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Polyherbal Tea Bag: - Antitussive And Bronchodilators

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Abstract: Bronchial asthma is quite simply a chronic inflammatory disease of the airways with management primarily relying on anti-inflammatory and bronchodilator agents. Herbal tea is an essentially herbal mixture of all natural ingredients. Indian native herb that is used for its Antitussive and Bronchodilator properties; which will helps people living with asthma, cold cough, digestive related problems. The purpose of this study is to formulate and evaluate polyherbal tea of a therapeutic effect from available natural products. The tea combines liquorice roots, lemon peels, lemongrass, turmeric, cinnamon bark, cardamom seeds, cumin seeds, fennel fruit, and Gokarna flowers, all processed into fine powders and packaged in tea bags. Rich in phytochemicals such as flavonoids, saponins, alkaloids, and glycosides, the tea is designed to offer antitussive and bronchodilator properties, potentially alleviating cough and respiratory congestion. Active compounds like alkaloids, flavonoids, and tannins contribute to its antioxidant, antibacterial and anti-inflammatory effects. The main objectives of the study include the evaluation of efficacy, safety and the acceptance of tea by the consumer. Evaluation methods encompass physicochemical tests (colour, pH, odour and taste), phytochemical analysis, LOD, antimicrobial testing against E. coli using the cup plate method, and quality assessment through ash value and acid insoluble ash tests. The synergistic effects of the herbal ingredients are mentioned in this research to establish the polyherbal tea as a natural remedy of the respiratory ailment, which is supported by scientific evaluation of the herbal properties.

Keywords- Bronchodilators, Ayurvedic medicine, Anti-inflammatory drugs, Antitussive, Polyherbal tea, Phytotherapy.

A. Introduction

Respiratory disorders including asthma, chronic obstructive pulmonary disease (COPD), cough etc. are influenced by various factors including environmental pollution, smoking, occupational hazards etc. As herbal tea is prepared from different natural ingredients which have multiple properties to cure diseases as well as disorders those are according to ayurveda system. It is a part of traditional ayurvedic medicine that uses polyherbal remedies for respiratory ailments from centuries. Based on the principle of synergistic action, polyherbal formulations, which consist of multiple herbs, are believed to increase therapeutic effects. This polyherbal tea formulating has an effective antitussive (cough suppressant) and bronchodilator (airway dilator). Our aim is to provide an effective prevention and management strategies, to develop an effective, safe & affordable polyherbal antitussive, bronchodilator tea that provides relief from cough & respiratory discomfort & to formulate & evaluate a ployherbal tea that reduce the frequency & severity of cough.

- Efficacy in Cough Relief.
- Bronchodilation Enhancement.
- Anti-inflammatory Properties.
- Mucus Clearance.

- Immune Support.
- Safety and Tolerability.
- Easy Affordability.

1. **Bronchial Asthma: -**

Hyper responsiveness of tracheobronchial smooth muscle to a variety of stimuli is a major feature of bronchial asthma, accompanied by narrowing of air tubes, often with increased secretion, mucosal edema and mucus plugging. Symptoms include dyspnoea, wheezing, cough etc, respectively [1].Therefore, this is a complex, chronic, inflammatory, respiratory disease of the airways characterized by airway hyper-responsiveness, airflow obstruction, and airway remodelling, & that is regulated by cellular and humoral factors [2]. The bronchial asthma pharmacotherapy includes anti-inflammatory agents (corticosteroids) and bronchodilators (β2-agonists) [3]. Worldwide, around 300 million people have asthma and in the U.S., more than 22 million, according to recent estimates [4]. The disease often starts in childhood and affects 6 million children in the US, though people of all ages can be afflicted. Around 255,000 people die from asthma every year worldwide [5]. The following changes occurred during asthma attack.

- The airways are narrowed by tightening the muscles around them.
- The airway is not able to pass as much air.
- As the airway narrows further, this causes more inflammation in the airways.
- Even more mucus is produced in the airways, further hampering the flow of air.
- Lower air is suitable to flow through the airway.
- Inflammation of the airways increases, further narrowing the airway.
- More mucus produced in airways, reducing the flow of air even more [6].

Asthma causes ordinary periods of wheezing (a whistling sound when breathe), chest tightness, shortness of breath, and coughing. The coughing frequently happens at night or early inside the morning [7].

1.1.Sign and Symptoms of Asthma [1, 8].

