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Review

Air pollution and chronic obstructive pulmonary disease

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Abstract

There is considerable epidemiological evidence indicating that air pollution has adverse effects on human health and is closely related to respiratory diseases, including chronic obstructive pulmonary disease (COPD). These effects, which can be divided into short- and long-term effects, can manifest as an exacerbation of existing symptoms, impaired lung function, and increased hospitalization and mortality rates. Long-term exposure to air with a high concentration of pollutants may also increase the incidence of COPD. The combined effects of different pollutants may become more complex in the future; hence, there is a need for more intensive research on specific at-risk populations, and formulating corresponding protective strategies is crucial. We aimed to review the epidemiological evidence on the effect of air pollution on COPD, the possible pathophysiological mechanisms underlying this effect, as well as protective measures against the effects of air pollutants in patients with COPD.

Keywords: Air pollution; Chronic obstructive pulmonary disease; Air pollutant; Health effect

Introduction

Air pollution is a major public health issue that affects all parts of the world. It is the leading cause of morbidity and mortality and contributes to the global disease burden. The Global Burden of Disease (GBD) study reported that exposure to $PM_{2.5}$ led to 4.2 million deaths and 103.1 million disability-adjusted life years (DALYs) worldwide in 2015, accounting for 7.6% of the total global deaths and 4.2% of the global DALYs.[1] China is the fastest growing and largest developing country; thus, air pollution in China is becoming increasingly severe, making air pollution-related mortality in China one of the highest worldwide. In addition, in 2017, ambient particulate matter (PM) pollution became the fourth-largest health risk factor contributing to the years of life lost among Chinese residents.[2]

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Exposure to ambient air pollutants can cause damage to multiple organs and systems of the human body, thus adversely affecting health.[3] The respiratory tract has direct exposure to external surroundings and is more vulnerable to pollutants than other systems. Epidemiological and mechanistic studies have shown a close relationship between air pollution and multiple respiratory diseases, especially in patients with underlying lung diseases such as chronic obstructive pulmonary disease (COPD).

COPD is a heterogeneous disease characterized by persistent respiratory symptoms and airflow limitations. This disease is associated with significant exposure to noxious particles or gases and influenced by host factors, including abnormal lung development.[4] The incidence and mortality of COPD have increased every year, leading to a serious economic and social burden. The GBD study estimates that the global prevalence of COPD is approximately 174 million.[5] The China Pulmonary Health (CPH) study showed that the prevalence of COPD was 13.7% among individuals aged 40 years and older.[6] According to the World Health Organization (WHO), COPD will become the third leading cause of death globally and the fifth leading cause of economic disease burden worldwide in 2020.[7] Smoking, air pollution, occupational exposure, respiratory infection, and genetic factors such as a_1-antitrypsin deficiency are all risk factors for COPD, with cigarette smoke being the largest contributor to COPD development, followed by air pollutants.[6,8]

In recent decades, many environmental epidemiological and toxicological studies have been conducted to explore the effects of air pollution on COPD. Here, we aimed to review the epidemiological evidence on the effect of air pollution on COPD, the mechanisms underlying this effect, as well as the protective measures that may help reduce the effect of air pollution, such as COPD development, among susceptible patients. This review will use epidemiological studies focusing on ambient air pollution to demonstrate the significant health effects of ambient air pollution on COPD.

Epidemiology of air pollution and classification of air pollutants

Owing to developments in global industrialization in recent years, air pollution has become one of the biggest environmental and public health problems globally. Although pollution levels in high-income countries have reduced over the past 25 years, the levels in low- and middle-income countries (LMICs) such as China and India have sharply increased over the same period.[9] According to the Global Air Quality Report released by the WHO, nine out of ten people around the world breathe air with high levels of pollutants.[10] With increased industrialization, air pollution in China has become severe. In 2013, severe and persistent haze-fog was observed in China.[11] To improve air quality, China formulated the national Air Pollution Prevention and Control Action Plan in 2013.[12] After a period of significant effort, the air pollutant levels in key regions improved significantly,[13,14] but the overall air quality has not reached an acceptable standard.

