the constituent substances which range from hydrocarbons to polycyclic compounds. The clinical symptoms, in which wood dust is assumed to be a causative agent, are various according to different authorities. **Kadlec and Hanslian (1983).** Summarized these symptoms in: Skin allergy, allergic conjunctivitis, allergic rhinitis, bronchial asthma, irritation of mucosae of upper respiratory tract, acute and chronic lung disorders. Studies of ventilatory functions in workers exposed to wood dust showed a reduction in FVC (forced vital capacity) and FEV 1.0 (forced expiratory volume in the first second) **(Zuhair et al., 1981).** In a study by **Acheson et al. (1968)** among wood workers in furniture industry in U.K. wood dust was strongly proposed to be the cause of nasal cancer.

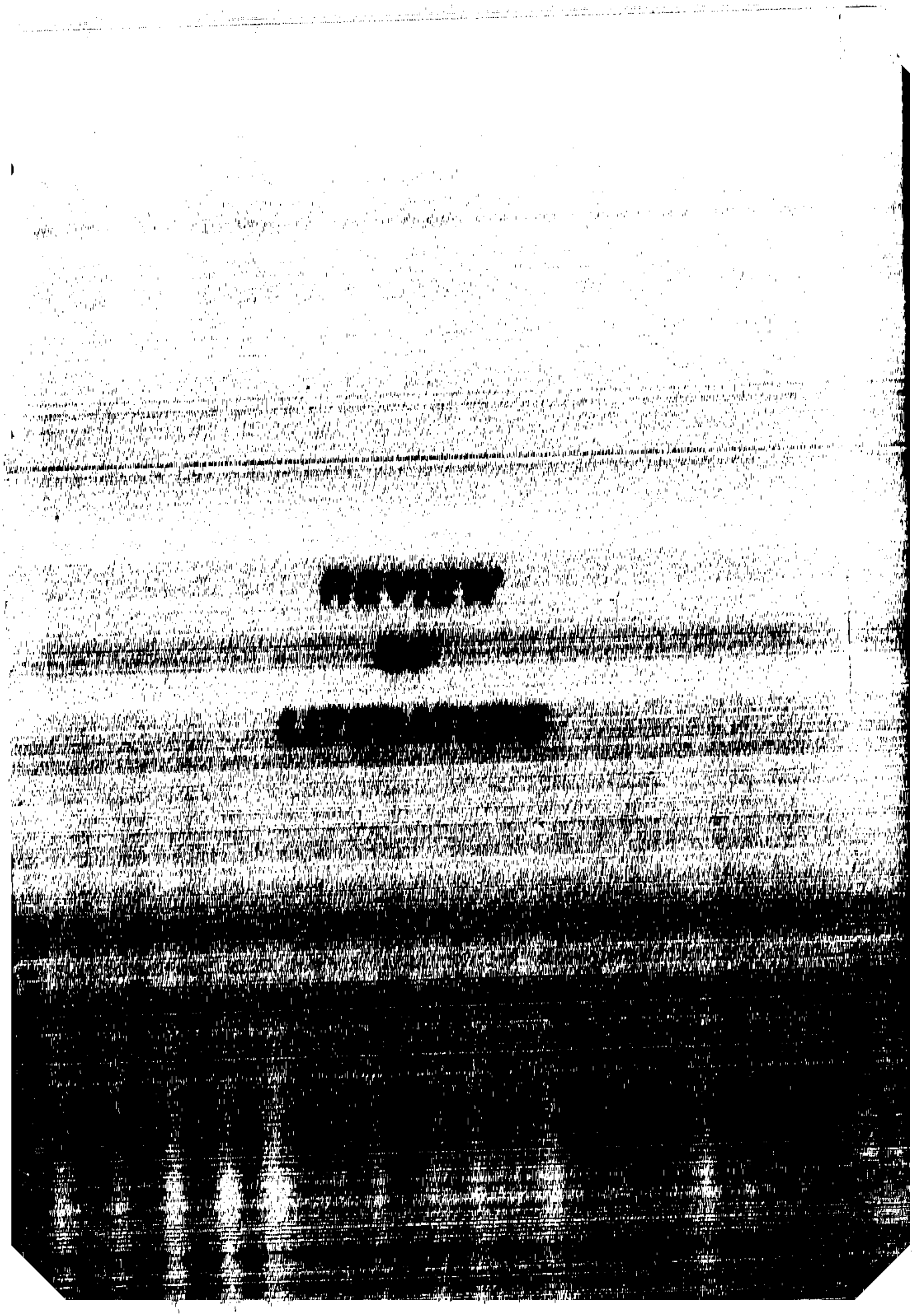
Different studies among asthmatics wood workers showed elevation in specific IgE levels to pliactic acid, which was considered to be the causative ingredient of asthma in wood dust **(Crofton and Douglas, 1981; Miguel et al., 1984; Cartier et al., 1986).**