The most common indicators of asthma include: –

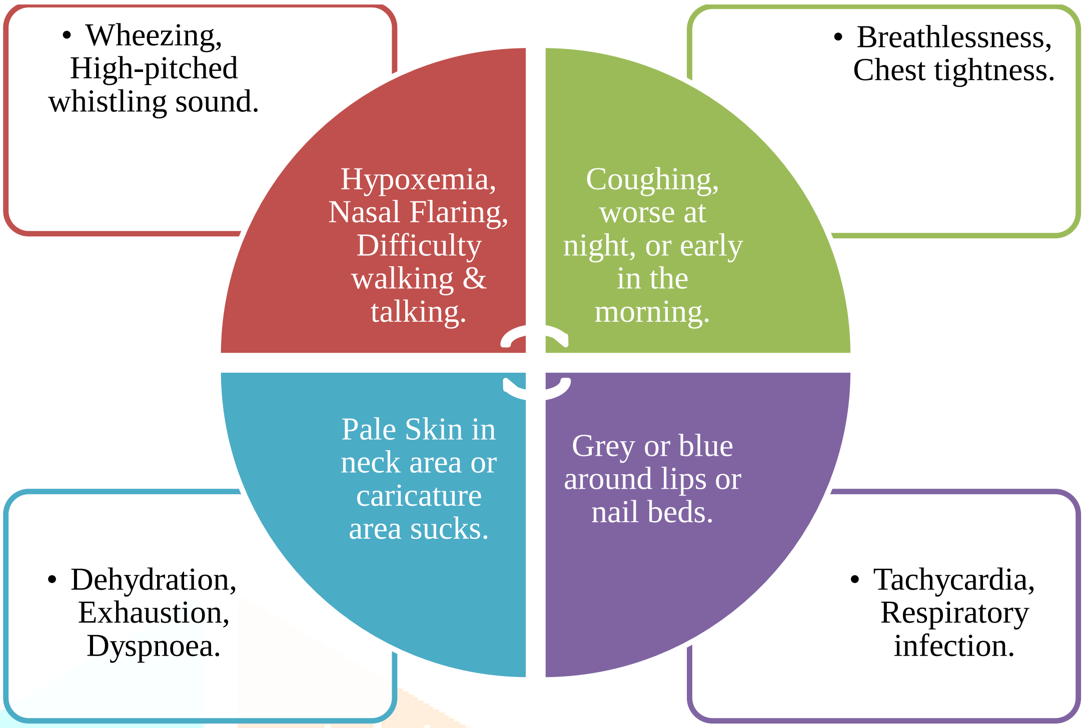


Chart 01: Sign & Symptoms of Asthma

1.2. Causes and Reason of Asthma

Currently it is thought that the development of asthma is due to genetic and environmental risk factors working together. In fact, the disease has been seen to have a few associated genes and family members of asthmatic individuals are more likely to develop this condition. Indoors and outdoors allergens (dust mites, pollen, and mould spores) and pollutants (such as cigarette smoke, chemicals or fumes) and respiratory viruses (such as influenza and common cold) may be other common triggers of asthma attacks. There is no one cause of asthma, but it is likely a genetic predisposition, intensified by environmental triggers. Allergens (pollen, mould spores, animal dander and dust mites) and non-biological substances (such as smoke, fumes and even certain chemicals) involved in acute viral illnesses the flu or viral upper respiratory infections contribute to the aggravation of asthma conditions [9].

Asthma occurs due to environmental and inheritable commerce.

- These relations impact both inflexibility and responsiveness to treatment of asthma.
- It changed into installation that the latest increased quotes of allergies are because of inheritable factors apart from the ones associated with the DNA collection & a changing living environmental.
- The occlusion of asthma before age 12 is due to inheritable influence, while onset after 12 is more likely due to environmental changes.
- Asthma is not as a result of any individual element, however by way of a selection of factors interacting with one another. One person's asthmatic factors may be fully different than another person's.
- Atopy is the inherited predilection to develop an antibody called immunoglobulin E (IgE) in response to exposure to environmental allergens.

- Asthma attacks are caused by airway hyperactive responsiveness that's an overreaction of the bronchi and colourful environmental and physiological stimulants, known as triggers.
- Several environmental factors have been associated with the development of asthma surroundings are allergens, air pollution, chemicals set up in [9-11].
- Common trouble factors responsible for both development and deterioration of the lungs include smoking, obesity & low physical exertion, pollutants, infection, exposure to allergens, malnutrition, metabolic pattern, conditions, depression & use of certain drugs.

1.2.1. Cold Air –

When the airways narrow, this is called asthma and the dry cold air congests lung tissue. In patients with asthma, airway inflammation or pre-existence irritation essentially makes this phenomenon common place. It is also noted that cold air is usually dry and will stimulate mucus production, further narrowing the airway and lead to shortness of breath, coughing and sneezing. The risk for colds increases in colder temperatures [cold as a trigger] [12]. More so: colds & other upper respiratory infections are more common during winter months that can lead to excess coughing, which worsens asthma symptoms. Exercise in the cold especially if not warmed up properly may trigger nausea & worsen symptoms. The air before it is breathed should be warm, the nose and mouth covered with a mask as well as not exercising in cold dry conditions to prevent asthma attacks [13].

1.2.2. Infection –

Infections have been identified as contributing factors in the development of psoriasis diseases, as well as lung diseases especially, asthma. As the lower respiratory tract microbiome is diverse, this helped to establish a link between asthma and the immune system and activate the interactions between these risk factors, which can lead to psoriasis HIV infection that the risk of not only psoriasis but also humoral immune diseases affected lungs can be, such as asthma, COPD, PVH and HIV-infected people can be increased as psoriasis progresses or leads to pneumonia [14-18]. In viral infections of rhinoviruses, respiratory syncytial viruses, and influenza, most especially in children, most common causes of asthma deterioration are these viruses [19]. These viruses affect the airways, causing inflammation, hyper-responsiveness, and wheezing; thus, involving antigen and asthmatic symptoms [20].

- Dysregulation of immune response: On the other hand, viral infections can cause negative immune response development for instance overriding immune response to Th2 dominant response which is associated with allergies and is often seen in asthma. This collapse of the immune equilibrium fuels further aggravation of the lung tissue inflammation hence contributing to the symptoms of asthma [21].
- Bacterial involvement: Often asthma is also inflamed by the presence of atypical bacteria like Chlamydia pneumoniae and Mycoplasma pneumoniae, which ease airway inflammation and instigate asthma symptoms. Their contribution to the condition is not as great as that of viruses but is nonetheless present [22].
- Children Infections: An earlier age when infections happen, especially during the exposure to viruses such as RSV, has a strong correlation with asthma symptoms being are found in later growth phases. Children with a history of severe infection with RSV are more likely to develop asthma that is persistent [23].