Air pollution is caused by a complex mixture of particles, vapors, and gases emitted from natural and synthetic sources and formed through the process of photochemical transformation. The most commonly monitored air pollutants are PM and gaseous pollutants. PM is a complex mixture of solid and liquid particles suspended in the atmosphere, and includes particulate matter (PM_{10}) and fine PM ($PM_{2.5}$). Industrial emission due to combustion, secondary inorganic aerosols, and secondary organic aerosols are the primary identified sources of fine PM.[15] When inhaled, PM_{10} mainly accumulates in the upper respiratory tract, such as the nasal cavity, pharynx, and larynx, while $PM_{2.5}$ accumulates in both the lower and upper respiratory tracts, and especially in the small air ducts and lung bubbles.[16] Ultrafine particles have a smaller diameter, and their impact on human health has not been well studied. In addition to directly damaging the human body, PM can adsorb other allergens, microorganisms, fungi, dust mites, and other pathogenic agents in the air, causing greater harm to the human body. Gaseous pollutants include sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and ozone (O_3). The primary sources of SO_2 in the atmosphere are sulfur compounds produced by natural processes such as burning of sulfur-containing fossil fuels and volcanic eruptions. NOx mainly comes from fossil fuel combustion, and vehicular emissions are a major source of this pollutant, while O_3 is produced by photochemical reactions. All types of gaseous pollutants can produce secondary aerosol pollution through photochemical reactions at a certain temperature and humidity, adding to the PM in the air.[17]

Air pollution and COPD: epidemiological evidence

The adverse effects of air pollution on health include acute (short-term), chronic (long-term), and

Table 1
Selected studies of the association between exposure to air pollution and COPD.

Study/year	Location	Design	Population sample	Health effects	Outcome (OR/HR,95% CI)
Schikowski et al./2005[26]	Germany (1985−1994)	Consecutive-cross sectional study	4757	prevalence	NO_2 (1.33, 95% CI,1.03−1.72, per 16 μg/m^3) PM_{10} (1.43, 95% CI,1.23−1.66, per 7 μg/m^3)
Schikowski et al./2014[23]	Europe (2008−2011)	Cohort study	6550(NOx) 3692(PM)	prevalence	No statistically significant correlation was found
Cai Y et al./2014[24]	Europe (1998−2011)	Cross-sectional study	15,279(NO_2) 10,537(PM)	prevalence	No statistically significant correlation was found
Atkinson RW et al./2015[90]	England (2003−2007)	Cohort Study	16,034	prevalence	$PM_{2.5}$ (1.05, 95% CI, 0.98−1.13, per 1.9 μg/m^3) SO_2 (1.01, 95% CI, 0.97−1.07), per 2.2 μg/m^3) NO_2 (1.06, 95% CI, 0.98−1.15, per 10.7 μg/m^3)
Liu S et al./2017[28]	China (2012−2015)	Cross-sectional study	5993	prevalence	2.416 (95% CI,1.417 to 4.118) for >35−75 μg/m^3 and 2.530 (95% CI, 1.280 to 5.001) for >75 μg/m^3 compared with the level of ≤35 μg/m^3 for $PM_{2.5}$ 2.442 (95% CI, 1.449 to 4.117) for >50−150 μg/m^3 compared with the level of ≤50 μg/m^3 for PM_{10}
Dany Doiron et al./2019[27]	UK (2006−2010)	Cross-sectional analyses	303, 887	prevalence	$PM_{2.5}$ (1.52, 95% CI 1.42−1.62, per 5 μg/m^3) PM_{10} (1.08, 95% CI 1.00−1.16, per 5 μg/m^3) NO_2 (1.12, 95% CI 1.10−1.14, per 10 μg/m^3)
Zanobetti A et al./2008[91]	USA (1985−1999)	Cohort study	1,039,000	mortality	PM_{10} (1.22, 95% CI: 1.17−1.27, per 10 μg/m^3)
Kazemiparkouhi F et al./2019[31]	USA (2000−2008)	Time-series study	22,200,000	mortality	O_3 (1.065, 95% CI 1.060−1.069, per 10 ppb)
Junfang Cai et al./2019[35]	China (2013−2015)	Time-series study	41,815	mortality	The excess risk (ER) is 8.24% (95% CI: 3.53−13.17) for per 10 μg/m^3 increase in $PM_{2.5}$

