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EFFECT OF BIOFUEL VULNERABILITY ON LUNGS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS.

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ABSTRACT

Background: Incurable COPD is preventable and treatable. Airflow restriction, persistent lung inflammation, and respiratory tract symptoms lead to bronchiolitis and emphysema. It's caused by constant chemical and gas exposure. Comorbidities and exacerbations aggravate COPD, which is caused by complex biological processes. Globally, COPD will be the third most significant cause of death by 2030. Since the 16th century, COPD, its diseases, and its clinical presentation have been understood. Smoking, tobacco, industrial pollutants, interior pollution, outdoor air pollution, gender, and genetic inheritance affect worldwide incidence, morbidity, and mortality. Chemical exposure and population aging will exacerbate COPD in the coming years. COVID-19 accelerates COPD and slows recovery.

Keywords: COPD, Airflow Restriction, Respiratory Inflammation, Environmental Factors, Covid-19 Synergies

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INTRODUCTION:

COPD, a complicated disorder caused by gas and chemical exposure, is characterized by recurring respiratory symptoms, airflow restriction, and increased airway and lung inflammation. Several host characteristics affect it (Colarusso et al., 2017). Inhaling harmful gases and particles from Biomass-based fuels and cigarette smoke causes emphysema and airway fibrosis, which damage parenchymal tissues and disrupt the body's defense and repair processes, increasing lung inflammation. Regardless of FEV1, smoking is strongly connected to disease development, emphysema, and poor prognosis. Never-smokers had a better COPD prognosis in Western nations (Mitra et al., 2022). With high mortality rates, COPD is the leading cause of worldwide deaths. It was the third most significant cause of mortality worldwide in 2010 and is expected to become the fourth and fifth top cause of COPD disease incidence by 2030. The most frequent COPD phenotype is partially caused by active smoking, among other

factors. Smokers do not always get COPD, indicating that intrinsic or extrinsic factors contribute to thedisease. COPD patients also vary clinically. It is also well-recognized that COPD patients' inception, early phases, and development vary greatly. Comorbidities, including coronary artery disease, diabetes, lung cancer, and osteoporosis, affect how COPD develops in older adults. Early COPD detection has been hampered by clinical delays, early pulmonary function loss, patient social and psychological variables and early pulmonary function loss (Lange et al., 2021). Epidemiological studies on COPD are limited to specific locations. This project aims to evaluate and assemble data on the epidemiological effect of COPD and its risk factors worldwide. This article also discusses advances in COPD therapy (Hu et al., 2015).

Origin and history of COPD

In 16th-century literature, emphysema was described as having "voluminous lungs." Morgagni described 19 air-induced "turgid lungs," and Baille portrayed Samuel Johnson's emphysematous lungs in 1789 (Ayilya & Nazeer, 2023). Persistent bronchitis and COPD wereInitially described by Badham in 1814. He labeled the main symptoms, a persistent cough with excessive mucus output, "catarrh," and bronchiolitis and chronic bronchitis, "disabling disorders." William Briscoe may have coined "COPD" during the "9th Aspen Emphysema Conference" to start a conversation. Current COPD definition: progressively worsening health concerns (Bustos, 2022). The 1962 US Thoracic Society Commission on Diagnostic Standards and the Central Institute for Brackish Thoracic Diseases defined COPD and its components. Water Aquaculture (CIBA) Guest Symposium, 1959. In 1993, The Lung Section of the National Heart, Lung, and Blood Institute (NHLBI) created "The National Lung Health Education Programme (NLHEP)" to study COPD. The disease's pathophysiology became clearer (Wu et al., 2021). In 2001, WHO and NHLBI launched GOLD to increase awareness of the condition's severity and encourage early detection and treatment. In 2004, 'The European Respiratory Society (ERS)' and 'The American Thoracic Society (ATS)' recommended COPD. Charles Fletcher meticulously investigates the natural course of COPD by identifying smoking risk indicators and the abrupt reduction in FEV1 insusceptible smokers to correlate to debilitating symptoms. The scientific foundation for stopping smoking at different illness stages was established. When high slumbers had the worst prognosis, Burrows et al. named it "The Horse Racing Effect." This showed the need for early diagnosis and treatment. Spirometry can show early physiological changes due to complicated biochemical and cellular responses that are damaged with loss of elastic recoil in the tiny airways and alveoli, lung expansion, and a rise in FVC. Clinical symptoms often emerge in severe and intermediate COPD (Ayilya & Nazeer, 2023).

