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# A Comparative Study of Montelukast and Salbutamol in Bronchial Asthma

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## ABSTRACT

Current asthma drug therapy is highly effective, having evolved from naturally occurring substances via logical pharmaceutical developments. Pharmacology has played an important role in the development of asthma drugs, and several key experimental findings have been published in this journal. Understanding the pharmacology of effective drug therapies has also taught us a lot about the mechanisms underlying asthma. 2-Adrenoceptor agonists, which evolved from catecholamines in the adrenal medulla, are the most effective bronchodilators, whereas corticosteroids, which evolved from catecholamines in the adrenal cortex, are by far the most effective controllers of the underlying inflammatory process in the airways. A combination inhaler containing a long-acting 2-agonist and a corticosteroid - an improved form of adrenal gland extract - is the current "gold standard" of asthma therapy.

Theophylline, a dietary methyl xanthine, and chromoglycate, a plant-derived substance, have both been widely utilized in the treatment of asthma, but their molecular mechanisms are still unknown. Pharmacology has been crucial in enhancing natural products to create effective, long-lasting, and secure asthma medications, but it has faced difficulties in developing new classes of anti-asthma treatments. Leukotriene antagonists, the only brand-new type of anti-asthma therapy established in the previous 30 years, are less efficient than currently available medications. Corticosteroids are less successful than new, more focused medicines that target particular cytokines, but more focused medications run the risk of having side effects that may not be tolerable. Pharmacology, not molecular genetics, appears to be the most likely direction for future advancements in asthma treatment.

**Keywords :** Bronchial Asthma, Bronchodilator, Muscarinic antagonist, Theophylline , Corticosteroids.

## I. INTRODUCTION

Chronic inflammatory and obstructive lung diseases known as bronchial asthma are varied, complex, and characterized by increased mucus production, airway, and bronchial hyperactivity, and, over time, structural and functional alterations in lung tissue. A third of the population is currently believed to suffer from asthma. On the planet, there are thought to be 235 million humans. Recurrent episodes of acute shortness of breath, which typically happen at night or in the early morning, are the primary symptom of bronchial asthma. A constriction in the chest as well as other symptoms like coughing and wheezing are also present. Asthma symptoms can appear often following demanding exertion.[1,2]

With the aid of asthma guidelines, the majority of asthmatics are under good control. But only a small proportion of asthma sufferers (5–10%) have severe asthma that is unresponsive to high doses of inhaled corticosteroids. For the management of severe asthma, phosphodiesterase inhibitors, long-acting 2 agonists (LABA) inhibitors, and/or anti-cholinergics with an extended half-life (LAMA) are also recommended, along with steroid injections and a second controller in case things get out of hand. In 2004, there were 2141 deaths attributed to asthma, according to the Statistisches Bundesamt in Germany. According to the World Health Organization (WHO), asthma is responsible for 15 million DALYs (disability-adjusted life years) lost annually, or 1% of all DALYs lost globally as a result of illness. This assessment serves as a diagnosis.[2,3]

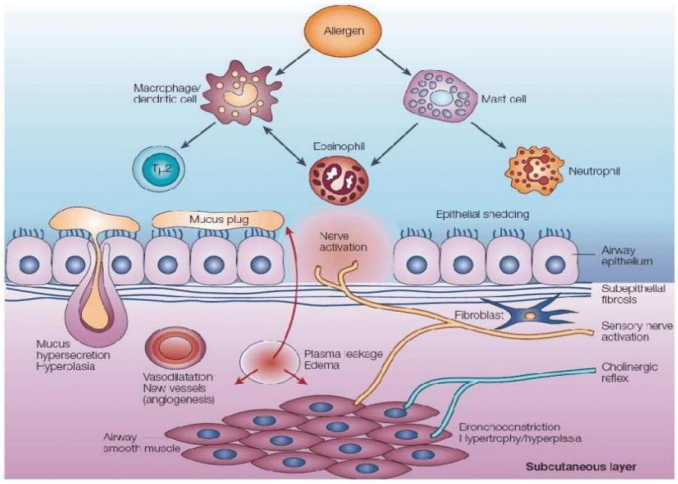
### Physiology of Bronchial Asthma:

Asthma symptoms are known to be exacerbated by cold air, intense emotional arousal, physical activity, aspirin, and other NSAIDs, beta-blockers, indoor allergens (such as pet dander and house dust mites in bedding, carpets, and stuffed furniture), outdoor allergens (particularly molds and pollen), tobacco smoke, chemical irritants in the workplace, and air pollution. In several published research, severe asthma phenotypes have been linked to genetic factors, age of onset, duration of the disease, flare-ups, concurrent si1nus disease, and inflammatory characteristics. Broncho alveolar lavage and endobronchial biopsies were used in the first evaluation of asthma patients and identified various inflammatory subtypes. According to the idea of molecular phenotyping, molecular pathways have been linked to clinical and physiological characteristics of the disease.[4,5]

### Pathophysiology of Bronchial Asthma:

Some people with asthma experience chronic respiratory impairment. Others experience an intermittent disease with episodic symptoms that can be brought on by a variety of factors, such as exercise, airborne allergens, and upper respiratory infections.[6,7]

An asthma attack is an acute exacerbation of the condition. Shortness of breath and wheezing, rapid breathing, prolonged expiration, a quick heartbeat, rhonchus lung sounds, and overinflation of the chest are the clinical signs. The accessory muscles of breathing are implicated in a severe asthma episode.People with severe asthma episodes may lose consciousness or become blue from a lack of oxygen. They may also have chest pain. Respiratory arrest and mortality may result from severe asthma attacks. Seven different types of stimuli are available: Asthma-inducing substances include aspirin and beta-adrenergic antagonists, common household insect waste, ozone, nitrogen dioxide, sulfur dioxide, industrial compounds, and other chemicals, particularly sulfide. Chlorinated swimming pools also produce chloramines, including monochloramine, dichloramine, and trichloramine, which are known to cause asthma.Infections in the early years of life, particularly respiratory virus infections. Exercise, which has considerably different impacts than the other triggers, is number six.[8,9]




During an asthma attack, inflamed airways react to environmental triggers like smoke, dust, or pollen.[10,11]

Due to the excess mucus that the inflamed airways produce, breathing can be difficult. In addition, asthma is the result of an abdominal immunological reaction to a bronchial immune reaction. The airways become hypersensitive to specific triggers, also known as stimuli. Asthma attacks are brought on by these factors narrowing the bronchial passages. Soon after, there is inflammation, which narrows the airways even more and causes an abundance of mucus to be produced, leading to coughing and other breathing issues.[12,13]

It is preferable to understand the mechanisms underlying allergic asthma, or asthma brought on by an immunological reaction to inhaled allergens, from the causes of asthma. Both asthmatic and non-asthmatic individuals who are exposed to allergens inhale them through the inner airway channel. Antigen-presenting cells then present the allergen to other immune system cells, which cause them to change into TH2 cells. The resulting TH2 cells stimulate the humoral immune system, which will create antibodies against the allergen breathed.[14,15]

In a later stage, when an asthmatic inhales the same allergen, these antibodies recognize it and trigger an inflammatory reaction that leads to inflammation, which releases chemicals that restrict the airways and release more mucus, as well as activating the cell-mediated immune system. The clinical presentation of an asthma episode is brought on by the inflammatory response.[16,17]

## HISTORY OF ANTI-ASTHMATIC DRUGS

Asthma mortality and hospital admissions have significantly decreased as a result of the development of extremely effective medications for its therapy. Thanks to the use of drugs with almost no side effects, the majority of asthma sufferers may today live a normal life.[18,19]

It's interesting to note how many of our excellent asthma treatments have their roots in organic compounds. Numerous substances, including atropine, dietary xanthine compounds like theophylline, and chromones from a Mediterranean medicinal herb, were identified from plants as a result of the development of herbal treatments.[20,21]

## MUSCARINIC RECEPTOR ANTAGONIST:

These treatments were readily available until late in the 20th century but were no longer used after the development of more potent bronchodilators made of adrenaline. The development of quaternary ammonium derivatives, which did not cross the blood-brain barrier and were, therefore, free of the central side effects, such as hallucinations, of naturally occurring atropine-like compounds, was a significant advancement in the use of muscarinic receptor antagonists for asthma. Ipratropium bromide, a synthetic quaternary anti-muscarinic chemical, is still utilized as a bronchodilator in patients with severe asthma even though these quaternary derivatives are not absorbed from the gastrointestinal tract.However, cholinergic bronchoconstriction only makes up a minor portion of the bronchoconstriction in asthma compared to the direct bronchoconstrictor action of other inflammatory mediators in the majority of patients, making it less efficient than a -agonist. But in the case of chronic obstructive

pulmonary disease (COPD), where the main modifiable factor appears to be cholinergic tone in the airways, antimuscarinic drugs have emerged as the preferred bronchodilators.The most recent development is the arrival of Boehringer Ingelheim's long-acting antimuscarinic, tiotropium, which causes bronchodilatation lasting for many days.