latent effects.[18] Common research methods include time series studies, cohort studies, case−control studies, cross-sectional studies, and panel studies.[19,20]

COPD prevalence

The incidence of COPD doubled after the great London fog of 1952 (as demonstrated by autopsy), suggesting that short-term exposure to ambient air pollution may adversely affect the health of people with COPD.[21] Whether chronic exposure to air pollution will lead to COPD remains unknown, with different regions reporting varying results, and sufficient confirmatory evidence remains lacking. Developed western countries have conducted large cohort studies to examine the relationship between chronic air pollution and COPD over the past decade, while only a few related studies have been conducted in China; several key studies have been summarized in Table 1. PM and NO_2 are the most studied pollutants, followed by O_3 and SO_2. The European Study of Cohorts for Air Pollution Effects (ESCAPE) is a large European cohort study evaluating the chronic health effects of air pollution and involves 13 countries with an average follow-up time of 14 years.[22] Using data from ESCAPE and land regression models, Schikowski et al[23] analyzed the correlation between the annual mean concentrations of PM and NOx and COPD prevalence during a 3-year follow-up period (2008−2011); NO_2 and PM levels showed no significant association with COPD prevalence in either the cohort or the meta-analysis. The team also analyzed some follow-up ESCAPE data from 1998 to 2011, and no consistent association between chronic bronchitis symptoms and current traffic-related air pollution was observed in adult European populations.[24] The Health Survey for England reported that living in close proximity to main roads had no adverse effect on the risk of COPD.[25] However, a 5-year continuous cross-sectional study of non-smoking women living near the major roads in Germany found that those living less than 100 m away from busy roads were 1.79 times more likely to develop COPD (95% confidence interval [CI]: 1.06−3.02).[26] Using the UK Biobank database, Doiron et al[27] performed a cross-sectional analysis among 30,887 individuals aged 40−69 years who were exposed to air pollution and found that $PM_{2.5}$, PM_{10}, and NO_2 concentrations were significantly associated with COPD prevalence (OR 1.52 [95% CI: 1.42−1.62] per 5 μg/m^3; OR 1.08 [95% CI: 1.00−1.16] per 5 μg/m^3; and OR 1.12 [95% CI: 1.10−1.14] per 10 μg/m^3, respectively), but PM_{coarse}

concentrations were not. Compared with previous studies, the main advantage of this study was its large sample size. The findings were similar to those of previous cross-sectional studies conducted in China in 2017.[28]

Air pollution exposure not only increases the risk of COPD in healthy people, but also in asthma patients, who develop asthma-COPD overlap syndrome (ACOS), which may contribute to the transition from asthma to COPD. A cohort study in Canada followed asthma patients aged 18 years and older diagnosed between 1996 and 2009 through 2014. The concentrations of $PM_{2.5}$ and O_3 were obtained from fixed monitoring sites, and Cox regression models were used to assess the association of air pollutants with the risk of ACOS. Results showed that asthma patients exposed to higher levels of air pollution were nearly three times more likely to develop ACOS.[29]

COPD mortality

Air pollution is the primary non-infectious factor that causes an increase in the COPD-related mortality rate. In 2012, 8% of global COPD deaths were attributed to air pollution, according to the statistics of the WHO (2016 air quality report). In 2015, the number of $PM_{2.5}$-related premature deaths in 161 cities in China was 652,000, accounting for 6.92% of the total deaths, of which deaths due to COPD accounted for 11.77%.[30]

Long- and short-term exposure to air pollution can affect COPD mortality. However, most studies have only focused on evaluating the short-term effects of exposure to air pollution, and most of them reported short-term effects of air pollution on respiratory mortality, rather than on COPD mortality.