Epidemiology of chronic obstructive pulmonary disease in LMICs (low- and middle-income countries)

Most LMICs are in Eurasia, Latin America, and Africa. Since primary care institutions in LMICs don't collect data, prevalence surveys are our primary source of information on COPD's etiology. Asia has conducted most surveys on chronic respiratory disorders in LMICs, and the following methods are used to estimate COPD prevalence: GOLD standard definition (clinical symptoms with past dangers verified by spirometry) orFEV1/FVC ratio, where applicable (Pollard et al., 2023). India, China, and Indonesia are three middle-income Asian countries where half the world smokes. Asia produces most tobacco. Also, Bangladesh has significant smoking rates. Based on specific risk variables and epidemiological connections, Hong Kong and Singapore have 3.5%, Vietnam 6.7%, and Malaysia. 4.7%, Thailand 5%, Indonesia 5.6%, the Philippines 6.3%, Taiwan 5.4%, South Korea 5.9%, and Japan 6.1% (Islami et al., 2015). East and South-East Asian COPD prevalence data is sparse. Using standardized spirometry, the 2014 BOLD study in India found overall COPD prevalence rates of 5.7% to 17.3% for men and 6.8% for females in Pune, Mumbai, and Srinagar. Hand-held spirometers were utilized to assess Bangladesh's 10.3% COPD incidence using GOLD-defining criteria and LLL standards. Males showed a higher COPD incidence than females, and over half of all cases were stage II (or "moderate") (Ho et al., 2019). The hidden COPD epidemic in Africa is a significant public health concern. Africans had 26.3 million COPD cases in 2010, up 31.5% over a decade, especially among older people. A systematic review of nine cross-sectional and spirometry-based studies including 3673 people from South Africa, Nigeria, Malawi, and Cape Verde) Depending on diagnostic criteria, COPD prevalence ranges from 4% to 25% (Rossaki et al., 2021). The risk of COPD in Africa south of the Sahara has gotten little attention. Statistics from spirometry generally reveal a higher prevalence. Spirometry data showed that the average incidence of COPD in those under 40 was 13.4%, compared to 4.0% for those using questionnaires to assess exposures, demographics, and symptoms (Bai et al., 2022). Ho et al. also discovered that chronic airflow obstruction prevalence varies by country and demographic. The high smoking rate (30%) in South and Central America increases respiratory problems in the US. The PLATINO and PREPOCOL population-based investigations estimated the prevalence of COPD in people under 40 in several major urban centers to be between 6.2% and 19.6%, with up to 89% underdiagnosis and excessive diagnosis due to the lack of spirometric confirmation. A recent study of studies evaluating COPD incidence using portable spirometry and questionnaires in a few Latin American locations reported a range of 7.8% to 19.7%. Larger and more Representative studies of numerous communities globally, especially in rural areas, have not been done.COPD seems to be growing worldwide. Although ICs have the highest formal prevalence and incidence, LMICs' official data, which show far lower rates, are thought to misrepresent reality. In 2019, 5.4% of HICs had COPD,

compared to 1.1% of LMICs. Unawareness, underdiagnosis, and underreporting of COPD in LMICs reduce its prevalence (Pleasants et al., 2016). Due to their reliance on forecasts rather than epidemiological data and risk factor exposure, most estimates of LMIC COPD incidence were inaccurate. Thus, lawmakers lack the data to create meaningful cost-cutting programs for treating illnesses. Due to expanding life expectancy, reducing infant mortality, and improved COPD awareness, LMIC occurrences are expected to rise (Prince et al., 2015).Differences between the distribution of tobacco and biofuel vulnerabilityCOPD and tobacco exposure are biomass products, but the social context distinguishes them epidemiologically. Tobacco smoke is intensively processed and industrialized and includes several inorganic harmful compounds. Most COPD knowledge originates from industrialized cigarette smoke derived from unprocessed organic components (Ramírez-Venegas et al., 2021). From 1913 until 1920, tobacco industry breakthroughs caused the tobacco pandemic. The tobacco industry flourished unfettered locally and worldwide in the 1960s, killing millions (Celli & Agustí, 2018). Since humans began utilizing organic fuels for heating and cooking, billions of women and children have been exposed to Biomass without knowing the health risks. Because poverty limited electricity, rural women had to fire Biomass, cook, and heat their houses (Bouza et al., 2020). The tobacco industry pushed recreational smoking to create a nicotine demand. Tobacco use, formerly associated with wealth and social status, is now connected to BE-COPD, which affects low- and middle-incomepeople. Unfortunately, biomass smoke has long been linked to poverty and underdevelopment (Bouza et al., 2020). Approximately 1,100 billion people have been exposed to tobacco. These figures are far lower than almost half the world's population. This proportion is higher in rural and impoverished countries (Alkan & Abar, 2020).