has a unique function and distribution, and has been a significant advancement in muscarinic pharmacology. The discovery that M3 receptors mediate the bronchoconstrictor action of cholinergic tone whereas M2 receptors served as feedback inhibitory receptors (autoreceptors) in animal parasympathetic nerves was a significant advancement in the field of lung pharmacology.This was subsequently verified in the airways of humans. The therapeutic implication is that nonselective muscarinic antagonists, such as atropine and ipratropium, stimulate acetylcholine synthesis from cholinergic nerves by inhibiting the M2 auto receptors and may thus overcome the blockage of the M3 receptors on airway smooth muscle cells. [22,23]This gave rise to the hypothesis that M3-selective antagonists would be superior bronchodilators. Tiotropium does, in fact, have a kinetic preference for M3 receptors since it separates from M3 receptors considerably more slowly than it does from M2 receptors. The lengthy duration of action is a more significant benefit, and it has not been conclusively demonstrated that M3 receptor selectivity has any significant clinical advantage.More long-acting muscarinic antagonists are now being developed, and they will be used both alone and in conjunction with long-acting 2-agonists, primarily for patients with COPD but also for those with asthma. Intriguingly, it has recently been discovered that the antimuscarinic drug glycopyrrolate, which has been used for many years by anesthesiologists to dry upper airway secretions, has similar pharmacology to tiotropium, with kinetic selectivity for M3 receptors and a prolonged half-life when administered via inhalation.

## β–ADRENERGIC RECEPTOR AGONIST

Ephedrine, which is derived from the plant Ephedra and is also referred to as Ma Huang in Chinese medicine, has been used to treat respiratory illnesses for more than 5000 years. Thus, the earliest known anti-asthma medication is ephedrine. By releasing endogenous catecholamines, it has an indirect effect that causes bronchodilation. Dale demonstrated its efficacy through inhalation as early as 1910.German chemists first created isoprenaline in the 1940s. It was later shown to have fewer cardiovascular adverse effects than adrenaline, and for about 20 years it was the most used inhaled asthma medication. Because the bronchial response to isoprenaline and noradrenaline differs, Ahlquist was able to discriminate between - and -adrenergic receptors thanks to its production in 1948. It showed that -receptors may be further classified into 1-receptors in the heart and 2-receptors in the airways using the rank order of potency of natural and synthesized sympathomimetic amines.In humans, verified by Lands' discovery that isoetharine is a highly selective agonist at beta-2 adrenoceptors. Due to the catechol ring's quick metabolism, isoetharine's effects were brief, just as those of isoprenaline. The discovery of the first 2-selective agonist with a longer duration of action than isoprenaline by the team at Glaxo led by David Jack represented a significant advance, and Cullum concisely detailed its pharmacology in the British Journal of Pharmacology (later Alabaster). Salbutamol is still the most often prescribed asthma medication in the world today.Salmeterol, the first long-acting 2-agonist with a bronchodilator action of over 12 hours, was discovered by Brittain and Jack as a result of the next logical development, which was to increase the duration of action of salbutamol by substitution in the side chain. In 1990, inhaled salbutamol, which had been shown to have an extended duration of effect in asthma patients, was introduced into clinical use. Salbutamol, another long-acting 2-agonist, was first utilized as tablets in Japan, where there was no

indication of a lengthy duration of action. This wasn't known until after inhaled salbutamol was administered to asthmatic patients and found to have a comparable half-life to salbutamol.[24,25]

## CORTICOSTEROIDS

It has been demonstrated that corticosteroids are the most effective asthma controllers, and it will be incredibly challenging to develop any other medication that even comes close to offering an equal therapeutic advantage. Given that adrenaline would be significantly metabolized upon absorption from the gastrointestinal system, it seems likely that the health advantages of the orally taken adrenal extract recorded by Solis-Cohen in 1900 were in fact brought on by the steroid content rather than any adrenaline present. This instantly suggested the necessity for inhaling corticosteroids to reduce systemic adverse effects, although doing so turned out to be of little use for cortisone and dexamethasone. Interestingly, Brown noted that the patients who fared the best had high eosinophil counts in their sputum, a finding that has been supported by a large number of later research. The main factor contributing to the recent decline in asthma mortality and morbidity is the widespread use of inhaled corticosteroids in treating asthma. With the development of budesonide, fluticasone propionate, and most recently ciclesonide, a prodrug that is activated by esterases in the lower airways, there is currently a quest for inhaled corticosteroids with superior therapeutic ratios and less systemic side effects.The molecular processes underlying the anti-inflammatory actions of corticosteroids in asthma have lately undergone a significant improvement, with a focus on how corticosteroids affect chromatin remodelling by increasing the recruitment of histone deacetylase-2 to activated inflammatory genes. Future research may lead to the development of selective glucocorticoid receptor agonists or dissociated steroids that have enhanced anti-inflammatory effects by suppressing activated inflammatory genes and reducing the binding of glucocorticoid receptors to DNA, which is thought to be a key mediator of side effects (see also Buckingham, this issue). It's interesting to note that the topically active corticosteroids now found in asthma inhalers already exhibit some degree of dissociation.[26,27]