The increased concentrations of various air pollutants, such as O_3,[31,32] NO_2,[33] SO_2,[34] $PM_{2.5}$,[32,35,36] and PM_{coarse},[37] can increase the risk of mortality in patients with COPD. Moreover, the impact of air pollutants on the mortality rate of COPD patients is higher than that observed in the population as a whole. Xu et al[38] conducted an impact assessment on the relationship between daily $PM_{2.5}$ changes in urban areas and suburbs of Beijing and the mortality rate from specific causes, and showed that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 0.17% (95% CI: 0.05−0.29) increase in non-accidental mortality, but a 0.96% (95% CI: 0.35−1.57) increase in COPD mortality. Wildfire smoke contains a variety of substances that are harmful to human health, including $PM_{2.5}$, CO, NOx, and benzene, which can cause varying levels of air pollution.[39] A previous United States study reported a 9.0% (95% CI: 0−18.0) increase in respiratory disease mortality on wildfire smoke days, and a 14.0% (95% CI: 2.0−26.0) increase in COPD mortality.[40] In addition, different PM sizes have different effects on COPD mortality. A previous survey conducted in Shanghai, China, found that daily COPD mortality was significantly associated with particle number concentrations for particles <0.5 μm, and that the magnitude of associations increased with decreasing particle size.[41] In recent years, O_3 has become the main environmental pollutant affecting the mortality rate of patients with COPD. In 2015, 8% of the total deaths worldwide occurred due to exposure to O_3, and 254,000 of these deaths were due to COPD.[1] A large prospective cohort study in the United States showed that long-term exposure to O_3 significantly increased the risk of death from COPD, with the risk of death increasing by 1.09 (95% CI: 1.03−1.15) when the annual mean concentration of O_3 increased by 10 ppb.[42]

Lung function and symptoms

Children[43−46] and teenagers[47,48] usually experience developmental delays or reductions in lung function when exposed to air pollution. However, few studies have investigated the effects of air pollution on lung function in adults with established lung diseases, such as COPD. Whether air pollution can cause a decline in lung function in patients with COPD remains unclear.

Kariisa et al[49] studied 1218 patients with COPD over a 5-year period, and reported that long-term exposure to $PM_{2.5}$ could aggravate the symptoms of patients and significantly reduce their lung function. A panel study from China investigated the effects of short-term exposure to outdoor PM on lung function in COPD patients and found that forced vital capacity (FVC) decreased by 3.3% (95% CI: −5.8 to −0.8) and 2.1% (95% CI: 3.9−0.3) when $PM_{2.5}$ and PM_{10} concentrations increased by 111.0 $\mu g/m^3$ and 112.0 $\mu g/m^3$, respectively. Similar results were found for forced expiratory volume per second (FEV_1).[50] However, a panel study in London investigated the short-term effects of exposure to outdoor air pollutants on lung function and respiratory symptoms in 94 COPD patients. Their findings suggested that the increase in pollution levels was not associated with either FVC or FEV_1, and exposure to PM_{10} was associated with dyspnea.[51] Similarly, a 3-month panel study found that short-term exposure to PM_{10} increased the risk of nocturnal chest discomfort in COPD patients, while no correlation was found between NO_2, SO_2, and CO concentrations and respiratory symptoms or lung function.[52]