The use of biogas fuel causes COPD in rural women.

In most countries, rural women lead their homes,udy, a, and cook when men are away for different reasons. Home pollution is women's most common source of pollutants, particularly in developing countries, due to biomass-based fuel's low burning efficiency, carbon monoxide, hydrocarbons, and chlorinated organics are produced, which are harmful to respiratory health and the leading cause of BE-COPD (Jindal & Jindal, 2021). High indoor smoking exposure, especially in poorer countries, doubles COPD risk. COPD was the third most significant cause of death worldwide in 2019, accounting for 6% of fatalities. WHO reports COPD as the fourth most important cause of mortality in lower-middle-income countries, causing overOne million deaths in 2019 (Ramírez-Venegas et al., 2018). Data linking biomass exposure to COPD is considerable. Women who cook with indoor smoke are more likely to have chronic bronchitis than those who use electricity or gas. The sickness is more common in men than women; hence, it may be considered a disorder that only affects women. Its exclusive impact on women and rising global female illness rates make it a potential public health issue (Ramírez-Venegas et al., 2019).

The Beginning Lung Damage Due to Biomass Exposure

Rural women spend long hours cooking, frequently inside in unventilated rooms. This cooking method emits air pollution. Biomass burnt in unvented open fires causes milligrams per cubic meter of pollution in homes (Lange et al., 2021). Most women and girls' cumulative exposures occur over time. On average, they spend four to eight hours a day in the cramped, poorly ventilated-kitchen. They stay awake for almost half the day in a tiny, poorly ventilated-kitchen. They may have been exposed for 40 years (Assad et al., 2016). They breathe nearly 25 million liters of smoke-

containing particles for over 60,000 hours. This sustained, high-level exposure increases the risk of airway wall inflammation, which may take years to show. Continuous biomass exposure in infancy may cause recurrent respiratory tract infections that modify airway walls or predispose (Sana et al., 2018).

Risk factors

Behavioral risk factors: tobacco smoking

The most significant contributing factor to COPD is generally believed to be tobacco use. More than 80% of the 1.3 billion smokers in the world reside in LMICs. In 2015, smoking alone was the cause of about 1.5 million Cardiovascular, Obstructive Pulmonary Disease-related deaths. However, LMICs are home to most non-parties (Keto et al., 2016). Quitting smoking lowers family danger from secondhand smoke and one's chance of acquiring COPD. It shouldn't, however, solely be seen as a preventative step. The only COPD intervention that has been shown to reduce COPD development is quitting smoking. This is also known as Article 14 of the FCTC. A significant number of smokers in many nations have access to Primary care for help quitting (Soneji et al., 2017).

Environmental risk factors: air pollution

Air pollution was blamed for almost 1.8 million COPD-related fatalities in 2015. Additionally, exposure to air pollution at work (such as particulate matter, fumes, as well as gases) was linked to 354,000 COPD-related fatalities. Household (interior) and ambient (outdoor) pollution are two different types of air pollution (To et al., 2016).Using fuels from Biomass (such as wood, agricultural waste, or animal dung) for heat and cooking is the leading cause of indoor air pollution. Other instances include lighting kerosene lights, lighting incense at places of worship, and lighting mosquito coils. Solid fuels are used by three billion people globally or 40% of homes, and 90% of rural LMIC families rely on them. The consumption of solid fuel is centered on LMICs, particularly in Africa. Air pollution in the home poses a significant risk to one's health. An exposure-related 41% increase in the risk of COPD was found in an up-to-date investigation that focused on residential air pollution in Thirteen LMICs. Women who live inrural locations are more at risk for acquiring COPD as a result of biomass exposure, probably because of the prolonged time spent indoors (de Miguel-Díez et al., 2019). It's interesting to note that the phenotype of COPD may vary depending on the disease's etiology. For instance, COPD caused by Biomass is more frequently seen in younger individuals. The exposure to ambient air pollution from

