1. Introduction
Man since time immemorial has been using herbs/plant products as medicine for developing immunity or resistance against diseases. The recent survey showed that about 40 percent prescriptions served in U.S.A contain one or more herbal drugs. More than 40 percent share of the medicine market in China belongs to traditional medicine and in rural areas 90 percent of the people rely and treat themselves through traditional health care; So, large number of herbal remedies coming out to treat so many dreadly ailments. Throughout Western culture, herbal medicine has donated many of the most potent medicines to the vast arsenal of drugs available to the Practitioners-both in crude form and as chemical model upon which medicines are manufactured; such is the popularity of herbalism that it remains the most common form of medicine available in the world today. Plants have proved to be better Chemists than humans, and many important phytochemical drugs are too difficult or impossible to synthesize.

India is one of the 12 leading biodiversity centres with the presence of over 45,000 different plant species. Our country has 15000-18000 flowering plants, 23,000 fungi, 2500 algae, 1600 lichens, 1800 bryophytes and 13 million microorganisms on its biodiversity region. From this flora 15000 to 20000 plants/micro-organism have good medicinal value. However out of this strong resources only about 7000 plants are used in Ayurveda, 600 in Siddha, 700 in Unani and 30 in modern medicine.

The herbal medicine industry is growing at an astounding rate. In the developed countries the interest in alternative medicine has increased 60 percent since last 10 years and the market is growing at the rate of 7 percent to 30 percent annually.
The increasing demand for herbal medicines inevitably led to the issue of obtaining and maintaining their quality. As a result, there has been a tremendous quality consciousness for the herbals in countries like U.S.A, Member of European Union, China and Australia etc. unfortunately however, in our country concept of quality did not get much attention. In this regards, an attempt has been made to review the current status of standardization of some herbs with special reference to chemical marker compound analysis. Standardization assures that products are reliable in terms of quality, efficacy performance and safety. So the consumer can be protected from exploitation. It also assures safety of user against hazard of medication. Standardization and development of reliable quality protocols for herbal formulation is one of such important issues. The objective of quality protocol development activities should be to ensure a 'minimum therapeutic guarantee' to the user. Such objective can be accomplished by ensuring a fairly consistent quality in the raw materials, a validated process control system and adequate checks on the finished products.

Asthma is one of the most common chronic diseases in the world. Perhaps the most commonly discussed respiratory disease after the common cold. The word asthma is derived from a Greek work meaning 'breathlessness or panting'' both of which accurately describe an attack of asthma. Asthma is a disease which claims many lives of people every year. The popularity of shared care in the management of asthma means that general Practitioner, Nurses and Pharmacy Staff are working together to get the best outcome for the patient.
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Asthma is not just a public health problem of the developed countries but also is the health problem of developing countries too; the incidence of this disease has become alarming. India has an estimated 40 million asthmatics. In India rough estimates indicate a prevalence of between 10 percent and 15 percent in 5-11 year old children. On 3rd May of every year, the World health Organization (WHO), together with its partners around the world, is observing World Asthma Day for many million asthmatic sufferers. The WHO recognizes asthma as a disease of major health importance and plays a role in the coordination of international efforts against the disease. (WHO)

Economic burden of asthma is very widespread in reality. In spite of the fact that an average 10 percent of the family budget will go towards the treatment for asthma sufferers. In 1998, the cost of asthma care was estimated to be US $ 11.3 billion in the United States of America; nearly a double increased from US $ 6.2 billion was estimated in 1990 (WHO 2000).

The human and economic burden associated with this condition is severe. But no clear data regarding economic burden are available in Indian society. The cost of the asthma to the society could be reduced to large extent though concerted international and national action

➢ Worldwide, the economic cost associated with asthma is estimated to exceed those of T.B. and HIV/AIDS combined.
➢ In the United States, for example, annual asthma care cost exceed $12.7 billion

At present Britain spends about US$1.8 billion of health care of asthma.\textsuperscript{11,12}

In the view of problems reported as above, it is thought worthwhile to undertake work on anti asthmatic herbal drugs.
1.1 Etiology of Bronchial asthma:

The strongest risk factors for developing asthma are wide range of provoking stimuli. 13-15

Fig. 1a Allergens for Asthma
Table 1 List of agents responsible as triggers in bronchial asthma:

<table>
<thead>
<tr>
<th>List of Agents</th>
<th>Events Triggering Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory infection</td>
<td>Respiratory syncytical virus (RSV), rhino virus, influenza, parainfluenza, Mycoplasma pneumonia</td>
</tr>
<tr>
<td>Allergens</td>
<td>Airborne pollens (grass, trees, weeds), house-dust mites, animal dander, cockroaches, fungal spores</td>
</tr>
<tr>
<td>Environment</td>
<td>Cold air, fog, ozone, sulphur dioxide, nitrogen, tobacco smoke, wood smoke.</td>
</tr>
<tr>
<td>Emotions</td>
<td>Anxiety, stress, laughter.</td>
</tr>
<tr>
<td>Exercise</td>
<td>Particularly in cold, dry climate.</td>
</tr>
<tr>
<td>Drugs</td>
<td>Aspirin, NSAIDs, sulphites, benzalkonium chloride, β-blockers.</td>
</tr>
<tr>
<td>Occupational stimuli</td>
<td>Bakers (flour dust); farmers (hay mold); spice and enzyme workers; printers (Arabicgum); chemical orkers (azodyes, anthraquinone, ethylenediamine, toluene. Diisocyanates, PVC); plastics, rubber and wood workers (formaldehyde, western cedar, dimethylethanolamine, anhydrides)</td>
</tr>
</tbody>
</table>
Table 2 Classification of Bronchial Asthma:

<table>
<thead>
<tr>
<th>Extrinsic asthma (allergic/atopic)</th>
<th>Intrinsic asthma (idiosyncratic/nonatopic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset usually in childhood or early adult life.</td>
<td>Onset usually (but not invariable) in older adults. Begins after the age of 30 and tends to perennial and more severe. Status asthmaticus is more common in this group.</td>
</tr>
<tr>
<td>Family history of multiple allergies (asthma, hayfever, eczema) common (50%) well defined allergic history to a variety of inhaled allergens (atopy)</td>
<td>Family history of multiple allergies less common (20%)</td>
</tr>
<tr>
<td>Known external allergens.</td>
<td>Not known external allergens</td>
</tr>
<tr>
<td>IgE raised in 50-60% of subjects.</td>
<td>IgE normal or low.</td>
</tr>
<tr>
<td>Other allergies (hayfever and eczema) often present (54%).</td>
<td>Other allergies uncommon</td>
</tr>
<tr>
<td>Positive immediate skin tests.</td>
<td>Negative skin tests.</td>
</tr>
<tr>
<td>Intermittent asthma.</td>
<td>More continuous asthma.</td>
</tr>
</tbody>
</table>
### Table 3 Types of asthma as per National Asthma Education and Prevention Program (NAEPP):