1.2.3. Irritants –

- **Irritant-Induced Asthma (RADS)**: Reactive airways dysfunction syndrome (RADS) develops after acute, high-level exposure to irritants like chemicals, vapours, or smoke. This exposure causes airway inflammation, leading to chronic hyper responsiveness and asthma-like symptoms [24].
- **Chronic Irritant Exposure**: Repeated or prolonged exposure to moderate levels of irritants, such as dust or fumes, can gradually lead to chronic airway inflammation, contributing to asthma development. This is especially common in occupational settings [25].
- **Immune Response and Inflammation**: Irritants like smoke or pollutants can increase airway inflammation and worsen asthma by triggering an exaggerated immune response, leading to airway swelling and mucus production [26].

1.2.4. Stress / Depression –

Stress is associated with severe asthma symptoms, partly due to behaviours such as smoking, but also through a direct effect on the immune system, which increases inflammation Psoriasis, a skin condition chronic inflammation, causes severe social and functional restriction, and leads to stress [27]. It adversely affects work, family life, social opportunities and sexual health, creating a physical and psychological challenge for patients. Depression is common in those with interstitial lung disease (ILD), a condition characterized by progressive pulmonary vascular lesions. Chronic ILD, as well as physical deficits and reduced quality of life, often contribute to depression in these patients [28].

1.2.5. Dust / Dust Mites –

Dust mites, in particular Dermatophagoides pteronyssinus, are a well-known cause for allergic allergies. These microscopic insects thrive in environments like bedding and carpets, where they launch allergens that aggravate the airways [29]. Exposure to dirt mite allergens, inclusive of Der p1, ends in airway irritation, accelerated lung sensitivity, and allergies exacerbations. Over time, this can make a contribution to pulmonary hypertrophy, further worsening allergies symptoms and lung feature. Dust mites are a sizable environmental component in allergies management [30].

1.2.6. Smoking –

Cigarette smoke aggravates bronchial asthma through constricting and inflaming the airlines [7]. Recent research show that individuals with psoriasis are a lot more likely to be lively and passive smokers, while even smoking might be related with extra extreme psoriatic lesions [17]. It is an important driver of COPD and other lung conditions, including asthma and idiopathic pulmonary fibrosis [28]. Cigarette smoke may even enhance allergies risks, aggravates signs and symptoms and additionally increases the threat of asthma-induced mortality [31-33]. Exposure to second hand smoke, especially in the case of children and adolescents also increases allergies risk significantly [27].

1.2.7. Psychogenic / Genes –

The development of asthma is heavily influenced by genetics, involving more than 100 genes, many of which affect immune function and inflammation. Approximately 60% of the population develops asthma, and having a parent with asthma increases the risk 3 to 6 times. Genetic and environmental interactions are also important, as is the CD14 gene variant (SNP C-159T) is recognized, which increases the risk of asthma through exposure to environmental toxins This highlights a complex interaction between genetic predispositions and factors between environments, highlighting the need for further research [27].

1.2.8. Airway Constriction/ Pollution –

Asthma signs and symptoms are significantly affected by environmental pollutants and weather conditions, which could exacerbate the underlying inflammatory responses within the lungs [8]. Key triggers include pollution like smoke, fumes, Sulphur dioxide, nitrogen oxide, & ozone [7]. For instance, smoke and chemical fumes can cause "bronchoconstriction “a tightening of the muscle tissue across the airlines, which restricts airflow, main to a sensation of chest tightness and problem respiration [34-35]. Cadmium, a heavy metal occurring in some batteries and industrial processes, is associated with respiratory diseases and elevated levels occur in non-smoker psoriasis and COPD patients [28, 36]. Cadmium, a pollutant that creates an inflammatory response in the lungs and skin [11]. Pollution and weather connected with environment triggers such as gas-cooked food, cool air, very humid improves asthma [27].

1.2.9. Allergies/ Inflammation –

Asthma is an inflammation of the airways, causing inflammation, excessive mucus, weak bronchial mucosa that prevents air from entering the lungs and causes symptoms such as wheezing, coughing, tightness of the chest, shortness of breath occurs with difficulty etc. [7, 34-35]. Epilepsy is one of the most widespread allergic diseases, including dust mites, pet scabies [27-28]. Abnormal response to intestinal stimuli to causes airway inflammation and irritation because these allergens can cause asthma and other potentially inflammatory diseases, such as psoriasis, suggesting an immune-related asthma interaction between the immune system and various allergic diseases [37-40].

1.2.10. Obesity & Depression –

Chronic conditions such as psoriasis and interstitial pneumonia are often associated with mental health issues, especially depression, reflecting the link between physical and mental health [27-28]. Stress encourages harmful behaviours such as smoking and exacerbates asthma by directly affecting the immune system, which can increase sensitivity to allergies and, depending on severity, build complex interactions emphasizing the interplay between stress, inflammation and disease progression [38-39].

1.3. Types of Asthma

- Allergic asthma & Non allergic asthma [1].
- Asthma in pregnancy [1].
- Occupational asthma [7].
- Child-onset asthma & Adult-onset asthma [7].
- Exercise induced asthma [27].
- Cough induced asthma [27].
- Nocturnal asthma [27].
- Steroid resistant asthma [42].
- Intrinsic asthma & Extrinsic asthma [42].
- Aspirin induced asthma [43].