sources such as forest fires, storms of dust, and vehicle and industrial exhausts is highest in developing nations. In locations with low- and low-middle sociodemographic indices during the past ten years, there has been a marked rise in DALYs attributable to ambient air pollution. As a result, air pollution continues to be an essential risk indicator for DALYs in LMICs, particularly in South Asia and eastern sub-Saharan Africa (Li et al., 2016). The seventh strategic recommendation of the Sustainable Development Goals is to convert to more accessible, greener energy. There are schemes for switching to greener fuels in Indonesia and India. If switching is not an option, using better cookstoves or enhancing ventilation are workable intermediate measures.However, various programs to provide better cookstoves Haven't shown sustained results. Research and discussion on the link between COPD and biomass exposure are ongoing. Several research studies have shown contradictory and erratic relationships between airflow restriction and indoor pollution, including the BOLD (Hu et al., 2015). The evidence supporting the involvement of environmental pollutants in the genesis of COPD is also suggestive but not definitive. According to the Global Burden of Illness research, exposure to environmental pollutants is a significant reason for COPD in low-income countries. However, chronic contact with polluted indoor air is challenging to assess. It may also be linked to other risk factors, such as poverty and a lower socioeconomic level, leading to poorer eating habits and reduced fetal lung development (Santos et al., 2021).

Genetic and physiological risk factors: host characteristics

The relationship between genetic predisposition to COPD and early-life risk factors, beginning with conception, is a relatively recent issue that has drawn interest. The development and trajectory of embryonic lung formation or lungthe function is influenced by maternal health and lifestyle factors (such as asthma, smoking, and diet). Lung function is strongly influenced by early-age difficulties such as low birth weight, preterm, starvation, childhood respiratory illnesses, and allergy disorders (Agustí et al., 2022). Similarly, prolonged exposure to air pollution slows the development of full lung capacity in children and hastens aging. It has become crucial since the development of COPD seems to be influenced by lung function. Similarly, having a low body weight, which is typically the result of malnutrition, increases your likelihood of developing COPD, has a worse prognosis, and affects your ability to breathe (Agustí et al., 2020). In terms of diseases, Human immunodeficiency virus and TB are both quite common in numerous LMICs, have devastating impacts on the lungs, or are thus COPD risk factors. A risk factor of COPD may also include non-infectious disorders. For instance, asthma is linked to COPD as a result of reduced lung function. Despite the reality that asthma is thought to be poorly diagnosed and poorly understood in LMICs, the frequency of the condition is rising there, and its considerableSeverity has been recorded there. It is clear that clinical tools are required for correct diagnosis, and incentives for systems that allow for routine evaluations to assure disease control are also needed (Lange et al., 2021).

Role of climatic factors and air pollution in spreading COPD

Climate variables, including air pressure, humidity, and air temperature, all affect how harmful air pollutants affect COPD. The relationship between air temperature and the pathogenic impact of contaminants on COPD is complex. However, there is debate concerning the distinctions between the influence of hot and cold temperatures. Lower temperature significantly exacerbated the impact of PM2.5, PM10, and the level of SO2 affecting the COPD hospitalization rate, as proved by research by Qiu et al. (Hansel et al., 2016). Several studies from China found the possibility that the admissions to the hospital for COPD “are more associated with SO2, NO2, and O3 in the colder seasons than in summertime" and "the association between air pollution and the hospitalization rate for COPD is stronger in autumn than in winter." These results can be explained by higher temperatures making a "substantially greater proportion of O3, as well asother pollutants, penetrate the atmosphere". More recent investigations in these countries have shown that "the days when the temperature is very high with a considerable variation in barometric pressure were significant in increasing the hospitalization rate of COPD in downtown elderly patients. The investigators postulated that fluctuating pressure may aggravate the symptoms of COPD. Moreover, increased atmospheric pollution on hot days could also play a role in worsening COPD. Gao et al.'s study showed that air contamination was more influential in the summer months (August to October) for those suffering from COPD than in winter (November to March). Admission for COPD may also be influenced by relative humidity. In one study, the likelihood of COPD hospitalizations increased by 1.070 (95% CI: 1.054-1.086) for every 1% increase in relative humidity. Furthermore, temperature and humidity significantly interacted (Alahmari et al., 2015). It is still unclear what exact impact air pollution has on the pathophysiology of people with COPD. However, current research focuses on factors such as oxidative stress, inflammatory damage, and DNA damage.