<table>
<thead>
<tr>
<th>Classification</th>
<th>Symptoms</th>
<th>Night symptoms</th>
<th>Lung function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1: Mild intermittent Asthma</strong></td>
<td>Symptoms occurring twice a week or less. No symptoms and normal PEF between exacerbations brief exacerbations (lasting a few hours to days) with variable intensity.</td>
<td>Symptoms occurring not more than twice a month</td>
<td>FEV₁/FVC is 80% or more of predicted PEF variability of less than 20%</td>
</tr>
<tr>
<td><strong>Step 2: Mild persistent Asthma</strong></td>
<td>Symptoms occurring more than twice a week Exacerbations may affect activity</td>
<td>Symptoms occurring more than twice a month</td>
<td>FEV₁/FVC is 80% or more of predicted PEFF variability of 20 to 30%</td>
</tr>
<tr>
<td><strong>Step 3: Moderate persistent Asthma</strong></td>
<td>Daily symptoms, daily use of inhaled short-acting beta agonist exacerbatons occur more than twice a week and may last for days.</td>
<td>Symptoms occurring more than once a week</td>
<td>FEV₁/FVC is greater than 60% but less than 80% of predicted PEF variability of greater than 30%.</td>
</tr>
<tr>
<td><strong>Step 4: Severe persistent Asthma</strong></td>
<td>Continual symptoms limited physical activity frequent exacerbations.</td>
<td>Frequent symptoms</td>
<td>FEV₁/FVC is 60% or less of predicted PEF variability of greater than 30%</td>
</tr>
</tbody>
</table>

PEF=Peak Expiratory Flow  
FEV₁=Forced Expiratory Volume in one second  
FVC=Forced Vital Capacity  
FEV₁/FVC % = FEV₁ as percentage of FVC.
1.2 Pathophysiology of Bronchial Asthma:

Asthma has been described primarily as an inflammatory process in the last one decade. The inflammatory process is now considered to be an immunologically initiated, mediator-driven event. The observation that asthma is associated with an inflammatory process in the lungs has dramatically changed the understanding of the pathophysiology and treatment of this disease.

![Structure of normal and asthmatic bronchiole](image)

Fig. 1b Structure of normal and asthmatic bronchiole

The asthmatic subject has intermittent attacks of dyspnea, wheezing and cough. In many subjects the asthmatic attack consists of two main phases as can be demonstrated by tests of FEV.

- The immediate phase
- The late phase

However, in some subjects, only one of the phases may be obvious.
1.2.1 The Immediate Phase:
Inhaled allergen challenged in allergic patients leads to an early allergic inflammatory reaction. Exposure to allergen leads to sensitization and formation of antibodies through differentiation of B-lymphocytes. It is initiated after activation of cells bearing allergen-specific Ig-E. Respiratory system, called as separate immune organ of the body, gets primed and ready to give allergic reaction after the second exposure. Interaction of allergen with mast cell fixed Ig E releases histamine along with LTC₄ and LTD₄, PGE₂, NK-A. Various chemokines and chemotaxins attracts inflammatory cells particularly eosinophila causing inflammation.₁⁶–₁⁸

1.2.2 The Late Phase:
It is a progressive inflammatory reaction occurs at variable time interval, generally 6-9 hrs after allergen provocation and may be nocturnal. Initiation of this phase occurs during first phase, the influx of Th2 lymphocytes is of particular importance. Inflammation here differs from other inflammatory reaction as it involves leakage of cells. It also infiltrates cytokines releasing Th2 cells, whose product causes damage and loss of epithelium. The other putative mediators of inflammatory process in late phase are adenosine, neuropeptides and bradykinin. Growth factors released from inflammatory cells act on smooth muscle cells, causing hypertrophy and hyperplasia. Eosinophil plays role as an inflammatory cell, as it secretes mediators- Eosinophilic Cationic Protein (ECP), Eosinophil-derived neurotoxin (EDNT), GM-CSF, TNF, PG and cytokines which results in epithelial shedding, bronchoconstriction and promotion of inflammation in
respiratory tract. However, in some subjects, only one of the phases may be obvious.
The gross pathology of asthmatic airways displays lung hyperinflation, smooth muscle hypertrophy, lamina reticularis thickening, mucosal edema, epithelial cell sloughing, cilia cell disruption, and mucus gland hyper secretion. Microscopically, asthma is characterized by the presence of increased numbers of eosinophils, neutrophils, and mucus. Initially, there is recruitment of leukocytes from the blood stream to the airway by activated CD4 T-lymphocytes. The activated T-lymphocytes also direct the release of inflammatory mediators from eosinophils, mast cells, and lymphocytes. In addition, the subclass 2 helper T-lymphocytes subset of activated T-lymphocytes produces Interleukin-4, IL-5 and IL-13. IL-4 in conjugation with IL-13 signals the switch from IgM to IgE antibodies. The cross-linkage of two IgE molecules by allergen causes mast cell to degranulate, releasing histamine, leucotrimes, and other mediators that perpetuate the airway inflammation. IL-5 activates the recruitment and activation of eosinophils. The activated mast cells and eosinophils also generate their cytokines that help to perpetuate the inflammation. Regardless of the triggers of asthma, the repeated cycles of inflammation in the lungs with injury to the pulmonary tissues followed by repair may produce long-term structural changes (remodeling) of the airways.17, 19-20

1.2.3 Asthma and immune System:
Ashtma has been described primarily as an inflammatory process in the last one decade. The inflammatory process is now considered to be
an immunologically initiated, mediator-driven event. The three main components of the immune system are
1. Antibodies
2. Inflammatory cells
3. Inflammatory mediators.

Antibodies are the specific proteins created by the immune system to identify and bind to foreign and potentially invading substances. Inflammatory cells circulate in the bloodstream and can “sense” the body’s surroundings or exposures to create immune responses directed against those exposures. Inflammatory mediators are chemical substances that are secreted by immune cells to induce an ongoing immune response generated against a specific exposure to the body.

1.2.3.1 Antibodies:

An antibodies or an immunoglobuline is a small protein molecule created by the immune system to have a close structural ‘fit’ to the surface of a foreign substance. The foreign substance is an antigen. The body manufactures five classes of antibodies, namely IgM, IgG, IgA, IgD, and Ig E.²¹

*Immunoglobuline E:*

The antibody class of allergic diseases, including allergic asthma, is IgE. It is fundamental to the allergic immune response. Although the usual antibody response to an antigen in some patients leads to generate IgM or IgG antibodies (or both), it is unclear why some antigens in some patients lead to generation of a specific IgE antibody response. An antigen that stimulates in IgE antibody is more specifically termed an allergen. These IgE antibodies are generally
directed against substances that are both harmful to the body, including pollens; from cat; certain mold spores; certain foods; certain drugs; and (most commonly) droppings from microscopic dust mites. After initial exposure of the patient to an allergen, the primary immune response is to generate unique IgE antibodies that become bound to the surface of mast cells. If the patient is later re-exposed to an allergen by inhalation, the allergen binds to the surface-bound IgE on the mast cells in the bronchi. Binding of at least two IgE molecules, bridged by a single allergen molecule, is termed Cross-linking. Cross-linking of IgE by allergen on the surface of the mast cell is the initial biologic event of an allergic reaction.