AIM OF STUDY

1. To find out those with respiratory symptoms and diseases among workers exposed to wood dust.

2. To estimate immunoglobulins (IgE) level among those diagnosed, as cases of work related asthma, by clinical sheet and ventilatory function tests.



REVIEW OF LITERATURE

Anatomo-physiologic consideration of the lung:

The main function of the lung is to enable air to come into close proximity to the red blood cells flowing in the pulmonary capillaries, so that oxygen may be taken into the blood and carbon dioxide given off. The structural adaptations needed to produce the required functional relationship include development of the right ventricle and the pulmonary vascular system. The available internal surface area of an adult human lung is approximately 1 m^2/kg. body weight at 75% total lung capacity. The airways can be classified longitudenally into three groups. The cartilaginous airways (trachea and bronchi), the membranous airways (bronchioles) and th gas exchange airways (respiratory bronchioles and alveolar ducts). The cartilaginous airways include the trachea and all its branchings down to the small bronchi (0.3 to 0.1 cm. in diameter at TLC). The main structural supports for the bronchi are incomplete cartilaginous rings and plates connected by a strong fibrous layer. Within this layer is a layer of circularly arranged smooth muscle, the motor innervation of which is via the parasympathetic (vagus nerve). The sympathetic branch is inhibitory, but whether it directly innervates the airways smooth muscle is not known. The smooth muscle is also capable of being contracted (narrowing the airways) by direct stimulation. The innermost layer is the mucous membrane, the surface of which is pseudostratified ciliated epithelium.

Interspersed between the ciliated cells are the mucus-secreting goblet cells. The sensory innervation of the large airways includes a variety of undifferentiated fibres whose receptor endings appear to be adjacent to the surface epithelial layer. These fibres are sensitive to irritation and appear to be identical to the cough receptors. They are particularly abundant in the trachea and largest bronchi. The cartilaginous airways are the conduits through which gas enters and leaves the respiratory gas exchange areas of the lung. They do not allow gas exchange across their walls, they contribute to that portion of the freshly inspired breath that does not undergo gas exchange (the anatomical dead space). The resistance to gas flow in the bronchi can be increased by reflex or local stimulation of the surface smooth muscle which causes narrowing of these airways. Both inert particles, impinging on the airway surface, and chemically active agents may stimulate the irritant constrictor mechanism. Chronic irritant stimuli appear to cause hypertrophy of the mucus-secreting glands of the upper airways with a parallel increase in the production and secretion of airway mucus. The bronchioles, are direct continuations of the cartilaginous airways. They end in the terminal bronchioles (approximately 0.06 cm. in diameter). Bronchioles differ from bronchi mainly in the absence of the fibrocartilage framework and of secretory glands. The structural support of the bronchioles is from their being directly embeded into the connective tissue framework of the lung. They contain a circular continuous layer of smooth muscle. Their mucosa

