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A Comprehensive Evaluation of Herbal Remedies for the Treatment of COPD

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Opinion

Airflow blockage is a defining feature of chronic obstructive pulmonary disease (COPD). Chronic bronchitis and emphysema are two of the most common illnesses in this category. COPD is a global public health problem, and the disease's incidence is rising. COPD was the sixth major cause of mortality and the twelfth most prevalent cause of illness globally in 1998, according to a World Health Organization report. COPD has significant societal economic implications, both direct and indirect. In the United States, 16 million people have symptomatic COPD, with a total economic cost of $23.9 billion due to COPD-related morbidity and death.

COPD affects around 1.5 million people in the United Kingdom, costing the National Health Service £491 million in direct expenses and £982 million in indirect costs per year. COPD has no known cure. The current standard of care is to alleviate symptoms, avoid recurring exacerbations, maintain optimal lung function, and improve quality of life. The only therapy strategy that has been found to slow disease progression is quitting smoking. Although traditional COPD management has improved, improvement has been gradual.

Unsatisfactory treatment outcomes from conventional pharmaceuticals, as well as harmful effects associated with various pharmacological groups, such as steroids and theophylline, have all contributed to the growing popularity of complementary and alternative medicine, particularly herbal medicine. Herbal medicines have long been used to treat COPD, notably in China, India, and other Asian nations. In several European nations, herbal expectorants based on extracts from Hedera helix or Thymus vulgaris are also quite popular.

Despite its popularity, there has yet to be a full systematic assessment of herbal medications for the treatment of COPD. As a result, the current review's goal was to comprehensively examine five electronic databases were searched without language restrictions from their introduction until August 2005. Bronchitis, chronic obstructive pulmonary disease, COPD, acute exacerbation of chronic bronchitis, AECB, emphysema, herb, botanic, phyto, Chinese medicine, plant extract, plant preparation, and individual common and botanical names were among the search phrases.

Hand searches were conducted in the authors' own library's files and journals. Three herbal remedy makers were approached and requested to provide further information, particularly unpublished data. To find additional suitable trials, the bibliographies of all included trials and other relevant reviews were searched. The clinical studies considered in this study had to be of herbal formulations provided systemically for CB, emphysema, or COPD, with patients of any gender and age receiving either herbal medications or placebo treatments.

Only trials that looked at clinical outcomes, overall clinical efficacy, symptom ratings, health-related quality of life, and the intensity and frequency of exacerbations were considered. Asthma patients were excluded from the trials. Chronic obstructive pulmonary disease (COPD) is a leading source of illness, death, and socioeconomic hardship, with an increasing prevalence. It is predicted that by the year 2020, it will have raised from fourth to third most prevalent cause of death worldwide. Although cigarette smoking is recognised as the cause of COPD in the vast majority of patients, why a minority of smokers develop clinical COPD remains a central question in respiratory medicine.

Inflammation of the airways caused by cigarette smoke is considered to have a key role in the pathogenesis of COPD. It's marked by neutrophil influx into the airway lumen and an increase in macrophages and T-lymphocytes in the airway wall, notably CD8+ cells. The role of oligoclonal CD4+ cells, B cells, and plasma cells has been highlighted in recent investigations. Only a very considerable reduction in smoking to less than five cigarettes daily was linked with lower FEV1 decline, and intermittent full abstinence from smoking resulted in rates of FEV1 decline that were intermediate between persistent quitters and continuing smokers.

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