An allergic reaction can be technically referred to as an "immediate hypersensitivity" reaction; this term derives from the key aspects of an allergic reaction:

- The reaction occurs very quickly after exposure to the substance that stimulates the reaction (allergen). The reaction may occur (and may be life-threatening) 5-10 minutes or less after exposure to the allergen and thus, is termed immediate.
- The person having the allergic reaction is more sensitive (i.e., shows hypersensitivity) to the offending substance than one who is not allergic. A person without allergies would be expected to have absolutely no discernible reaction to the very same substance that could be fatal to one who has exquisite allergic sensitivity to that substance.

1.2.3.2. Inflammatory cells:
Inflammatory cells can be of three types
- Resident cells: mast cells, macrophages
**Mast cell:**

Following the release of IgE into the circulation, these cytokines bind to the high affinity IgE receptors in tissue mast cells are found throughout the walls of the respiratory tract, and threefold to fivefold increases have been described in the airways of atopic patients with asthma. Once binding and cross-linking of allergen to cell bound IgE occurs, mediators such as histamine, eosinophil and neutrophil chemotactic factors, leukotrienes (LT) B₄, C₄, and D₄, prostaglandins, platelet activating factor, and others are released from mast cells. Histologic examination has revealed degranulated mast cells in the airways of patients who have died from acute asthma attacks. Mast cell degranulation is believed to be the primary event that produces decreased lung function and symptoms immediately following inhalation of an allergen. In addition, infiltration of mast cells into the airway smooth muscle may be responsible for ongoing airway hyperresponsiveness in asthma.²²⁻²⁴

Mast cells degranulation is felt to be important mechanism for exercise-induced bronchospasm (EIB). In this case, the degranulation appears to be secondary to cooling or increased osmolarity of the bronchial fluids or both.

**Alveolar macrophages:**

The primary function of alveolar macrophages in the normal airway is to serve as "scavengers", engulfing and digesting bacteria and other foreign materials. They are found in large and small airways, ideally
located for affecting the asthmatic response. A number of mediators produced and released by macrophages have been identified, including platelet-activating factor, leukotriene B4, leukotriene C4, and leukotriene D4. Additionally, alveolar macrophages are able to produce neutrophil chemotactic factor and eosinophil chemotactic factor, which, in turn, further the inflammatory process.25

**Eosinophils:**
Eosinophils play an effect or role in asthma by release of proinflammatory mediators, cytotoxic mediators, and cytokines. Circulating eosinophils migrate to the airways by cell rolling, through interaction with selectins, and eventually adhere protein (vascular cell adhesion molecule 1 [VCAM-1] and intercellular adhesion molecule 1 [ICAM-1]). As eosinophils enter the matrix of the membrane, their survival is prolonged by interleukin 5 (IL-5) and Granulocyte-macrophage colony-stimulating factor (GM-CSF). On activation, eosinophils release inflammatory mediators such as leukotrienes and granule proteins to injure airway tissue.25

**Lymphocytes:**
Mucosal biopsy specimens from patients with asthma contain lymphocytes, many of which express surface markers of inflammation. There are two types of T-helper CD4+ cells. Type-1 T-helper (Th1) cells produce IL-2 and interferon-γ (IFN-γ), both essential for cellular defense mechanisms. Type-2 T-helper (Th2) cells produce cytokines (IL-4, IL-5, IL-6, IL-9 and IL-13) that mediate allergic inflammation. It is known that Th1 cytokines inhibit the production of Th2 cytokines, and vice versa. It is hypothesized that allergic asthmatic...
inflammation results from a Th2 mediated mechanism (an imbalance between Th1 and Th2).  

**Neutrophils:**
Neutrophils may play a pivotal role in the diseases process, at least in the sudden-onset fatal cases and in some patients with long standing or corticosteroid dependent asthma. The neutrophils can also be a source for a variety of mediators, including platelet-activating factor, prostaglandins, thromboxanes, and leukotrienes, that contribute to BHR and airway inflammation.

**Epithelial cells:**
Bronchial epithelial cells traditionally have been considered as a barrier, participating in mucociliary clearance and removal of noxious agents. However, epithelial cells also participate in inflammation by the release of eicosanoids, peptidases, matrix proteins, cytokines, and nitric oxide (NO). IgE dependent mechanisms, viruses, polluents, or histamines can activate epithelial cells. In fatal asthma, extensive epithelial shedding occurs. The integrity of airway epithelium may influence the sensitivity of the airways various provocative stimuli. Epithelial shedding include increased airway responsiveness, altered permeability of the airway mucosa, depletion of epithelial-derived relaxant factors, and loss of enzymes responsible for degrading proinflammatory neuropeptides.

**Adhesion molecules:**
Adhesion molecules help adhesion of the various cells to each other and the tissue matrix to facilitate infiltration and migration of these
cells to the site of infiltration. The adhesion molecules are divided onto families on the basis of their chemical structure. Some of which though to be important in inflammation includes the integrins, immunoglobulin supergene family, selectins and carbohydrate lignans including ICAM-1 and VCAM-1. Adhesion molecules are found on a variety of cells, such as neutrophils, monocytes, lymphocytes, basophils, eosinophils, granulocytes, platelets, endothelial cells, and can be activated by the many inflammatory mediators present in asthma.

**Fibroblasts and Myofibroblasts:**
Fibroblasts are found frequently in connective tissue. Human lung fibroblasts may behave as inflammatory cells on activation by IL-4 and IL-13. The myofibroblasts may contribute to the regulation of inflammation via the release of cytokines and to tissue remodeling. In asthma, myofibroblasts are increased in numbers beneath the reticular basement membrane, and there is an association between their numbers and the thickness of the reticular basement membrane.

**TH₁ and TH₂ cell imbalance:**
The Th1/Th2 imbalance contributes to the cause and evolution of atopic diseases, including asthma. The T-cell population in the cord blood of newborn infants is skewed toward a Th2 phenotype. The extent of the imbalance between Th1 and Th2 cells during the neonatal phase may predict the subsequent development of allergic diseases, asthma, or both. It has been suggested that infants at high risk of asthma and allergies should be exposed to stimuli that up regulate Th1 mediated responses in order to restore the balance during
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a critical time in the development of the immune system and the lung\textsuperscript{25}.