2. COPD [Chronic Obstructive Pulmonary Disease]

- Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory condition characterized by chronic airflow limitation and inflammation often due to long-term smoking or exposure to pollutants.
- COPD is a condition in which air sac becomes narrow & cause difficulty in breathing & is not reversible & causes constriction, swelling, bronchitis of alveoli sac (air sac).
- COPD is slowly progressive diseases which are characterised by using a slow loss of lung feature, tissue destruction, also includes continual bronchitis, persistent obstructive bronchitis, or emphysema, or combination of these conditions. The main causes of COPD are air flow resistance, chronic bronchitis, Bronchiolitis, Emphysema.
- COPD is a common place and treatable disease and is related to structural lung changes because of continual inflammation from prolonged exposure to noxious debris or gases maximum usually cigarette smoke. Chronic inflammation reasons airway narrowing and reduced lung cringe. The

ailment regularly affords with signs and symptoms of cough, dyspnoea, and sputum manufacturing. Symptoms can range from being asymptomatic to respiratory failure [44].

- The disorder causes persistent expiratory airflow limitation, which stays extraordinarily strong through the years, although exacerbations can occur due to respiration infections, pollutants, and different elements.
- COPD is strongly related to smoking and generally develops after age 40. Quitting smoking can gradual the decline in lung function.
- There is minimum improvement (<15%) in forced expiratory quantity (FEV1) after the usage of a β-agonist bronchodilator, indicating that the airway obstruction is basically irreversible [11, 45].
- Most sufferers with COPD are middle-aged or aged. In 2000, 16 million office visits had been attributed to COPD-associated situations, with the caseload expected to growth with the ageing of the population [46-46].
- To raise awareness of COPD, the Global Program had on COPD (GOLD) began in 1997, as a state-owned enterprise Heart, Lung, and Blood Institute, National Institutes of health, and WHO to broadcast information on causes of COPD and providing implementation instructions [46]. The prevalence of COPD, characterized by using an irreversible issue of expiratory airflow, is developing within the United States and global, and no cure is available. Smoking is the main reason for this ailment, as a consequence smoking cessation in smokers is crucial. Regular use of inhaled bronchodilators to save you and relieve symptoms is the mainstay of control. Short-performing inhalers provide instantaneous symptom relief, but long-appearing inhaled bronchodilators are more effective and offer more comfort; for this reason, combining inhalers is frequently advocated [47].

2.1. Sign and Symptoms of COPD [48-49].

The most common indicators of COPD include: –

- Weight loss, Back pain.
- Impaired nutrition, Sputum production.
- Skeleton muscles dysfunction.
- Breathlessness & Frequent lung infections.
- Chronic cough, the mucus may be clear, white, yellow or greenish.
- Wheezing & Chest tightness.
- Anxiety & Fatigue.
- Reduced exercise capacity.
- Sleep disturbances & Lack of energy.
- Depression, and cognitive issues.
- Swelling in ankles, feet or legs.

2.2. Causes and Reason of COPD

Smoking tobacco, chemical fumes, vapours, and dust at work for a long time. It is the main reason people get COPD in developed countries. In most developing nations, COPD often shows up in people who breathe in smoke from burning fuel used to cook and heat ventilated homes [49].

Most common bacterial and viral pathogens isolated from patients with COPD exacerbations are [50]: -

Sr. No.	Bacteria	Viruses
1.	Haemophilus influenza	Rhinovirus
2.	Moraxella catarrhalis	Coronavirus
3.	Streptococcus pneumoniae	Influenza
4.	Pseudomonas aeruginosa	Parainfluenza
5.	-	Adenovirus
6.	-	Respiratory syncytial virus

Table 01: Bacterial and viral pathogens isolated from patients with COPD exacerbations

Common Trouble factors responsible for COPD are as follows: -

2.2.1. Tobacco, Cigarette Smoking

Cigarette smoking is an exquisite threat factor for chronic obstructive pulmonary sickness (COPD), especially in advanced nations, wherein lengthy-term exposure to risky chemicals damages lung tissue. The threat will increase with cigarettes and pipes of prolonged and intense smoking. Smoking additionally contributes to COPD in smokers and non-individuals who smoke [49]. In developing worldwide places, vast in rural areas together with India and Kashmir, indoor air pollution from wooden and gasoline combustion drastically will growth COPD danger with tobacco smoke chemicals extra than 4000, more than 60 of which can be carcinogenic, and nicotine is pretty addictive. Carbon monoxide in tobacco smoke reduces oxygen transport and locations pressure at the coronary heart [51]. The health risks of smoking, consisting of COPD and lung most cancers, became obvious within the mid-20th century [52].

2.2.2. Asthma

Asthma is a situation in which the airlines slim and swell and may produce extra mucus. Asthma may be a hazard element for growing COPD. The blend of asthma and smoking raises the chance of COPD even greater [49].

2.2.3. Workplace & Occupational Exposure

In developing countries, exposure to fumes from burning fuels for cooking and heating in poorly ventilated houses is an extensive risk component for COPD. Occupational exposures additionally make a contribution heavily to COPD risk. Prolonged publicity to chemical fumes, vapours, dirt, and smoke common in positive industries which include mining, brick production, pottery, & cement work can inflame and irritate lung tissue, main to COPD [49]. Specifically, industries involving crystalline silica, together with gold mining and ceramics, pose high dangers because the inhaled debris cause persistent lung infection and damage through the years [53]. Together, these environmental and occupational elements exacerbate breathing troubles, increasing COPD prevalence amongst uncovered individuals [51].