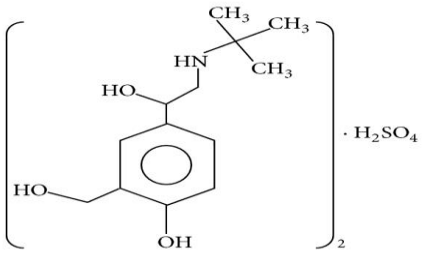
## MECHANISM OF ACTION

When compared to isoproterenol, salbutamol has a preferential action on beta2-adrenergic receptors, according to in vitro and in vivo pharmacologic investigations. Beta2-adrenoceptors in the human heart make up 10% to 50% of all beta-adrenoceptors, despite beta1 adrenoceptors being the major receptors in the heart and beta2 adrenoceptors being the predominant receptors in bronchial smooth muscle. Although the precise role of these receptors is unknown, their presence suggests that even selective beta2-agonists could have cardiac effects.

Adenyl cyclase is activated by the activation of beta2-adrenergic receptors on airway smooth muscle, and the intracellular concentration of cyclic-3′,5′-adenosine monophosphate rises as a result (cyclic AMP). As a result of this rise in cyclic AMP, protein kinase A is activated, which prevents myosin from being phosphorylated and decreases intracellular calcium concentrations, causing the muscle to relax. All airways, from the trachea to the terminal bronchioles, have smooth muscles that can be relaxed by salbutamol. Regardless of the spasmogenic agent involved, salbutamol functions as a functional antagonist to relax the airway, defending against all bronchoconstrictor assaults. Increased levels of cyclic AMP are also linked to a reduction in the release of mediators from mast cells into the airway.

In the majority of controlled clinical trials, salbutamol was found to relax bronchial smooth muscles more than isoproterenol did at equivalent doses, while having less adverse cardiovascular effects. Inhaled salbutamol, like other beta-adrenergic agonist medications, has been demonstrated in controlled clinical research and other clinical experience to

induce a significant cardiovascular effect in some patients as determined by pulse rate, blood pressure, symptoms, and/or electrocardiographic changes.[28]



## ADVERSE DRUG REACTIONS:

It's possible to have anxiety, vertigo, shaking (tremor), headache, nausea, mouth/throat dryness or irritation, or strange tastes. Inform your doctor or pharmacist as soon as possible if any of these side effects persist or get worse. Keep in mind that your doctor has recommended this medication because they believe it will benefit you more than it will harm you. Many users of this medicine report no significant negative effects.If you have any really significant side effects, including as chest pain, an irregular heartbeat, rapid breathing, or confusion, seek medical attention right once.

## Precautions

Before taking salbuterol, let your doctor or pharmacist know if you have any allergies to it, any related medications, or any other conditions. To learn more, speak with your pharmacist. (such salmeterol, levalbuterol, or metaproterenol); or if you experience any additional allergies. This product may include inactive components that can cause allergic responses, like lactose and milk proteins.Tell your doctor or pharmacist about all of your medical conditions before using this drug, especially any that may affect your heart (such as an irregular heartbeat, angina, or a history of heart attacks), high blood pressure, or seizures.You can feel lightheaded after using this medication. You may feel more lightheaded after consuming alcohol or cannabis. Till you can do it safely, avoid operating machinery, driving, or doing anything else that requires alertness. Limit your alcohol consumption.