Hospital admission

Exposure to air pollutants is related to an increase in hospitalization rates for COPD patients, and previous studies conducted in different countries and regions showed consistent findings. $PM_{2.5}$ and PM_{10} are the main pollutants globally and are associated with an increased risk of hospitalization in COPD patients. For a 10 μg/m^3 increase in $PM_{2.5}$ concentration, the risk of hospitalization increases by 1.61% in COPD patients in the United States,[53] by 0.82% in Beijing,[54] and by 1.72% and 6.87% in cool and warm weather, respectively, in Taiwan, China.[55] A recent systematic review and meta-analysis reported a temporal correlation between PM_{10} and COPD hospitalizations. After short-term exposure to a 10 μg/m^3 increase in PM_{10} concentration, the COPD hospital admission enrollment increased by 1% in China, 2% in the United States, and 1% in the European Union.[56] In addition to PM, a variety of gaseous pollutants are also associated with the COPD-related hospital admissions. A prospective study conducted by the European Apache study (air pollution and health, a European approach) found that the levels of SO_2, O_3, NO_2, black smoke, and total suspended particulates were all associated with the daily number of COPD-related hospitalizations in six European cities, and there was a lag effect of 1–3 days.[57]

A study from Iran also showed that O_3, NO_2, and SO_2 had a significant impact on the number of hospital admissions for COPD. The number of COPD-related hospital admissions increased by 2.0% (95% CI: 0.8–3.1), 0.7% (95% CI: 0.1–1.8), and 0.5% (95% CI: 0–1.0) per 10 μg/m^3 increase in O_3, NO_2, and SO_2 concentrations, respectively.[58] Epidemiological studies evaluating the relationship between CO levels and COPD admissions have yielded different results. A retrospective study of 162,338 patients admitted to hospitals for COPD exacerbation from 2004 to 2013 in Spain found that higher CO levels was associated with increased admission of COPD patients.[59] However, some epidemiological studies found that low concentrations of CO may have a protective effect under certain conditions. A time series study in Hong Kong, China, found that short-term exposure to CO was associated with a lower risk of COPD hospitalizations, which showing the protective effect of CO exposure on COPD admissions, and after adjusting for NO_2 and $PM_{2.5}$ levels, the negative correlation between CO levels and COPD became stronger.[60] A similar study conducted in Shanghai also found that short-term exposure to CO at low ambient concentrations may be associated with a reduced risk of COPD-related hospitalization.[61] However, further studies are needed to confirm the direct clinical effect of CO exposure in patients with COPD.

Role of climatic factors on the effect of air pollution and COPD

The impact of air pollutants on COPD varies with climatic factors such as air temperature, air pressure, wind speed, and relative humidity. Air temperature plays an important synergistic role in the pathogenic effects of pollutants on COPD, but the differences between the effects of low and high temperature remain controversial. Qiu et al found that low temperature significantly increased the effect of $PM_{2.5}$, PM_{10}, and SO_2 levels on the COPD hospitalization rate.[62] A study conducted in Hong Kong, China also showed that SO_2, NO_2, and O_3 had a greater effect on COPD admissions in the cold season than during the warm season.[63] Moreover, the effect of air pollutants on the COPD hospitalization rate is greater in warm seasons than in cold seasons. This could be because higher temperatures increase the concentration of O_3 and other pollutants in the air. Studies in Taiwan, China have shown that $PM_{2.5}$ and O_3 have a more significant effect on the COPD hospitalization rate among older patients with COPD on days with high temperature and large pressure differences.[64] Gao et al[54] found that compared with the cold season (November to March), the impact of air pollutants on COPD admission was higher in the warm season (August to October). Relative humidity can also have an effect on COPD admission. In one study, for every 1% increase in relative humidity, the relative risk of COPD hospitalization increased by 1.070 (95% CI: 1.054–1.086).[65] In addition, there was a significant interaction effect between temperature and humidity in patients with COPD.

Effect of air pollution on the pathophysiology of COPD and the possible underlying mechanism

The pathogenic effect of air pollution on COPD remains unclear. However, current research focuses on factors such as oxidative stress and inflammatory damage, as well as DNA damage.