2.2.4. Genetics/ Heredity

Alpha-1 antitrypsin (AAT) deficiency, an autosomal recessive genetic sickness due to mutations inside the SERPINA1 gene, is an awesome genetic motive of COPD, especially the emphysema form. AAT, produced in the liver, usually protects lung tissue from harm by means of inhibiting proteolytic enzymes, however AAT deficiency ends in unregulated enzyme hobby, resulting in lung damage and elevated COPD risk [49]. The situation is surprisingly rare, with only 1-2% of the populace affected, primarily the ones carrying the Z allele variation of SERPINA1. Although smoking is a giant COPD danger component, a few human beings with AAT deficiency increase COPD regardless of a lack of smoking or environmental exposure, frequently offering symptoms after age 40 [53]. Research shows that COPD probably includes more than one genetic elements beyond AAT deficiency, as simplest a subset of people who smoke expand the ailment, hinting at complicated interactions between diverse gene polymorphisms that make contributions to COPD susceptibility [51]. Louis included the information of James Jackson, a postgraduate student in Paris (France) from Boston (Massachusetts, USA), who accrued facts on 41 instances in 10 months. Jackson made a strong case for heredity in the ailment: 18 of his 29 sufferers with emphysema have been offspring of dad and mom who had the sickness, while 50 healthful humans, most effective three had mother and father with emphysema; a precursor of Peter Paré's Christie lecture on susceptibility of genes [52].

2.2.5. Viral Infections

COPD is often caused by viral infections, especially in the winter when respiratory viruses such as rhinovirus, coronavirus, influenza, RSV, etc. are common. These viral infections cause infection more severe, longer recovery time and more frequent hospitalizations [50]. The virus is found in approximately half of human outbreaks, although the incidence of the virus varies from place to place. For example, 22% and 64% of cases in Hong Kong and Singapore, respectively, show the presence of the virus. PCR technology has increased detection rates, suggesting that rhinovirus can enter the lower airways, exacerbating inflammation. Even though influenza vaccination mitigates its effects, respiratory viruses remain a major cause, underscoring the need for further research in the management of COPD [54-65].

2.2.6. Infection

COPD is thought to be caused by recurrent respiratory infections from childhood, leading to progressive deterioration of lung function and eventual manifestation of COPD in adulthood. Increased risk of developing chest infections in individuals with COPD due to decreased ability to clear lung mucus, viral infections account for about 50-75% of these cases and COPD are typical examples. Furthermore, it appears that individuals with pneumonia severe illness in childhood are more likely to develop COPD later in life [51]. Evaluating the specific role of bacteria in exacerbations of COPD has been difficult, as common bacteria such as Haemophilus influenza, Streptococcus pneumoniae, Moraxella catarrhalis, Staphylococcus aureus, and Pseudomonas aeruginosa were found in patients with exacerbations of COPD of the 2% in one study. The presence of atoms is shown, which increased to 69% during the prevalence, indicating a remarkable increase in bacterial count [50].

2.2.7. Pollution

In urban settings, developing air pollutants exacerbated with the aid of deforestation and industrialization poses great risks to lung fitness. While air pollution alone won't without delay reason COPD, it increases the probability of the ailment in individuals who have different predisposing factors [51]. Air pollution contributes to multiplied exacerbations and health facility admissions for COPD sufferers, with research like the APHEA examine across European cities, as well as research from Taiwan, Brazil, and Hong Kong, displaying an immediate dating among pollution ranges and hospitalizations [50, 60-69]. Common pollution, along with nitrogen oxides and particulates, may engage with viral infections to exacerbate COPD, especially for the duration of iciness months. Additionally, long-term publicity to air pollutants and dust is linked to COPD development [70]. In conclusion, enhancing air first-rate may want to assist lessen the frequency of COPD exacerbations and hospitalizations [46].

2.2.8. Emphysema

It is a disorder circumstance in which the destruction of alveolar walls takes place with the expansion of airspaces distal to terminal non-respiratory bronchioles. Thus, it ends in the crumble of the lungs whilst a patient exhales and impairs airflow while expiration [51].

2.2.9. Age

COPD develops slowly over years, so maximum human beings are as a minimum forty years antique when signs and symptoms start [51]. Recurrent respiration infections in adolescence have additionally been proven to be related to development of COPD in grownup age [53]. The working guy of middle age in manchester is bronchitic" "the solid particles constantly floating in the surroundings from our manufacturing unit chimneys, and different assets, preserve up a steady infection within the air tubes, generating in the long run persistent bronchitis and emphysema of the lungs [52]. In the year 2000, for the first time there were extra deaths from COPD in women than men. Between 1980 and 2004, the age-adjusted mortality elevated by 162% in Caucasian girls (from 14.2 to 37.2 deaths/100 populace) and 187% in African-American women (from 6.2 to 17.8 deaths/100 populace) [71].

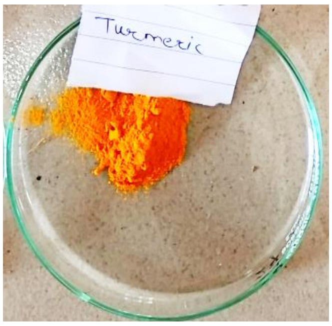
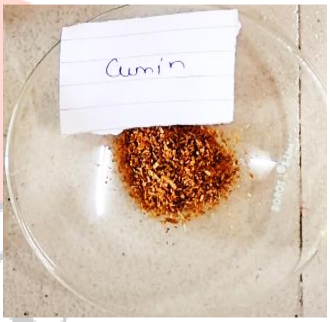
B. MATERIALS AND METHOD

1. MATERIALS

Liquorice, Fennel, Lemongrass, Cinnamon, Cumin, Gokarna Flower, Lemon peels, Turmeric, Cardamom were handpicked from Ayurvedic store.
Empty tea bags were purchased from FEORA 9X online trading company.