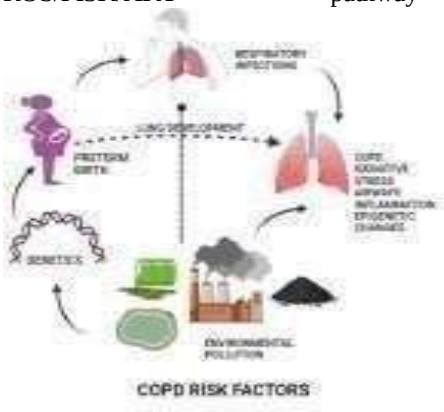
Oxidative stress damage

The pathophysiology of COPD is significantly influenced by free radicals generated by oxidative stress, mainly oxygen free radicals. Reactive oxygen species, also known as ROS, can be produced in huge quantities by cells when free radicals of oxygen, which are made by PM, are inhaled. Additionally, the vast concentrations of metal and organic debris carried by PM might cause the creation of ROS in cells. The fundamental source of harm brought on by PM exposure may be the oxidative breakdown of lung cells caused by ROS (Kruk et al., 2019).

Genetic damage

Within a particular concentration range, contact with air contamination can result in genotoxicity, which damages chromosomes in living organisms. In addition to harming lung epithelial cells with alveolar macrophages, exposure to PM2.5 can increase the formation of ROS brought on by oxidative stresses, which results in DNA damage and alterations in gene expression. Without any changes to the DNA sequence, epigenetic modifications to DNA, such as

DNA methylation, histone modifications, and non-coding RNA modifications can affect how genes are expressed. The development of COPD may be influenced by air pollution through changes to epigenetic alterations (Boas et al., 2018). Nitric oxide (FeNO) exhaled while breathing is regarded as a sensitive indicator of airway inflammation and can provide insight into the severity of airway infection in COPD patients. A Shanghai panel research that looked at the connection between PM2.5 and DNA methylation in COPD discovered that PM2.5 may control the generation of FeNO by altering the methylation of biomarkers in the NOS2A promoter zone and escalating airway inflammation (Song et al.) discovered that PM2.5 may enhance the expression of IKK- and maintain NF-B activation in human airway epithelium cells by inhibiting miR-331 by disrupting the ROS/PI3K/AKT pathway (Heffler.



Protective measures Policy intervention

Improvements in air pollution levels are primarily due to government regulation. For instance, there was historically significant air pollution in the United States. As a result, the American government created several policies to prevent and reduce air pollution, including the amendments to the Clean Air Act in 1990 and the NOx State Strategy Plans Call in 2002. PM2.5 and O3 concentrations drastically dropped in the United States after 1990 (Zhang & Samet, 2015). The burden of COPD mortality has dramatically lowered due to air quality improvements. China saw the worst pollution occurrences in the previous ten years, a period of tremendous industrial expansion andurbanization. The government has enacted several effective preventative and control measures to reduce air pollution in China. The Air Pollution Strategy Control and Prevention Plan (2013-2017) was created by the state council in 2013. The Three-Year Action Strategy Towards Winning the Blue Sky Defense Conflict, published by the state council in 2018, served as a roadmap for the subsequent phase of air pollution management and prevention (Zhang et al., 2016). The Beijing City Strategic Plan (2016-2035) is one of the comparable policies that the provincial and municipal administrations have also released (Duan et al., 2020). Since 2013, PM2.5, PM-10, and SO2 levels have dramatically dropped, and most Chinese cities have improved air quality. Emission regulations decreased PM2.5 mortality by 88.7%, and these measures also had positive health effects. According to Liang et al., the levels of SO2 and PM2.5 were 68 percent and 33% lower in 2017 than in 2013.

The number of advanced acute COPD exacerbations caused by PM2.5 exposure also showed a trend toward decline (Hooftman et al., 2018). Despite a decrease in air pollution, the WHO requirements for air quality have not yetBeen met. The levels of O3 have drastically increased, the NO2 concentration is unchanged, and the PM quantities are still high. Therefore, to limit emissions from industrial pollution sources and improve air quality, the government should develop effective air prevention and control measures (Maloney & McCormick, 2017).