1.2.3.3 Inflammatory mediators:

Asthma is a complex chronic inflammatory disease of the airways that involves the activation of many inflammatory and structural cells, all of which release inflammatory mediators that result in the typical pathophysiological changes of asthma. Inflammatory mediators produce many effects in the airways, including bronchoconstriction, plasma exudation, mucus secretion, neural effects, and attraction and activation of inflammatory cells. There is increasing recognition that mediators may result in long-lasting structural changes in the airways that are also mediated by the release of inflammatory mediators. These changes may include fibrosis resulting from the deposition of collagen, which is seen predominantly under the epithelium even in patients with mild asthma. The airway smooth muscle layer is also thickened in asthma, and this is likely the result of increases in the number of smooth muscle cells (hyperplasia) and increases in their size (hypertrophy). There may be proliferation of airway vessels (angiogenesis) and of mucus-secreting cells. There may also be changes in the intervention of the airways. The role of a mediator in asthma may be difficult to assess when the mediator has a long term effect on airway function. It is easy to measure the effect of a mediator on airway smooth muscle, but it is more difficult to determine its effect on airway microvascular leakage and mucus secretion. However, prevention of the long term consequences of asthmatic inflammation, such as irreversible airway narrowing, may be an important goal of asthma therapy, and it is
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necessary to devise methods to investigate how mediators may affect these long term consequences of asthma.28-30

Histamine:
Histamine [2-(4-imidazole) ethylamine] is the first mediator implicated in the pathophysiological changes of asthma. Histamine is synthesized and released by mast cells in the airway wall and by circulating and infiltrating basophils. Although airway mast cells are likely to be the major cellular source of histamine in asthma, there is increasing evidence that basophils may be recruited to asthmatic airways and may release histamine in response to cytokine histamine-releasing factors. Acute release of histamine following an allergic or non-allergic condition may lead to bronchoconstriction, which can be attenuated by selective H1-receptor antagonists. There is evidence that histamine may also stimulate sensitized afferent nerves.31-33

Leukotrienes:
Leukotrienes (LTs) play an important role in the pathophysiological changes of asthma. LTs are potent lipid mediators produced by arachidonic acid metabolism in cell or nuclear membranes. They are derived from arachidonic acid, which is released from membrane phospholipids via the activation of phospholipase A2. Arachidonic acid is subsequently metabolized by the enzyme 5-LO, to produce LTs. Cys-LTs are very potent contractile agents for human bronchi, being approximately 1000 times more potent than histamine, and they elicit this effect via activation of cys-LT1 receptors. Cys-LTs increase mucus secretion, both directly via effects on goblet cells and submucosal gland cells and indirectly via the activation of airway
nerves, leading to reflex secretion from submucosal glands. Cys-LT production is increased in asthma in response to various challenges that worsen asthma. Cys-LTs are potent mediators of bronchoconstriction, plasma exudation, and mucus secretion, and there is now a growing body of evidence that they may also increase eosinophilic inflammation.\textsuperscript{34-38}

LTC\textsubscript{4}, LTD\textsubscript{4}, and LTE\textsubscript{4} increase the sensitivity of the airways to inhaled histamine, which may be because the leucotrienes may alter the excitability of afferent nerves. There, by increasing the sensitivity of the airways to indirect acting stimuli.

**Prostanoids:**
Immediately following acute antigen challenge of asthmatic subjects, increased levels of prostanglandin (PG) F2, PGD2 and thromboxane (TX) B2 are detected in bronchoalveolar lavage fluid, which are derived from macrophages, airway epithelium and activation of mast cells. When inhaled, these prostanoids cause bronchoconstriction and increase airway responsiveness to spasmogens unrelated to alterations in airway caliber, which suggests that prostanoids may play a greater role in modulating airway responsiveness.

Prostanoids are synthesized by cyclo-oxygenase (COX). Various proinflammatory cytokines stimulate the induction of COX-2 in human airway epithelium and smooth muscle in culture suggesting that during inflammation, COX-2 expression may be augmented. Some of the studies have shown that inhaled PGE2 inhibits the development of the late asthmatic response unrelated to functional antagonism of airway smooth muscle contraction and attenuates the attendant increase in sputum eosinophilia, while other studies have
shown that PGE2 may have proinflammatory properties, which might play a role in the development of the allergic response by down regulating interferon (IFN) γ and interleukin (IL)-12 productions from T-lymphocyte development. The potential anti- and pro-inflammatory properties of PGE2 may reflect activation of different prostanoid receptor subtype on different cells.39-44

**Bradykinins:**
The release of kinins within the airway could lead to the activation of bradykinin receptors including β1 receptors whose expression is regulated by inflammatory cytokines and β2 receptors present on various cells within the airway wall including vascular endothelium, airway smooth muscle, submucosal glands, nerves and airway epithelium. Clinical study suggests that bradykinin induces bronchoconstriction via activation of afferent nerves. Animal studies reveal that bradykinin simulates sub-population of afferent nerves, C-fibers and mediates the release of sensory neuropeptides from these. The increase in airway responsiveness to bradykinin correlates with the number of eosinophils in bronchoalveolar lavage fluid, bronchial biopsies and sputum.45,46

**Endothelins:**
Endothelins were originally discovered as potent vasoconstrictor peptides, which are encoded by three distinct genes. They are formed via the action of endothelin converting enzyme (ECE). The expression of mRNA for the endothelins and ECE has been documented in human bronchial epithelial cells and can be upregulated by a variety of pro-inflammatory cytokines. The biological effects of endothelins are
mediated via two receptors, designated as $\text{ET}_A$ and $\text{ET}_B$, which are characteristic of G-protein coupled receptors. Endothelin is a potent contractile agonist of human airway smooth muscle and augments cholinergic nerve mediated responses in human airway in-vitro, both effects mediated via the activation of $\text{ET}_B$ receptors. Few studies have examined the pro-inflammatory action of endothelins in the airways. $\text{ET}_A$ but not $\text{ET}_B$ receptor antagonists attenuated allergen induced recruitment of eosinophils in a murine model of inflammation in part by increases production of $\text{IFN}_\gamma$ from pulmonary lymphocytes. Endothelins can not be stored and requires de-novo synthesis, which may occur several hours after acute allergen challenge.\textsuperscript{47-52}

1.2.4 Growth Factors:

A variety of growth factors are though to play a role in altering the structure of the airways. A number of growth factors, including platelet derived growth factor (PDGF), transforming growth factor (TGF) and epidermal growth factor (EGF) has been investigated in bronchial biopsies from asthmatic subjects. A number of in-vitro studies have shown that PDGF is a potent mitogen of human airway smooth muscle. TGF$\beta$ is a potent stimulant for fibroblast mitogenesis and is important in wound healing and fibrosis plays a pleiotrophic role in the immune system and inhibits proliferation of airway smooth muscle. Eosinophils, fibroblasts and epithelial cells are the major sources of TGF$\beta$. EGF induces airway smooth muscle proliferation and ET-1 potentiates EGF induced airway smooth muscle proliferation.\textsuperscript{53-59}
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Proteases:
Tryptase, which is a mast cell serine protease affect fibroblast proliferation, degrade fibrinogen, generate C3a, simulate mucus secretion and degrade sensory neuropeptides. Thus, mast cell tryptase could play a role in regulation of haemostasis, mucus secretion and vascular permeability. Other proteases like thrombin induce proliferation of human airway smooth muscle.\textsuperscript{59-61}