1.1 INGREDIENTS OF POLYHERBAL TEA BAG

DRUGS	USES	BIOLOGICAL SOURCE	FAMILY	IMAGES
Liquorice	Digestive Aid, Anti-Inflammatory, Antiviral, Antibacterial, Respiratory Relief, Adrenal Support, Anti-Cancer.	Dried roots and rhizomes of Glycyrrhiza glabra.	Fabaceae (Leguminosae).	 **Figure 1: Liquorice powder**
Lemon Grass	Anti-Inflammatory, Antioxidant, Antimicrobial, Anxiety, Stress Relief, Immune-System Support, Anti-Diabetic.	Leaves of Cymbopogon citratus.	Poaceae (Gramineae).	 **Figure 2: Lemon Grass powder**

Lemon Peels	Boosts Immune System, Antioxidant, Anti-Inflammatory, Weight Loss, Cold & Flu Relief, Cardiovascular health, Anti-Aging, Detoxification.	Peels of fruit citrus lemon.	Rutaceae.	 **Figure 3: Lemon Grass powder**
Turmeric	Anti-Inflammatory, Antioxidant, Improves Heart Health, Brain Health, Anti-Cancer, Digestive Aid, Immune System Support, Menstrual Relief, and Eye Health.	Rhizomes of Curcuma Cyminum.	Zingiberaceae.	 **Figure 4: Turmeric powder**
Cumin	Respiratory issues, Anti-inflammatory, Antimicrobial, Carminative, Digestive aid, Immunomodulatory, Anti-diabetic.	Dried seeds of Cuminum cyminum.	Apiaceae (Umbelliferae).	 **Figure 5: Cumin powder**
Cinnamon	Digestive aid, Anti-diabetic, Cardio protective, Anti-cancer properties, Neuroprotective.	Bark of Cinnamomum verum or Cinnamomum cassia.	Lauraceae.	 **Figure 6: Cinnamon powder**

Cardamom	Carminative, Antimicrobial, Anti-inflammatory, Antioxidant, Expectant.	Seeds of Elettaria cardamomum.	Zingiberaceae.	 **Figure 7: Cardamom powder**
Fennel	Carminative, Antimicrobial, Anti-inflammatory, Antioxidant, Estrogenic, Respiratory relief, Cardiovascular health.	Dried seeds of Foeniculum vulgare.	Apiaceae (Umbelliferae).	 **Figure 8: Fennel powder**
Gokarna Flower	Antimicrobial, Antipyretic, Anti-inflammatory, Analgesic, Diuretic, Local anaesthetic, Antidiabetic, Insecticidal, Blood platelet aggregation-Inhibiting.	Fruits of Tribulus terrestris, Clitoria ternatea.	Fabaceae.	 **Figure 9: Gokarna Flower powder**

Table 02: Ingredients of polyherbal tea bag

2. Methods

2.1 Methods Steps

It involves following steps: -

- Conversion of all crude drugs into Powder.
- Drugs weighed accurately and mixed completely.
- Filling of mixed drugs in empty tea bag.
- Packing of tea bag.
- Dipping of tea bag into hot water.
- Herbal tea was prepared.
- Physicochemical test performed like odour, taste, pH, and colour.
- Phytochemical test performed by using different sub type methods with different reagents.
- Loss on drying, Ash value were performed using hot air oven, muffle furnace respectively.
- The chromatographic evaluation was done using TLC, Ethyl acetate: acetic acid: CHCL3 in ratio 4: 2: 4 was used as mobile phase.
- Antimicrobial testing was performed using MacConkey agar media.
- All testing reports were recorded.

2.2 METHODOLOGY

2.2.1 Contents of Tea Bag

Sr. No.	INGREDIENTS	QUANTITY TO BE TAKEN (GRAM)
1	Liquorice	0.5 gm
2	Lemon Grass	0.5 gm
3	Lemon Peels	0.2 gm
4	Turmeric	0.2 gm
5	Cumin	0.5 gm
6	Cinnamon	0.5 gm
7	Cardamom	0.4 gm
8	Fennel	0.2 gm
9	Gokarna Flower	0.2 gm
10	Empty tea bag	0.3 gm
	Total Weight	**3.5 gm**

Table 02: Composition of tea bag

2.3 Physicochemical Test

The physicochemical test of the herbal tea was performed as shown in Table 4.

2.3.1 Colour –

The herbal tea bag was prepared, the water was kept for boiling and the polyherbal tea bag was then dipped into hot water and after 30 to 45 seconds of dipping the colour was observed to be light green-brown.

2.3.2 Odour –

The herbal tea was prepared, and odour was tested by 8 volunteers and odour was found to be sweet.

2.3.3 Taste –

The herbal tea was tested by 8 volunteers to check the taste and the taste was found to be pleasant.

2.3.4 PH –

The ph of herbal tea was tested by using ph paper and it found to be neutral.

2.3.5 Water Solubility Test –

All herbal ingredients were packed in empty tea bag to check the solubility of drugs in hot & cold water. Tea bag was dipped in hot & cold water and it was found that all the herbal ingredients were soluble in both hot & cold water.

2.3.6 Solubility Time –

The solubility time for both hot & cold water was found to be 30-40 seconds.

2.4 Phytochemical Test [72].

The phytochemical test of the herbal tea was performed as shown in Result Table 5.