Group intervention

Burning solid Biomass for cooking is the primary cause of indoor air pollution, which is linked to several respiratory conditions. Around a third of the worldwide population, particularly in rural LMICs, utilizes biofuels like charcoal and wood for heating or cooking. Numerous studies have found a connection between exposure to biofuels and a higher risk of COPD. To investigate the relationship between solid fuel consumption and the chances of developing both acute and persistent respiratory illnesses, cohort research was carried out in China, which included 277,838 Chinese people who had never used tobacco and had not had significant persistent diseases in the previous nine years (Balmes, 2019). According to the findings, solid fuel consumers had an increased hazard ratio forCOPD of 1.10, which is (95% CI: 1.03-1.18) When compared to clean fuel users. Using clean fuel with ventilation equipment can decrease the danger of respiratory infections. Additionally, using ventilation kitchenware or clean fuel can significantly lower the threat of respiratory illnesses (Assad et al.).Zhou et al. carried out a non-randomized intervention to evaluate the long-term effects of better kitchen ventilation and alternative biomass fuel consumption on lung function in COPD patients. According to the findings, the use of biogas in place of Biomass for cooking and better kitchen ventilation was associated with a lower chance of having poor FEV1 and COPD. Additionally, a dose-response connection was seen: the longer enhanced kitchen ventilation and cooking fuel utilization lasted, the more of an influence it had on slowing the loss of lung function (Duan et al., 2020). Using upgraded cookstoves also helped lower the risk of COPD, the frequency of respiratory symptoms, including coughing, expectoration, and wheezing, and the pollution caused by biofuels. To reduce the incidence of COPD, better cookstoves, ventedCookware, better kitchen ventilation, and pure fuel must be utilized.

Individual intervention

When smog concentrations are high, it is best to limit the time and intensity spent outside and to wear masks that are good at trapping PM2.5 particles to lessen exposure. Wearing personal protection equipment, like the N95 mask or a comparable product, may help reduce your exposure to ambient air pollutants' detrimental effects. The majority of studies on the preventive effects of masks so far have been conducted on healthy individuals or those in specific

vocations, and there have been very few studies done on sensitive persons like COPD sufferers (Laumbach et al., 2015). Research by Sundblad et al. involved 36 healthy individuals. According to the results, the participants using masks had a lower concentration of systemic inflammatory markers and relatively more excellent lung function indicators than the group not wearing masks. The study proved that wearing a mask might help to protect your respiratory system. Indoor purifiers for air can lower the amount of PM2.5 in the air and help with indoor air pollution. Uncertainty persists onwhether this will enhance cardiopulmonary function (Jiang et al., 2016). A more efficient experimental investigation is required to assess the real preventive impact of air purification systems and masks in people with chronic pulmonary disease (COPD).

Medical intervention

In addition to the aforementioned activities, various pharmacological interventions, such as nebulization therapy, may help encourage the removal of PM in the lower respiratory system in COPD patients and reduce the adverse impacts of air pollution. However, there are currently no suggested limits for lowering air pollution linked to atomizer inhalers (Laumbach et al., 2015).

ADVANCES IN DRUG DEVELOPMENT FOR COPD

Chronic obstructive pulmonary disease, also known as COPD maintenance therapy, which may include a variety of pharmacological treatments, concentrates on symptom relief and lowering the risk of disease progress, exacerbation, and death. Due to the variability of COPD, pharmaceutical therapies can causePeople do respond differently. Over time, therapy choices for COPD have changed, moving from accuracy in pharmacologic strategy to optimizing treatments based on data from integrating clinical and biomarkers. The best available evidence supports the use of combination therapy at varying levels, according to studiesundertaken by several researchers (Barnes et al., 2015). The GOLD team strongly advised the need for pharmacological therapies for exacerbations and dyspnea and symptom-specific therapeutic approaches to reduce risks, reduce symptoms and frequency, and improve health and exercise tolerance. On the other hand, there is currently no adequate clinical data to support any COPD treatment that can enhance lung function over the long term. Pharmacological treatment may be customized for people with different pathophysiological causes using biomarkers. Vaccinations are effective early prevention methods (Doryab et al., 2016). Pharmacological treatment is prescribed based on the degree of lung damage and symptoms, including cough, sputum production, dyspnea, and exacerbation levels. The class of drugs that includes anticholinergics, asthmatics, inhaled corticosteroids (ICS), beta2- agonists, antimuscarinic medications, methylxanthines, phosphodiesterase-4 inhibiting agents, anti-inflammatory agents, and mucolytic agents include about 37 generic drugs, which are widely used worldwide. Although most medications work to relieve symptoms, some have unpleasant side effects. This emphasizes the need for more naturally occurring bioactive substances with fewer adverse effects (Diebold et al., 2015).