Cytokines:
Cytokines are small protein mediators produced by different cell types including immune cells like T-lymphocytes that play an integral role in the coordination and persistence of inflammation in asthma. Th2 lymphocytes produce a panel of cytokines, including IL-3, IL-4, IL-5, IL-9, IL-10, IL-13, and GM-CSF. The primary signals that activate Th2 cells are unknown but may be related to the presentation of a restricted panel of antigens in the presence of appropriate cytokines. Dendritic cells are ideally suited to act as the primary contacts between the immune system and external allergens. Interaction of co-stimulatory molecules on the surface of antigen-presenting cells (in particular, the B7.2/CD28 interaction) may lead to proliferation of Th2 cells, thus perpetuating mast cell activation and eosinophilic inflammation. This may lead to the production of specific IgE by B lymphocytes under the influence of IL-4, which plays a critical role in the isotype switching of B lymphocytes from IgG to IgE production. Other cytokines, including TNF-\(\alpha\) and IL-6, may also be important. IL-4 also increases the expression of an inducible form of the low affinity receptor for IgE on B lymphocytes and macrophages. This may account for the increased expression of CD23 on alveolar
macrophages from asthmatic patients, which in turn could account for the increased release of cytokines from these macrophages. In addition, IL-4 is very important in driving the differentiation of CD4+ Th precursors into Th2-like cells. The differentiation, migration, and pathobiological effects of eosinophils may occur through the effects of GM-CSF, IL-3, and IL-5. Once recruited from the circulation, mature eosinophils in the presence of these cytokines change phenotype into hypodense eosinophils, which show increased survival in bronchial tissue. These eosinophils are primed for ligand-initiated generation of increased amounts of cys-LTs and for cytotoxicity to other cells, such as those of the airway epithelium.

Airway macrophages are normally poor at antigen presentation and suppress T cell proliferative responses [possibly via release of cytokines such as IL-1 receptor antagonist (IL-1ra)], but in asthma there is evidence for reduced suppression after exposure to allergen. Both GM-CSF and IFN-γ increase the ability of macrophages to present allergen and express HLA-DR. IL-1 is important in activating T lymphocytes and is an important co-stimulator of the expansion of Th2 cells after antigen presentation. Airway macrophages may be an important source of "first wave" cytokines, such as IL-1, TNF-α, and IL-6, which may be released upon exposure to inhaled allergens. These cytokines may then act on epithelial cells to release a second wave of cytokines, including GM-CSF, IL-8, and the regulated on activation, normal T cell-expressed, and secreted protein (RANTES), which amplify the inflammatory response and lead to influx of secondary cells such as eosinophils, which themselves may release multiple cytokines. Cytokines may also exert an important regulatory effect on the expression of adhesion molecules, both on endothelial
cells of the bronchial circulation and on airway epithelial cells. IL-4 increases the expression of vascular cell adhesion molecule-1 (VCAM-1) on endothelial and airway epithelial cells, and this may be important in eosinophil and lymphocyte trafficking. IL-1 and TNF-α increase the expression of ICAM-1 in both vascular endothelium and airway epithelium.\textsuperscript{62-67}

**Chemokines:**
Chemokines are chemotactic cytokines, which are potent chemoattractant of eosinophils, basophils, monocytes and T-lymphocytes. They are classified into two major groups
(a) CXC chemokines, in which the cysteine residues are separated by an amino acid and
(b) CC chemokines, in which the cystein residues are adjacent to each other. Exacerbation of asthma leads to the synthesis and release of various chemokines, which can contribute to the recruitment of inflammatory cells to the airways. Chemokines are known to exert their effect through chemokine receptors CCR3, which belong to rhodopsin like G-protein coupled receptors. CCR3 mediates biological effects of eotaxin and other eosinophil chemokines, such as RANTES, monocyte chemotactic protein (MCP-3, MCP-4) and is expressed predominantly on eosinophils.\textsuperscript{68}

**Mucus Production:**
The mucociliary system is the lung’s primary defense mechanism against irritants and infectious agents. Mucus composed of 95% water and 5% glycoprotein is produced by bronchial epithelial glands and goblet cells. Mucus either too viscous or too watery will not be
transported optimally. The exudative inflammatory process and sloughing of epithelial cells into the airway lumen impair mucociliary transport. The bronchial glands are increased in size and the goblet cells are increased in size and number in asthma. Expectorated mucus from patients with asthma tends to have a high viscosity.69

**Neural control/Neurogenic inflammation:**
The airway is innervated by parasympathetic, sympathetic and nonadrenergic inhibitory nerves. The normal resting tone of human airway smooth muscle is maintained by vagal efferent activity. The nonmyelinated C fibres of the afferent system lie immediately beneath the tight junction between epithelial cells lining the airway lumen. These endings probably represent the irritant receptors of the airways. Stimulation of these irritant receptors produces reflex bronchoconstriction.
The nonadrenergic, noncholinergic nervous system has been described in the trachea and bronchi. Substance-P, neurokinin-A, neurokinin-B, Vasoactive intestinal peptide are the best characterized neurotransmitters in the NANC nervous system. VIP is an inhibitory neurotransmitter. Inflammatory cells in asthma can release peptidases that can degrade VIP, producing exaggerated reflex cholinergic bronchoconstriction. The NANC system may play an important role in amplifying inflammation in asthma by releasing NO.69

**Airways remodeling:**
Acute inflammation is a beneficial, nonspecific response of tissues to injury and generally leads to repair and restoration of the normal structure and function. In contrast, asthma represents a chronic
inflammatory process of the airways followed by healing. The end result may be an altered structure referred to as a remodeling of the airways. Repair involves replacement of injured tissue by parenchymal cells of the same type and replacement by connective tissue and its maturation into scar tissue. In asthma, the repair process can be followed by complete or altered restitution of airways structure and function, presenting as fibrosis and in smooth muscle and mucus gland mass. 27

Nitric Oxide: 70
Nitric Oxide is produced by cells within the respiratory tract. It has been thought to be a neurotransmitter of the NANC nervous system. Endogenous NO is generated from the amino acid L-arginine by the enzyme NO synthase. There are three isoforms of NO synthase. One isoform is induced in response to proinflammatory cytokines, inducible NO synthase (iNOS), in airway epithelial cells and inflammatory cells of asthmatic airways. NO produces smooth muscle relaxation in the vasculature and bronchioles; however, it appears to amplify the inflammatory process and is unlikely to be of therapeutic benefit. Recent investigations measuring exhaled NO concentrations have suggested that it may be a useful measure of ongoing lower airways inflammation in patients with asthma and for measuring effectiveness of therapy.