Inflammatory damage

COPD is characterized by chronic inflammation of the airway and lung parenchyma; accumulation of

inflammatory cells, including neutrophils, activated macrophages, and lymphocytes; and increase in inflammatory factors such as interleukin-6 (IL-6), IL-8, and tumor necrosis factor-α (TNF-α). A previous study showed that acute exposure to $PM_{2.5}$ induced inflammatory cell infiltration and hyperemia in the lung tissues and increased the number of inflammatory cells in the bronchoalveolar lavage fluid.[66] The inhaled $PM_{2.5}$ is engulfed by lung macrophages, stimulating the release of inflammatory factors. However, these inflammatory factors accumulate in the damaged area and stimulate inflammatory cells to release more inflammatory factors, thus leading to a vicious cycle that damages lung endothelial cells and further aggravates lung injury.[67]

Oxidative stress damage

Free radicals produced by oxidative stress, especially oxygen free radicals, play an important role in the pathogenesis of COPD. PM has the ability to generate oxygen free radicals, which — after inhalation — can stimulate cells to produce a large number of reactive oxygen species (ROS). The large amounts of metal components and organic matter carried by PM can also induce ROS production in cells.[68,69] Such ROS-induced oxidative damage to lung cells may be the primary cause of damage due to PM exposure.

Genetic damage

Exposure to air pollution can induce genotoxicity and cause chromosome damage in cells, and there is a dose-dependent relationship within a certain range of concentrations. Exposure to $PM_{2.5}$ can cause damage to lung epithelial cells and alveolar macrophages and increase the ROS production induced by oxidative stress, which leads to DNA damage and changes in gene expression.[70] Epigenetic changes in gene expression may occur through DNA modifications, including DNA methylation, histone modification, and non-coding RNA modification, without any change in the DNA sequence. Air pollutants can also play a role in the pathogenesis of COPD by altering epigenetic modifications. Exhaled nitric oxide (FeNO) is considered a sensitive marker of airway inflammation, and can reflect the state of airway inflammation in COPD patients. A panel study conducted in Shanghai examined the relationship between $PM_{2.5}$ and DNA methylation in COPD, and found that $PM_{2.5}$ may regulate the production of FeNO by changing methylation markers in the *NOS2A* promoter region and further aggravating inflammation in the airway.[71] Song et al[72] found that $PM_{2.5}$ can reduce the expression of *miR-331* through the ROS/PI3K/AKT pathway, resulting in increased expression of IKK-β and sustained activation of NF-κB in human airway epithelial cells.

Protective measures

Policy intervention

Government control plays a decisive role in improving air pollution levels. The United States, for example, previously had severe air pollution. Hence, the United States government developed a series of air pollution prevention and control measures, such as the Clean Air Act Amendments in 1990 and the NOx State Implementation Plans Call in 2002. After 1990, the concentrations of $PM_{2.5}$ and O_3 in the United States decreased significantly. Air quality improvements have significantly decreased the COPD mortality burden.[73] In the last decade, which saw rapid industrial development and urbanization, China experienced the most serious smog episodes. The government has promulgated a series of powerful prevention and control measures to control air pollution in China. In 2013, the state council formulated the Air Pollution Action Prevention and Control Plan (2013–2017).[12] In 2018, the state council issued the Three-Year Action Plan to Win the Blue Sky Defense War, which was used as a guide for the next stage of air pollution prevention and control.[74] Provincial and municipal governments have also issued corresponding policies, such as the Beijing City Master Plan (2016–2035).[75] Since 2013, the air quality in most Chinese cities has improved, and $PM_{2.5}$, PM_{10}, and SO_2 concentrations have decreased significantly.[76] Moreover, these policies have had some health benefits, and emission controls reduced the $PM_{2.5}$ mortality by 88.7%. Liang et al found that compared with the levels in 2013, the SO_2 and $PM_{2.5}$ levels in 2017 were 68% and 33% lower, respectively. In addition, there was a decreasing trend in the number of cases of acute exacerbations of COPD advanced due to $PM_{2.5}$ exposure.[77] Although air pollution has decreased, air quality has not yet reached the standards set by the WHO. PM concentrations are still high, the O_3 concentration has increased dramatically, and the NO_2 concentration remains the same. Therefore, the government should formulate strong air pollution prevention and control measures to reduce emission from enterprise pollution sources, in order to achieve an overall improvement in air pollution levels.