Anti-COPD compounds from natural sources

In cultured airway human epithelial cells, nine bioactive substances screened from underwater brown algae (Ecklonia cava, Ishige foliacea, Ishige Okamura, and Hizikia fusiformis) and Apo9 1 fucoxanthin one obtained from Undariopsis peteseniana show superior protection against the cytotoxicity induced by cigarette smoke by preventing cigarette smoke-induced apoptosis, DNA damage, and mitochondria- According to research, astaxanthin and xanthophyll carotenoids with a naturally occurring reddish-orange color that is abundant in marine species including algae,Crab, shrimp, krill, and salmon have the most potent antioxidant effects of all the carotenoids and vitamin E (Rahman et al., 2022). Raising the expression of Nrf2 and HO-1 in the lung prevents mice from developing emphysema by cigarette smoke. While the astaxanthin content in the mice's blood and its bioavailability were not established, and the ideal concentration of astaxanthin has not yet been tested for efficacy, the molecule was effective in vitro and a mouse model of COPD. Brevenal, a marine dinoflagellate called Karenia brevis, has been suggested as a viable anti-inflammatory medication for mucociliary clearance since it lowers pre-inflammatory mediators while maintaining anti-inflammatory release from the cells on various cell lines. However, a vivo investigation would be necessary to demonstrate the compound's physiological benefit without affecting the immune system's mending ability (Yang et al., 2020). Two polyphenols from Jaboticaba, 3,3′-dimethyellagic acid-4-O-sulfate and Jaboticaba, showed anti-inflammatory effects in cell lines and may be used to treat COPD; however,animal studies are still needed to corroboratethese results. Naringenin, an organic flavanone derived from dormant Prunus persica flower petals, was hypothesized to have potential pharmacological effects against various phases of COPD. Humans ingest it, which is abundant in citrus veggies and fruit and has a wide range of bioactivity (Inada et al., 2021). Although the biological effect of this flavanone has been thoroughly investigated both in vitro and in vivo, additional clinical research is required to support its impact on humans, which are constrained by its poor bioavailability and solubility in aqueous solutions and need an efficient drug delivery mechanism. Most of the potentially effective anti-COPD compounds investigated were from plant sources, with very few coming from marine algae. These compounds tended to be more phenolic and flavonoid-based, with less bioavailability and hence heavier for stomach absorption and thus needed stronger supporting data based on in vivo investigations (Ayilya & Nazeer, 2023).

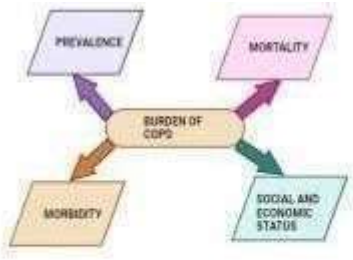
Burden of COPD

One of the leading causes of illness and mortality worldwide is COPD, according to experts. Depending on the environmental factors thatVarious people are exposed to the

incidence, mortality, and morbidity of COPD may differ around the world. The most common cause of COPD is tobacco use. At the same time, other variables, including occupational exposure and air pollution from both indoors and outdoors, also play a substantial role in many nations (López-Campos et al., 2016). The prevalence and incidence of COPD are predicted to significantly increase over the next several decades when taking into account ongoing exposure to hazardous chemicals and the aging of the global population (Zhu et al., 2018).

Prevalence

Between 1990 and 2015, the prevalence of COPD grew by approximately 44.2% worldwide. 'The World's Burden of Disease Study' projected that 251 million people worldwide had COPD in 2016. Over the years, several meta-analyses and systematic reviews have been carried out, and the combined prevalence of COPD from 37 studies was found to be 7.6%, and the prevalence from 26 approximated spirometrics to be 8.9%. Similar to this, research done in over 28 nations between 1990 and 2004 indicated that smokers and ex-smokers over the age of 40 had a higher prevalence of COPD than non-smokers (Lee & Rhee, 2021). Additionally, it was discovered to be somewhat higher in men than in women. The Burden of Obstructed Lung Disease (BOLD) program examines the prevalence and risk of COPD among the global population over 40 years of age across 38 countries, nine of which are still being studied, using standardized pre- and post-questionnaires and data retrieved from various spirometric studies. According to BOLD, the prevalence of grade 2 or above was 10.1% overall, 11.8% among men, and 8.5% among women, with a frequency of 3-11% among non-smokers. 9.2% of people in low- and middle-income nations have COPD (Machado- Duque et al., 2023). Over 90% of COPD-related fatalities, as reported by WHO (2021), are attributable to them. Additionally, estimations from meta-analyses indicated a prevalence of version 10.6 in LMICs among those over the age of 30. From 28.1 million cases recorded in 1990 to a startling 55.3 million cases reported in 2016 in India, COPD prevalence increased from 3.3 percent to 4.2%. Additionally, in less developed lower Epidemiologic Transitional Levels (ETL) states of Uttar Pradesh as well as Rajasthan inIndia, the DALY rates and age-standardized COPD prevalence were highest in 2016 (Selçuk, 2020).