1.3 Signs and symptoms of asthma:
Signs and symptoms of asthma can range from mild to severe. You may have only occasional asthma episodes with mild, short-lived symptoms such as wheezing. In between episodes you may feel
normal and have no difficulty in breathing. Some people with asthma have chronic coughing and wheezing punctuated by severe asthma attacks. Warning signs precede most asthma attacks. Recognizing these warning signs and treating symptoms early can help prevent attacks or keep them from becoming worse.

Warning signs and symptoms of asthma in adults may include:

- Increased shortness of breath or wheezing
- Disturbed sleep caused by shortness of breath, coughing or wheezing
- Chest tightness or pain

Increased need to use bronchodilators medications that open up airways by relaxing the surrounding muscles. A fall in peak flow rates as measured by a peak flow meter, a simple and inexpensive device that allows you to monitor your own lung function.  

Children often have an audible whistling or wheezing sound when exhaling and frequent coughing spasms.

1.3.1 Diagnosis:

A doctor suspects asthma largely on the basis of a person's report of characteristic symptoms. A diagnosis of asthma can be confirmed using spirometry tests. During an asthma attack, the test reveals decreased airflow, but over hours or days, narrowing improves and is therefore reversible. More commonly, the doctor performs spirometry or pulmonary function tests before and after giving the person an inhaled beta-adrenergic agonist. If results are significantly better after the person receives the beta-adrenergic agonist, asthma is thought to be present. If the airways are not narrowed at the time of the first test, a diagnosis can be confirmed by a test in which the person inhales a
Introduction

chemical (usually methacholine but histamine may be used also) in
doses too low to affect a normal person but which causes airway
narrowing in a person who has asthma.

Spirometry is also used to assess the severity of the airway obstruction
and to monitor treatment. Peak expiratory flow (the fastest rate at
which air can be exhaled) can be measured using a small handheld
peak flow meter. Often, this test is used at home to monitor the
severity of asthma. Usually, peak flow rates are lowest between 4:00
and 6:00 a.m. and highest at 4:00 p.m. However, more than a 30%
difference in rates at these times is considered evidence of moderate to
severe asthma.

Determining up to what triggers a person's asthma is often difficult.
Allergy testing is appropriate when there is a suspicion that some
avoidable substance is stimulating attacks. Skin testing can help
identify allergens that may trigger asthma symptoms. However, an
allergic response to a skin test does not necessarily mean that the
allergen being tested is causing the asthma. The person still has to note
whether attacks occur after exposure to this allergen. If a doctor
suspects a particular allergen, a blood test that measures the level of antibody produced in response to the
allergen (the radioallergosorbent test [RAST]) can be performed to
determine the degree of sensitivity.

To test for exercise-induced asthma, an examiner uses spirometry
before and after exercise on a treadmill or stationary bicycle to
measure forced expiratory volume in 1 second. If the forced expiratory
volume in 1 second decreases more than 15%, the person's asthma can
be induced by exercise.
A chest x-ray is not generally helpful in diagnosing asthma. Doctors use chest x-rays when considering another diagnosis. However, a chest x-ray is often obtained when a person with asthma needs to be hospitalized or is treated in the emergency department with severe asthma.

1.3.2 Treatment:
Asthma cannot be cured, but it can be controlled with proper asthma management. The first step in asthma management is environmental control. Asthmatics cannot escape the environment, but through some changes, they can control its impact on their health.

Listed below are some ways to change the environment in order to lessen the chance of an asthma attack:

- Clean the house at least once a week and wear a mask while doing it
- Avoid pets with fur or feathers
- Wash the bedding (sheets, pillow cases, mattress pads) weekly in hot water
- Encase the mattress, pillows and box springs in dust-proof covers
- Replace bedding made of kapok or foam rubber with synthetic materials
- Consider replacing upholstered furniture with leather or vinyl
- Consider replacing carpeting with hardwood floors or tile
- Keep the humidity in the house low

The second step is to monitor lung function. Asthmatics use a peak flow meter to gauge their lung function. Lung function decreases before symptoms of an asthma attack - usually about two to three days
prior. If the meter indicates the peak flow is down by 20 percent or more from your usual best effort, an asthma attack is on its way. 71

The third step in managing asthma involves the use of medications.

Drug Treatment Goals: Asthma

- Reverse inflammation and bronchoconstriction
- Achieve normal or near normal pulmonary function
- Decrease frequency of attacks

There are two major groups of medications used in controlling asthma

1. Bronchodilators
2. Anti-inflammatory agents (corticosteroids)

1.4 Clasification of antiasthmatic drugs:

1.4.1 Bronchodilators:

- Beta-adrenergic agonists: e.g. Metaproterenol, terbutaline, albuterol, formoterol, bitolterol, salmeterol, pirbuterol.
- Anticholinergics: e.g. Ipratropium bromide, tiotropium bromide.
- Methylxanthines: e.g. Theophylline, Aminophylline.

1.4.2 Anti-inflammatory agents:

- Corticosteroids: e.g. Prednisolone, Dexamethasone

There are several types of medications available for treating asthma. Most people use a combination of long-term control medications and quick relief medications. Your doctor can help you decide which option is best for you based on your age and the severity of your symptoms. In general, the main types of asthma medications are:
**Introduction**

**Long-term-control medications:** These are used regularly to control chronic symptoms and prevent asthma attacks.

**Quick-relief medications:** You use these as needed for rapid, short-term relief of symptoms during an asthma attack.

**Medications for allergy-induced asthma:** These decrease your body's sensitivity to a particular allergen and prevent your immune system from reacting to allergens.

### 1.5 Ayurvedic concept of Asthma:

Ayurveda originated in India long back in pre-vedic period. Rigveda and Atharva-veda (5000 years B.C.), the earliest documented ancient Indian knowledge have references on health and diseases. Ayurved texts like Charak Samhita and Sushruta Samhita were documented about 1000 years B.C. The term Ayurveda means ‘Science of Life’. It deals elaborately with measures for healthful living during the entire span of life and its various phases. Besides, dealing with principles for maintenance of health, it has also developed a wide range of therapeutic measures to combat illness. These principles of positive health and therapeutic measures relate to physical, mental, social and spiritual welfare of human beings. Thus Ayurveda becomes one of the oldest systems of health care dealing with both the preventive and curative aspects of life in a most comprehensive way and presents a close similarity to the WHO’s concept of health propounded in the modern era.

The ancient ayurvedic system of medicine has an elaborate description of this disease from the earliest times. Shwasa word in normal terminology means respiration. Looking into Sanskrit origin of the word ‘Shwasa’, it derives its root from “swasha jeevane” meaning the
existence of life through prana vayu. In the present context; shwasa means disease pertaining to breathing. According to ayurveda, different types of shwasa (asthma) are Kshudra Shwasa, Maha Shwasa, Urdhva Shwasa, Chhinna Shwasa, Tamak Shwasa. The Acharayas have further classified Tamaka Shwasa into Pratamak and Santamak Shwasa.