Test	Procedure	Observations (Indicating Positive Test)
1. Lead acetate test	1mL extract + few drops of 10% lead acetate solution	A yellow precipitate
2. Froth/ Foam test	0.2ml extract + 5mL distilled water; shaken well; heated to boiling	Appearance of creamy miss of small bubbles
3. Wagner's test	Few mL filtrate + 1-2 drops of Wagner's reagent (Along the sides of test tube)	A brown/reddish precipitate
4. Molish's test	2mL filtrate + 2 drops of alcoholic α–naphthol + 1mL conc. H2SO4 (along the sides of test tube)	A violet ring
5. Biuret test	2mL filtrate + 1 drop of 2% copper sulphate sol. + 1mL of 95% ethanol + KOH pellets	A pink coloured sol. (in ethanoic layer)
6. Salkowski's test	Extract (5 ml) was mixed with chloroform (2 ml), and concentrated sulphuric acid (3 ml) was carefully added to form a layer	A reddish brown coloration
7. Ferric chloride test	Extract aqueous solution + few drops 5% ferric chloride sol.	Dark green/bluish black colour
8. Braymer's test	1mL filtrate+ 3mL distilled water + 3 drops 10% Ferric chloride solution	Blue-green colour
9. Libermann-Burchard's test	50gm extract is dissolved in 2mL acetic anhydride + 1-2 drops of conc. H2SO4 (along the side of test tube)	An array of colour change
10. Borntrager's test	10mL 10% ammonia sol. + few ml filtrate (shaken vigorously for 30 sec.)	A pink, violet, or red coloured solution
11. Ammonium hydroxide test	10mg extract is dissolved in isopropyl alcohol + a drop of conc. ammonium hydroxide solution	Formation of red colour after 2 min.
12. Benedict's test	0.5mL filtrate + 0.5mL Benedict's reagent + Boiled for 2 min.	Green/yellow/red colour
13. Fehling's test	1mL each of Fehling's solution A & B + 1mL filtrate+ boiled in water bath	A red precipitate

Table 03: Procedure of phytochemical test of the herbal tea

2.5 Loss On Drying [Lod] / Moisture Content

The World Health Organizational (WHO) approach indicated for excellent control of medicinal plant substances was adopted (WHO 1998) [73]. One tea bags were selected from formulation and the content emptied into a Petri dish. A 3.253 g quantity of the herbal material was weighed and transferred evaporating dish containing the herbal material was placed into an oven maintained at 60-70°C, removed every 20 min and weighed until no change in weight was obtained over 4 consecutive readings. This was repeated 4 times. The LOD/ moisture content value was determined using Formula 1.

Formula 1 :-

LOD (Average weight) = Initial – Final weight (gm)

2.6 % Lod / % Moisture Content

The percentage moisture content was determined by using Equation 1.

$$\% \text{ Moisture Content} = \frac{W2-W3}{W2-W1} X\ 100 \qquad (1)$$

Where: W1 =weight of empty evaporating dish
W2 = weight of crucible + sample before heating
W3 = weight of crucible = sample after heating.

The percentage loss on drying was determined by using Equation 2.

$$\% \text{ LOD} = \frac{\text{Weight loss of sample}}{\text{Weight of sample}} X\ 100 \qquad (2)$$

2.7 Ash Value

The International Organization for Standardization (ISO) 1575, methods for determination of total ash in tea reviewed and confirmed in 2015 (ISO-1575 2015) [73]. Among 2 herbal tea bags 1 tea bag were selected from formulation and the content emptied in a large Petri dish. A 2.846 gm quantity of the herbal tea was weighed and transferred into a crucible. The empty weight of crucible and empty crucible + weight of sample was weighed. The crucible containing the herbal material was then incinerated. The heating temperature was 525 ± 25 oC. The crucible was allowed to cool in desiccator before the weight was determined. Then it was removed from desiccator and reweighed & constant weight was obtained. The percentage of ash value was determined using Equation 3.
Formula: -

$$\% \text{ Ash value} = \frac{W3-W1}{W2-W1} X\ 100 \qquad (3)$$

Where
W1= weight of empty crucible.
W2= weight of crucible & sample before ash.
W3= weight of ash & crucible.

2.8 Evaluation of Acid Soluble Extractive Matter

The Ash of sample which was obtained to check the ash value was used to determine acid soluble extractive value. The ash sample was emptied into 200ml beaker. To beaker 100 ml of water and 3N HCL was added. Then it was boiled for 5-10 min, & filtered. Then ash present on filter paper was collected into crucible & incinerated at 425± 50 oC. Then cool it and weighed. The percentage of % acid soluble ash value was determined using Equation 4: -

$$\% \text{ Acid insoluble ash value} = \frac{\text{Weight of acid insoluble ash}}{\text{Weight of sample}} X\ 100 \qquad (4)$$

2.9 Evaluation of Antimicrobial Activity

The antimicrobial activity was evaluated on MacConkey agar media by using E.coli bacteria & it was observerd that the formulation shows the antimicrobial activity [74].