Group intervention

Indoor air pollution is mainly caused by the burning of solid fuel for cooking and is associated with a variety of respiratory diseases. Approximately one-third of the world's population uses biofuels such as wood or charcoal for cooking or heating, especially in rural areas in LMICs. Studies in many countries have linked biofuel exposure to an increased risk of COPD.[78–81] A cohort study conducted in China followed up 277,838 Chinese individuals who had never smoked and had not developed major chronic diseases in the last 9 years to examine the association of solid fuel use with the risks of acute and chronic respiratory diseases. Results showed that compared with clean fuel users, solid fuel users had an adjusted hazard ratio of 1.10 (95% CI: 1.03–1.18) for COPD. The use of clean fuel and ventilation equipment can reduce the risk of respiratory diseases.[82] Moreover, the use of clean fuel or ventilation kitchenware can effectively reduce the risk of respiratory diseases.

Zhou et al[83] conducted a non-randomized intervention to assess the long-term impact of alternative biomass fuel use and improved kitchen ventilation on lung function in patients with COPD. Results showed that the use of biogas instead of biomass cooking and improved kitchen ventilation were related to the reduction in the risk of poor FEV_1 and COPD. Moreover, a dose–response relationship was observed: the longer was the duration of improved cooking fuel usage and kitchen ventilation, the greater was the impact on reducing the decline in lung function. Use of improved cookstoves also contributed to the reduction in biofuel pollution and the incidence of respiratory symptoms such as cough, expectoration, and wheezing, and the risk of COPD.[84,85] Therefore, improved cookstoves, ventilated kitchenware, improved kitchen ventilation, and clean fuel must be used to reduce the risk of COPD.

Individual intervention

The time and intensity of outdoor activities should be reduced as much as possible when smog levels are high, and masks that are effective against $PM_{2.5}$ particles should be worn to reduce the exposure to $PM_{2.5}$ outdoors. Wearing of personal protective equipment such as the N95 mask or an equivalent may help avoid the harmful effects of ambient air pollutants.[86] So far, research on the protective effects of masks has mainly focused on healthy adults or people of specific occupations, and the number of studies conducted in sensitive individuals such as COPD patients are limited. Sundblad et al[87] conducted a study involving 36 healthy volunteers. Results showed that compared with the group without masks, the volunteers wearing masks had lower levels of systemic inflammatory factors and relatively higher lung function indicators. The study demonstrated that the use of masks can protect the respiratory system. For indoor air pollution, indoor air purifiers can reduce the level of $PM_{2.5}$ in the air. However, whether this can really improve cardiopulmonary function remains unclear.[88,89] Hence, a more effective experimental study is needed to evaluate the actual protective effect of air purifiers and masks in COPD patients.

Medical intervention

In addition to the above interventions, some medical interventions such as nebulization therapies may help to promote the elimination of PM from the lower respiratory tract in COPD patients and alleviate the adverse effects of air pollution. But so far, there are no recommended guidelines for reducing air pollutants associated with atomizer inhalers.

Conclusion

There is a great deal of evidence to support the notion that air pollution is associated with respiratory diseases, including COPD. Epidemiological and clinical studies have confirmed the link between air pollution and COPD. Both short- and long-term exposure to air pollution has a negative effect on the occurrence and development of COPD. Air pollution, one of the most important risk factors in the prevention and treatment of COPD, is modifiable and should attract more attention. In the future, comprehensive clinical studies and in-depth research on basic mechanisms are needed, which will not only help improve care for individual patients, but also help persuade decision-makers to promote public health policies to eliminate air pollution globally.

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Declaration of Competing Interest

None.

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