1.5.1 Kshudra Shwasa
When Maruta (Vata) on being aggravated by exertion & overeating, produces Kshudra Shwasa, which subsides by, itself (without any medication) i.e. heavy breathing on exertion, passes of soon by rest & is thus not very troublesome. This condition does not give much pain; it does not interfere in the course of food and breathing. It does not disturb the sensory organs. This condition is mainly because of excessive intake of ruksha eatable and excessive exercise. It is however not harmful to the body as compared to the other types of shwasa.

1.5.2 Maha Shwasa
The patient feels heavy breathing, helplessness (unable to withstand the trouble). Respiration is accompanied with sounds resembling those of high pitch sounds from the nose, resembling that of a bull in heat. Common sense & intelligence are lost; eyes & face are unsteady, chest constricted and obstruction of urine & feces. The other symptoms are broken voice, dryness of throat, frequent delusions and severe pain in ears, temples & head. This condition is caused because of disturbance in respiratory movement of Vayu. In this condition, usually the voluntary control disappears and the wheezing sounds are audible.
from a distance. The allopathic system of medicine indicates such conditions in Biot’s breathing which is generally found in heart, kidney and brain disorders as a complication. Ayurveda describes it as a dyspnoea major where the patient generally scumbs to it.

1.5.3 Urdhva Shwasa
Under this condition there is prolonged upward breathings (expiration) but difficult respiration, obstructed movement of vata, An upward gaze, rolling eye balls, terrified look, severe pain as though his vital organs are being cut, speech choked. The ayurvedic physicians as harmful for the life describe such condition. The allopathic system of medicine under stertorous breathing and falling of inspiration describe such conditions. Such a condition can be found in pneumonia, abscesses of the lungs, gangrene or acute inflammation in the lungs and also in different types of epilepsy.

1.5.4 Chhinna Shwasa
Patient breathes with interruptions, cutting pain in the vital organs, sweating, fainting, flatulence, burning sensation, obstruction in the urinary bladder, eyes are unsteady (full of tears), delusion (or coma), one eye is angry - (red in colour), dryness of mouth, irrelevant talks, feeling of helplessness (inability to do anything), loss of chhaya (complexion), loss of consciousness. The patient normally breaks down with such a difficult breath ultimately losing his life. The allopathic system of medicine groups such condition under interrupted respiratory dyspnoea (Cheyne-stoke’s respiration).
1.5.5 Tamaka Shwasa (brochial asthma)

Under this condition patient feels pain in head, neck, chest & flanks, cough accompanied with cracking sound, delusion, loss of taste, appetite, running nose, thirst forceful bouts of respiration, feeling of going into darkness (i.e. losing consciousness of the surrounding) momentary comfort after expectoration, inability to breathe on lying, comfortable on sitting, eyes wide open, perspiration on forehead, dry mouth, desire for hot comforts. Acharya Sushruta has clearly defined shwasa as condition in which prana vayu leaving its normal functions moves upward associated with kapha and thus producing igasing and labored breathing. It is classified into two types i.e. Pratamaka Shwasa and Santamak Shwasa. Febrile dyspnoea appears in a patient with fever and fainting in Pratamaka Shwasa. It is excited by misperistalsis, inhalation of dust, indigestion, old age or debilitated condition or the suppression of natural urges while Santamak Shwasa or cardiac asthma is greatly during night and alleviated by cold medicines and in which the patient feels as if he is submerged in a sea of darkness.72,73

1.6 Holistic approaches for asthma:

A set of breathing exercises called Buteyko breathing techniques has been reported to significantly reduce the need for prescription drugs for people with asthma. Although the people in this controlled trial experienced an improved quality of life while doing these exercises, objective measures of breathing capacity did not improve, despite the decreased need for drugs.74
1.6.1 **Antibiotic** use during the first year or two of life has been associated with an increased risk of asthma in preliminary studies. Whether, this association might result from allergic versus non-allergic effects remains unknown. However, the association does suggest that, until more is known, gratuitous use of antibiotics in early childhood (e.g., to inappropriately treat viral diseases) should be reconsidered. Of course, the appropriate use of antibiotics in the treatment of infections as necessary should not be avoided. Concerns should be discussed with the prescribing physician.\textsuperscript{75,76}

1.6.2 **Acupuncture** might be useful for some asthmatics. Case reports and preliminary trials have suggested acupuncture may be helpful for people with asthma, either as a treatment for an acute attack or as a longer term therapy for reducing the number or severity of attacks, decreasing the need for medications, and so on. Placebo-controlled trials using sham ("fake") acupuncture, however, have been quite contradictory, many of them showing a strong placebo effect that is not significantly improved upon by real acupuncture. It is possible that needle insertion in non-acupuncture points has a stimulating effect that benefits asthma. The success of acupuncture may also depend on other factors, such as the type of asthma being treated and certain characteristics of the patient. Nonetheless, since some controlled research has demonstrated positive effects of real acupuncture, people with asthma may want to consider a trial of acupuncture treatment to see if it helps their individual cases.\textsuperscript{77-86}
1.6.3 Chiropractic

Physicians have reported that manipulation may be helpful for patients with asthma. In a controlled study, chronic asthmatics received either real or sham chiropractic manipulations for four weeks, after which the treatments were switched for another four weeks. No improvement in measurements of lung function was found at the end of the study. In addition, while both the manipulation and the sham treatment groups reported significant decreases in asthma frequency and severity, there were no differences between the treatments. A larger controlled study compared chiropractic manipulation to sham manual treatments in children whose asthma was still a problem despite usual medical management. Both groups experienced a significant decrease in symptoms and need for medication, as well as small increases in ability to breathe. These benefits lasted for four months after the treatments were discontinued. Although there was no additional benefit of chiropractic compared to the sham treatments, it is possible that improvements in both groups were real, rather than placebo effects. The sham therapy, which consisted of “soft tissue massage and gentle palpation [touching],” may have had real effects. More research is needed to address this confusing issue.87-91

1.6.4 Herbs and Asthma:

The medicines meant for treating Allergy & Asthma treat only the symptoms. They do not treat the underlying cause. The medicines used for Asthma have numerous undesirable side effects. The corticosteroids are among the most widely used drugs for the treatment of Type 1 hypersensitivity diseases like Asthma. The steroid based Asthma inhalers are fraught with various complications.
Steroids weaken the bones and stunt growth. The side effects of steroids can range from dental complications (weakening of teeth due to calcium depletion) to diabetes to impotence.

None of the medicines can/or actually intended to heal person with asthma. In almost all the cases the person is made to take an assortment of medicines and steroid sprays life long. The dosages are periodically increased as the person builds up tolerance.