C. Result And Discussions –

Figure 4 (A)

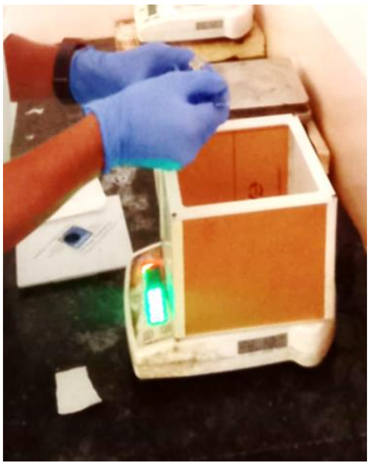


Figure 4 (B)

Figure 4 (C)

Figure 4 (D)

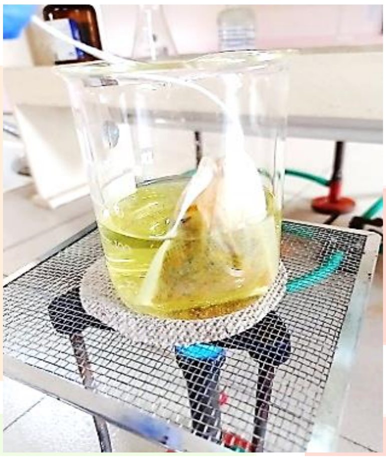


Figure 4 (E)

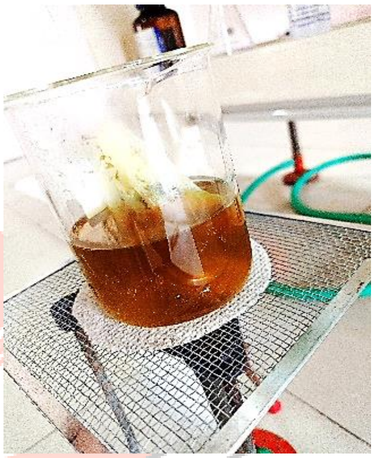


Figure 4 (F)

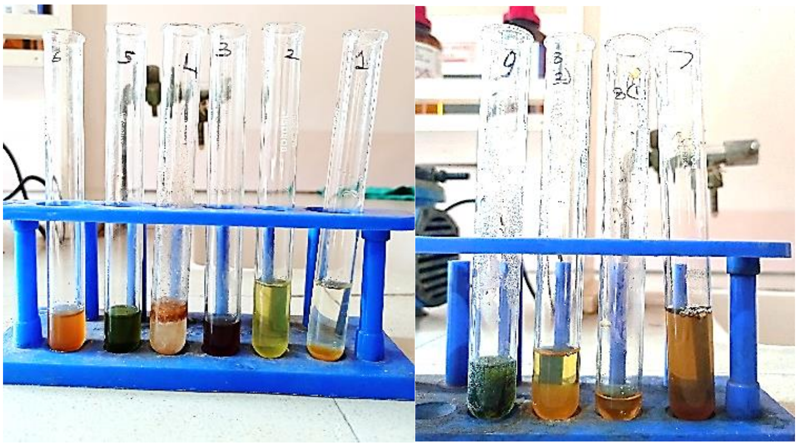


Figure 4 (G)

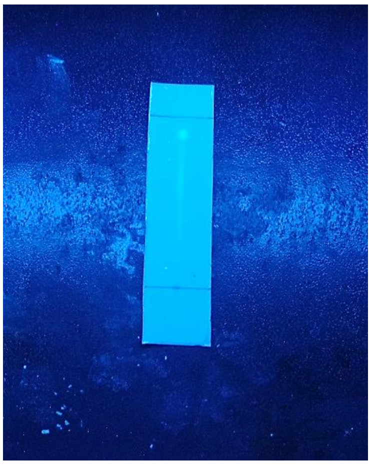


Figure 4 (H)

Figure 4 (I)

Figure 4 (J)

Figure 4:-

- **(A)** Step 1 - Powder formation.
- **(B)** Step 2 - Weighing.
- **(C)** Step 3 - Mixing & filling of drug.
- **(D)** Step 4 - Filled tea bag.
- **(E)** Step 5 - Preparation of tea.
- **(F)** Step 6 - Formulated tea Drug.
- **(G)** Step 7 - Chemical test.
- **(H)** Step 8 - TLC test.
- **(I)** Step 9 - Antimicrobial test.
- **(J)** Step 10 - Ash value test.

D. Discussion

Exploring a polyherbal tea as an antitussive and bronchodilator presents a promising avenue for enhancing respiratory health through natural means. By focusing on the aims outlined in this we can contribute to the growing body of knowledge surrounding herbal medicine and its application in modern healthcare.

Table 4. Physicochemical test –

Sr. No.	Tests	Sub types	Result
1.	**Physicochemical test**	a. Colour	Light green-brown
		b. Odour	Sweet
		c. Taste	Pleasant
		d. pH	Neutral
		e. Water solubility test	Soluble in hot & cold water
		f. Solubility Time	30-40 seconds

Table 5. Phytochemical test –

SR. NO.	TEST	SUBTEST	RESULT
1.	**Phytochemical Test**		
	a. Flavonoids	Lead acetate test	+ve
	b. Saponins	Froth/ Foam test	+ve
	c. Alkaloids	Wagners test	+ve
	d. Carbohydrates	Molish test	+ve
	e. Proteins	Biuret test	-ve
	f. Terpenoids	Salkowski test	-ve
	g. Phenol	Ferric Chloride test	+ve
	h. Tannins	Braymer's test	+ve
	i. Steroids	Liberman buchnard test	- ve
	j. Glycosides:-	Detection of Coumarin :-	
	1) Anthraquinones	a) Borntrager's test	-ve
		b) Ammonium hydroxide test	-ve
	2) Reducing Sugar	a) Felhing test	+ve
		b) Benedicts test	+ve
2.	**Loss On Drying (LOD)**	-	0.466gm
3.	**Percentage LOD**	-	14.3252
4.	**Ash Value**	-	21.082%
5.	**Acid Insoluble Test**	-	14.05%

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