is continuous with that of the bronchi. The epithelial layer consists of low, ciliated, columnar cells which gradually become cuboidal as the terminal bronchioles are approached. The bronchioles contribute to the dead space and airflow resistance, but normally the contribution is less than that of the bronchi. Both the dead space and the portion of airway resistance located in the bronchioles are affected by lung volume as these airways are embeded in the lung tissue and are dilated during lung inflation and narrowed during lung deflation. Oedema of the bronchiolar mucosa or active contraction of the circular smooth muscle, reflexly or by local irritation, can increase the resistance to airflow through these airways. The respiratory bronchioles and alveolar ducts are the final branchings of the airways. The main distinguishing feature of the gas exchange is the presence of the alveoli, in the walls of which the pulmonary capillaries are distributed and where 0_2 and $C0_2$ exchange occurs. The mucosa is a single layer of epithelial cells in the respiratory bronchioles, some of which contain cilia, there are no goblet cells. A new type of cells, believed to be a serous-secreting ones are present interspersed with the low cuboidal epithelium. There is no airway epithelium in the alveolar ducts. The gas exchange airways serve to distribute the inspired gas within the terminal respiratory units. These airways do not contribute to the anatomic dead space since they contain alveoli. They dilate and contract with lung volume and account for approximately one-third of the alveolar gas exchange volume (Staub, 1972).

Bronchial reactions due to exposure to vegetable dusts:

According to a **WHO report (1983)** vegetable dusts, when inhaled as aerosol, may exert a variety of harmful effects on the airways and lungs. The first effect is an allergic (atopic) response that occurs either in the upper airways (hay fever) or in the bronchi (bronchial asthma) or in both. The second type of response is byssinosis. The third type involves immunological changes in the lung parenchyma, which may become irreversible after prolonged exposure. The diseases caused by this type of response are collectively known as extrinsic allergic pneumonitis. The fourth type of response is simple non-specific irritation of the respiratory tract which, with prolonged exposure, may lead to chronic obstructive pulmonary disease "COPD". The irritation of the airways, probably through the receptors on the bronchial epithelium, gives rise to a cough reflex or an acute bronchoconstriction, and after repeated exposure, may cause an increase in the number of mucus-producing cells, which represents the first phase of chronic bronchitis. Since different dusts contain a wide range of different components, they may exert more than one type of response. An attempt is made to classify vegetable dusts according to the main response they evoke.

Dusts causing allergic airway response "Occupational asthma" **(WHO, 1983):**

Dust type	Worker affected	Other effects
Grain: Cereals and their products	Grain handlers, farm workers, Millers Bakers	COPD and extrinsic allergic Pneumonitis.
Wood dusts: Red cedar, iroko	Lumber and wood workers	COPD and exterinsic allergic pneumonitis
Teas	processors, blenders, packers.	Possibly COPD
Tobacco	processors, cutters, blenders	

Definition of terms:

Ciba Symposium (1959) proposed that emphysema should be defined anatomically, but chronic bronchitis clinically as chronic cough and phlegm for at least three months each year for at least two successive years; whereas asthma was defined as airways obstruction which varied spontaneously or in response to treatment and all the three were induced in the general term generalized obstructive airways disease.

According to the WHO definition, chronic bronchitis is a persistent increase of bronchial secretion. For epidemiological purposes may be defined as a productive cough at some time of the day for at least three months per year in two consecutive years. According to the degree of airways obstruction, one may distinguish two forms of the disease; nonobstructive chronic bronchitis and chronic bronchitis with airways obstruction (**WHO, 1980**).

Emphysema is a pathological condition of the lung, characterized by an increase beyond the normal size of the air spaces, distal to the terminal bronchioles with destruction of the walls. Emphysema may be suspected in patients with dyspnoea accompanied by functional changes such as hyperinflation, decrease of transfer factor, loss of elastic recoil, with or without hypoxaemia at rest (**WHO, 1980**).

Bronchial asthma is a disease characterized by acute attacks of shortness of breath induced by different agents or by exercise, accompanied by clinical signs of obstruction, totally or partially reversible between the attacks (**WHO, 1980**).

Asthma is common. It is estimated that perhaps 5% of the population has recognizable asthma in the course of a lifetime. Its prevalence is greatest amongst children (**Brewis, 1985**). According to **Crofton and Douglas (1981)** adults prevalence rates of asthma