Asthma can never be cured or controlled by pumping our body with factory made medicines, laboratory designed molecules, artificially made chemicals and steroids. It is the artificial environment like this that caused asthma in first place.92

Certain combination of herbs have similar properties as steroids in enhancing ease of breathing and building up strong immunity against allergy without any side effects.

The history of drugs is intimately linked with plants from the earliest times and even today plant products have extensive use in ethnomedicinal traditional system of medicine as well as in the armamentarium of the modern physician. In many developing countries, phytopharmaceuticals form the main base of national health care program. This perhaps is not only reason for the developing countries to invert in plant based pharmaceuticals. There has also been a global resurgence of interest in plant based drugs. The developing countries could have a sizable share of this market, endowed as they are not only with a large and varied endemic flora of wide variety of climate zones but also with a rich heritage of traditional system of medicine using plant based drugs in healthcare.

Many ayurvedic plants have been describe to be useful in the treatment of various bronchial disorders including bronchial asthma.
The use of medicinal plants and natural products increased dramatically in the last two decades in all over the world. More than 400 medicinal plant species have been used ethnopharmacologically and traditional to treat the symptoms of asthmatic and allergic disorder worldwide.

The following drugs are found to have anti-asthmatic activity and are used in the treatment of bronchial asthma.93-96

Table 4 List of some Antiasthmatic plant drugs:

<table>
<thead>
<tr>
<th>Plant</th>
<th>Part used</th>
<th>Extract/Active principle</th>
<th>Proposed mechanism of action</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Adhatoda vasica</em></td>
<td>Leaves, roots</td>
<td>Alkaloids</td>
<td>Bronchodilator, Anti-anaphylactic</td>
</tr>
<tr>
<td>(Acanthaceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Albizia lebbeck</em></td>
<td>Stem bark</td>
<td>Aqueous extract</td>
<td>Mast cell stabilizing activity</td>
</tr>
<tr>
<td>(Mimosaceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Boswellia serrata</em></td>
<td>Root</td>
<td>Boswellin, boswellic acids</td>
<td>Inhibit leukotriene biosynthesis and block synthesis of 5-lipoxygenase products including 5-HETE and LTB4</td>
</tr>
<tr>
<td>(Bursaraceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Cedrus deodara</em></td>
<td>Wood</td>
<td>Himacholol</td>
<td>Mast cell stabilizing activity</td>
</tr>
<tr>
<td>(Pinaceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Centipeda minima</em></td>
<td>Whole plant</td>
<td>Pseudoguainolid sesquiterpene lactones, flavonoids</td>
<td>Inhibits passive cutaneous anaphylaxis in rats</td>
</tr>
<tr>
<td>(Asteraceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Curcuma longa</em></td>
<td>Rhizome</td>
<td>Tumerones, curcuminoinds</td>
<td>Inhibits histamine release from rat peritoneal mast cells</td>
</tr>
<tr>
<td>(Zingiberaceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Clerodendron serratum</em></td>
<td>Leaves</td>
<td>Aqueous extract</td>
<td>Bronchodilator</td>
</tr>
<tr>
<td>(Verbenaceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Inula racemosa</em></td>
<td>Roots</td>
<td>Aqueous alcoholic extract</td>
<td>Anti-histaminic, anti-5-HT</td>
</tr>
<tr>
<td>(Asteraceae)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plant Name</td>
<td>Part Used</td>
<td>Extract/Component Used</td>
<td>Activity</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-----------------</td>
<td>---------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Picrorrhiza kurroa</td>
<td>Roots</td>
<td>Picrorrhizin</td>
<td>Inhibits histamine release and SRS-A.</td>
</tr>
<tr>
<td><em>Sarcostemma brevistigma</em></td>
<td>Twigs</td>
<td>Alkaloidal fraction</td>
<td>Inhibits passive cutaneous anaphylaxis in rats</td>
</tr>
<tr>
<td><em>Tephrosia purpurea</em></td>
<td>Whole plant</td>
<td>Ethanolic extract</td>
<td>Bronchodilatory, Antianaphylactic</td>
</tr>
<tr>
<td><em>Tinospora cordifolia</em></td>
<td>Stem</td>
<td>Aqueous extract</td>
<td>Mast cell stabilizing activity, Antihistaminic</td>
</tr>
<tr>
<td><em>Tylophora indica</em></td>
<td>Whole plant</td>
<td>Indolizidine alkaloids</td>
<td>Bronchodilatory, membrane stabilizing</td>
</tr>
<tr>
<td><em>Vitex negundo</em></td>
<td>Leaves</td>
<td>Alcoholic extract</td>
<td>Bronchodilatory, membrane stabilizing</td>
</tr>
<tr>
<td><em>Benincasa hispida</em></td>
<td>Roots</td>
<td>Root powder with water</td>
<td>Bronchospasmolytic</td>
</tr>
<tr>
<td><em>Moringa oleifera</em></td>
<td>Root bark</td>
<td>Alkaloid</td>
<td>Broncholytic</td>
</tr>
<tr>
<td><em>Ocimum sanctum</em></td>
<td>Herb</td>
<td>decoction</td>
<td>Bronchodilators, Mast cell stabilizer</td>
</tr>
<tr>
<td><em>Opuntia dillentii</em></td>
<td>Fruit</td>
<td>Syrup</td>
<td>Antispasmodic and Expectorant</td>
</tr>
<tr>
<td><em>Pavetta crassipes</em></td>
<td>Leaves</td>
<td>Aqueous extract</td>
<td>Bronchodilators</td>
</tr>
<tr>
<td><em>Allium cepa</em></td>
<td>Bulb</td>
<td>juice</td>
<td>Mast cell stabilizer</td>
</tr>
<tr>
<td><em>Azadiracta indica</em></td>
<td>Bark</td>
<td>Aqueous extract</td>
<td>Mast cell stabilizer</td>
</tr>
<tr>
<td><em>Bacopa monnieri</em></td>
<td>Entire herb</td>
<td>Aqueous extract</td>
<td>Mast cell stabilizer</td>
</tr>
<tr>
<td><em>Calotropis procera</em></td>
<td>Powdered flower</td>
<td>Aqueous extract</td>
<td>Mast cell stabilizer</td>
</tr>
<tr>
<td><em>Euphorbia hirta</em></td>
<td>Entire herb</td>
<td>Hydroalcoholic extract</td>
<td>Smooth muscle relaxant</td>
</tr>
<tr>
<td><em>Sphaeranthus indicus</em></td>
<td>Aerial parts</td>
<td>Hydroalcoholic extract</td>
<td>Bronchodilators</td>
</tr>
<tr>
<td><em>Cassia alata</em></td>
<td>Leaves and flower</td>
<td>Decoction</td>
<td>Mast cell stabilizer</td>
</tr>
</tbody>
</table>
Following plants also reported for their anti asthmatic effects.