Morbidity and mortality

Morbidity includes illnesses that require medical attention, ER visits, and hospital stays. Studies show that COPD morbidity rises with age, and people with COPD develop complications at a relatively young age since the information gathered from these variables is less trustworthy and not as easily accessible as that from the mortality data. Other chronic disorders, such as cardiovascular disease, diabetes mellitus, etc, also influence the morbidity among COPD patients. COPD was ranked as the third leading cause of mortality in America in 2011 (May & Li). With over 90% of deaths occurring in low- and middle-income countries, COPD is a progressive respiratory disease that caused 3 million deaths in 2012, 5.7% of all deaths globally, 3.2 million deaths in 2017, and3.23 million deaths out of 55.4 million worldwide in 2019. The WHO had predicted that COPD would be the third most common cause of death by 2030. However, the condition was instead rated as the second-highest rate of death in 2017. In the next 40 years, the prevalence of COPD will rise due to rising rates of smoking in several developing countries and an aging population in high-income nations (Ko et al., 2016). By 2060, it is anticipated that there will be 5.4 million per year related to COPD along with its associated conditions. Additionally, the growing smoking epidemic, lower mortality rates from other diseases like heart attacks and strokes, the aging of individuals in high-income nations, and relatively lax measures to prevent illness in developing nations can all be linked to the significant rise in deaths from COPD (Criner & Han, 2018).

Social and economic burden

In 2016, India was responsible for 32 percent of all DALYs worldwide caused by chronic respiratory disorders, with COPD accounting for 75.6% of all DALYs in that country. This very high illness burden and enormous health loss in India, especially in the states with low epidemiological transition levels, highlight the need for more converging policy actions to look into the country's enormous disease burden. In 2016, India's DALYs per case of COPD were 1.7 times Ligher than the global average, and even most The states in the nation had higher DALY rates than most other regions with comparable Socio-demographic indices globally (Kalkana et al., 2016; ur Rehman et al., 2020). Around 6% of the total annual health budget in the European Union, or 38.6 billion euros, is estimated to be set aside for respiratory diseases, of which COPD accounts for 56%. In the United States, the cost of COPD is predicted to be $32 billion or $20 billion, respectively. Previous research indicates that the impact on the Indian economic growth in terms of direct and indirect medical expenditure was indicated to be considerably high (direct medical cost: roughly Rs. 29,885 11,995.33, or US$300-500; direct nonmedical cost: roughly Rs. 7,441.25 2,228.90, or US$90-155) and is also related to a lack of daily wages for an essential amount of time (Zafari et al., 2021).

Conclusion

Much data supports the claim that respiratory illnesses like COPD are linked to air pollution. Epidemiological and clinical investigations have proved the relationship between pollution in the air and COPD. Air pollution has a detrimental influence on the onset and development of COPD, both over short and extended periods. It is crucial to pay greater attention to air pollution because it is one of the main risk factors for COPD and may be modified. In the future, extensive clinical research and in-depth study of fundamental mechanisms will be required, which can improve patient treatment and encourage policymakers to support healthcare initiatives to eradicate air pollution internationally.

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Summary

The Review examines how biofuel inhalation affects lung health in Chronic Obstructive Pulmonary Disease (COPD) patients. Studies establish that biomass fuel combustion remains a primary source of indoor pollution that specifically affects rural women together with low-income families. Across low- and middle-income countries (LMICs) researchers study COPD epidemiology by identifying smoking as well as environmental pollutants as main risk factors. The researched document explains how biomass exposure affects individuals through genetic vulnerability together with oxidative damage in combination with inflammatory reactions that build up from sustained biomass contact. The article investigates protective measures for COPD progression which encompass both improved stove technology and policy enforcement with private protection actions such as mask use and medicine-based therapy. The report documents the necessity of public health actions along with enhanced air quality legislations and directed COPD therapy approaches to minimize disease impacts on susceptible communities.

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