## Interactions

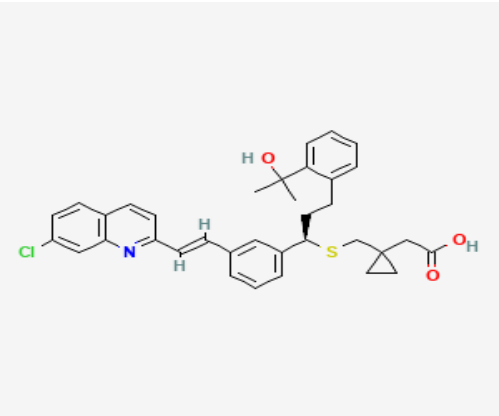
Drug interactions could alter how your medications function or raise the possibility of major negative side effects.Not all medication interactions are included in this document. Keep a list of everything you use, including herbal products, prescription, and over-the-counter medications, and provide it to your doctor and pharmacist. Without your doctor's approval, never start, stop, or change the dosage of any medications.When using salbutamol, avoid taking drugs that include levalbuterol.[29]

Uses

1. Wheezing and shortness of breath brought on by breathing difficulties are prevented and treated with salbutamol (such as asthma, and chronic obstructive pulmonary disease).
2. It is also used to stop exercise-induced asthma. It is a drug for fast relief. Salbutamol is a member of the bronchodilator drug class

Mechanism of Action

When such CysLT bind to associated CysLT receptors, such as CysLT type-1 receptors found on respiratory airway smooth muscle cells, airway macrophages, and on various pro-inflammatory cells like eosinophils and some particular myeloid stem cells, activities that facilitate the pathophysiology of airway bronchoconstriction, occluding mucous secretion, vascular permeability, and eosinophil recruitment are all types of



As opposed to this, in allergic rhinitis, CysLTs are released by the nasal mucosa when exposed to

allergens during both early and late phase reactions and help to cause symptoms of allergic rhinitis such a stuffy nose and congested airways.

As a result, montelukast is a leukotriene receptor antagonist that binds to the CysLT type 1 receptor with high affinity and selectivity. This helps to block any physiological activities of CysLTs like LTC4, LTD4, and LTE4 at the receptor that might support asthma.

Interaction

- Drug interactions could alter how your medications function or raise the possibility of major negative side effects. All probable medication interactions are not included in this document.
- Keep a list of everything you use, including prescription and over-the-counter medications as well as herbal remedies, and provide it to your doctor and pharmacist.
- Without your doctor's approval, never start, stop, or change the dosage of any medications.

Precautions

- Tell your doctor or pharmacist if you are allergic to montelukast or if you have any other allergies before taking the medication. Inactive chemicals in this product have the potential to trigger allergic reactions or other issues. To learn more, speak with your pharmacist.
- Inform your doctor or pharmacist about your medical history before using this medication, especially if you have liver disease or mental/mood issues (such as anxiety, depression, or thoughts of suicide).
- Aspartame could be present in chewable tablets. Ask your doctor or pharmacist about using this medication safely if you have phenylketonuria (PKU) or any other condition that requires you to limit or avoid aspartame (or phenylalanine) in your diet.

Uses:

- Asthma symptoms are managed and prevented with the use of the drug montelukast (such as wheezing and shortness of breath). It is also taken prior to exercise to avoid respiratory issues when exercising (bronchospasm).
- The frequency with which you need to use your rapid relief inhaler may be reduced with the aid of this medicine. Sneezing, stuffy/runny/itchy nose, and other allergic rhinitis symptoms can be treated with montelukast.
- This medicine should only be used for this condition when other allergy medications cannot be used or do not work effectively, as there are other allergy medications that may be safer (see also the Warning section).
- To be successful, this drug needs to be used on a daily basis. Since it takes time to start working, it shouldn't be used to treat sudden asthma attacks or breathing issues. Use your quick-relief inhaler as directed if you experience an asthma attack or abrupt shortness of breath.
- Leukotrienes, which may cause or aggravate allergies and asthma, are blocked by this medication. By lowering swelling (inflammation) in the airways, it facilitates breathing.[30]

## CONCLUSION

A significant, multicenter study found that over the course of eight years, a greater percentage of participants reported using asthma drugs. Symptomatic asthma has increased in the study population at the same time. In order to maintain and regulate asthma symptoms, controller drugs are crucial. To treat acute asthma symptoms, all patients require a "quick-relief" 2 adrenergic agonist drug as well as leukotriene modifiers. The goal of drug therapy for long-term asthma control is to reduce and eventually stop airway inflammation. After conducting this market research, we have come to the conclusion that salbutamol is not as widely utilized as

montelukast, a leukotriene modulator. Montelukast has shown to be very helpful in treating asthma brought on by exercise and asthma linked with allergic rhinitis. Asthma in obese people, asthma in smokers, aspirin-induced asthma, and virally produced wheezing episodes are other phenotypes where montelukast is helpful.

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