Review Article

Air pollution and COPD in China

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Abstract: Recently, many researchers paid more attentions to the association between air pollution and chronic obstructive pulmonary disease (COPD). Haze, a severe form of outdoor air pollution, affected most parts of northern and eastern China in the past winter. In China, studies have been performed to evaluate the impact of outdoor air pollution and biomass smoke exposure on COPD; and most studies have focused on the role of air pollution in acutely triggering symptoms and exacerbations. Few studies have examined the role of air pollution in inducing pathophysiological changes that characterise COPD. Evidence showed that outdoor air pollution affects lung function in both children and adults and triggers exacerbations of COPD symptoms. Hence outdoor air pollution may be considered a risk factor for COPD mortality. However, evidence to date has been suggestive (not conclusive) that chronic exposure to outdoor air pollution increases the prevalence and incidence of COPD. Cross-sectional studies showed biomass smoke exposure is a risk factor for COPD. A long-term retrospective study and a long-term prospective cohort study showed that biomass smoke exposure reductions were associated with a reduced decline in forced expiratory volume in 1 second (FEV_1) and with a decreased risk of COPD. To fully understand the effect of air pollution on COPD, we recommend future studies with longer follow-up periods, more standardized definitions of COPD and more refined and source-specific exposure assessments.

Keywords: Biomass smoke; air pollution; haze; chronic obstructive pulmonary disease (COPD)

Submitted Jun 29, 2014. Accepted for publication Dec 24, 2014.
doi: 10.3978/j.issn.2072-1439.2014.12.47
View this article at: http://dx.doi.org/10.3978/j.issn.2072-1439.2014.12.47

Introduction

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death globally presently, and it is predicted to become the third leading cause by 2030 (1), making this disease one of the major health challenges in the future (2). The best way to reduce the prevalence of COPD is to control its risk factors; and it is general accepted that smoking is an important risk factor for COPD, although there are other aetiologies of COPD (3). Recently, air pollution as a COPD risk factor has been increasingly recognized.

The Chinese haze is a phenomenon caused by the presence of fine particles ($PM_{2.5}$) at high concentration in the atmosphere, resulting in diminished atmospheric visibility. A few regions were seriously affected by the haze such as the Hua Bei Plain in Northern China, the Yangtze River Delta and the Pearl River Delta (4). In these regions, Air Pollution Index reached above 500, the maximum of the scale. Less than 1% of the 500 largest cities in the People's Republic of China can meet the air quality guidelines recommended by the World Health Organization. Seven of these cities were ranked among the ten most polluted cities in the world (5). So far, studies of the health effects of haze have mainly focused on $PM_{2.5}$, as these fine particles can penetrate deeply into the lung, irritate and corrode the alveolar wall, and consequently impair lung function, clinically leading to cough, wheeze, respiratory disorders and other symptoms. $PM_{2.5}$ exposure, hence, increases the risks of COPD, emphysema and other respiratory diseases (6).

Biomass smoke is the main source of indoor air pollution in the developing world. Biomass fuel is the domestic energy source for almost 3 billion people (7), and the

resulting smoke contributes largely to the global burden of mortality, accounting for about 1.6 million of the 59 million deaths annually (8,9), and accounting for 2.7% of disability adjusted life years (DALYs) lost (8,9). Such air pollution has been reported to be associated with an increased risk of COPD, respiratory symptoms, and impaired lung function in a number of studies (10-14).

In this review we will summarize the effect of outdoor air pollution and biomass smoke exposure on long-term development of the chronic obstructive pathology and the short-term superimposed acute exacerbations. In this context, we also discuss the interaction of air pollution exposure with cigarette smoking in China.

Haze and development of COPD

Haze and lung function

In China, the effects of $PM_{2.5}$ or haze on human lung function have been investigated in both children and adults. The general purpose of the studies in children was to examine the effect of haze on lung function development, while that of the studies in adults was to understand the damage on the magnitude of lung function. Wang *et al.* (15) tested eight lung function parameters of 216 healthy students between 7 and 11 years old from two primary schools in Beijing in October 2008. At the same time, levels of $PM_{2.5}$ in the schoolyards were monitored; and PM_{10} data were also collected from a nearby monitoring site. They found that $PM_{2.5}$ and PM_{10} had short-term adverse health effects on children's lung function. The effects on parameters reflecting large airway characteristics such as FVC and forced expiratory volume in 1 second (FEV_1) were even more obvious. Roy *et al.* (16) had measured lung function of 3,273 children (6-12 years of age) from eight schools in four Chinese cities twice a year for up to 3 years. Meanwhile, $PM_{2.5}$ and PM_{10} of each schoolyard were measured. They found that an increase of 10 $\mu g/m^3$ of $PM_{2.5}$ was associated with decreases of 2.7 mL FEV_1 [95% confidence interval (CI): −3.5 to −2.0], 3.5 mL FVC (95% CI: −4.3 to −2.7), 1.4 mL/year FEV_1 growth (95% CI: −1.8 to −0.9), and 1.5 mL/year FVC growth (95% CI: −2.0 to −1.0). Wang *et al.* (17) monitored the ambient levels of $PM_{2.5}$ and sulfur dioxide (SO_2) in urban and suburban areas and performed pulmonary function testing on 1,075 adults 35 to 60 years of age who had never smoked and did not use coal stoves for cooking or heating. The mean concentration of SO_2 in the urban areas (213 mg/m^3) was twice as high as that in suburban areas (103 mg/m^3). Mean $PM_{2.5}$ levels were high in both urban (143 mg/m^3) and suburban (139 mg/m^3) areas. They found that estimated differences in FEV_1 between the urban and suburban areas were 199 mL (SE =50 mL) for men and 87 mL (SE =30 mL) for women, both statistically significant. When the men and women were pooled, the estimated difference in FEV_1 was 126 mL (SE =27 mL). Similar trends were observed for FVC and FEV_1/FVC ratio. Wu *et al.* (18) have investigated the effects of $PM_{2.5}$ on lung function in a panel of 21 healthy university students from the healthy volunteer natural relocation study in the context of suburban/urban air pollution in Beijing, China. Each study subject used an electronic diary meter to record peak expiratory flow (PEF) and FEV_1 twice a day for 6 months in three periods before and after relocating from a suburban area to an urban area with changing ambient $PM_{2.5}$ in Beijing. They found that $PM_{2.5}$ was associated with reductions in evening PEF and morning/evening FEV_1. Gao *et al.* (19) also reported that long-term higher levels of airborne fine particulate matter exposure impaired pulmonary function. Liu *et al.* (20) found that exposure to outdoor $PM_{2.5}$ was significantly associated with PEF among college students in winter, but not in autumn. We have reported a 4-year study (21) in China, in which the data suggested that subjects with an improvement on outdoor air pollution had less decline of FEV_1 than those living in a relatively worse environment. To assess the effect of reduction in air pollution levels on lung function, Mu *et al.* (22) collected data before, during, and after the Olympics, respectively. Linear mixed-effect models and generalized estimating equation models were used to compare measurements of PEF, across three time points. They found that the mean values of PEF were 346.0, 399.3, and 364.1 L/min over the three study periods, respectively. PEF levels increased in 78% of the participants when comparing during- with pre-Olympics time points, while PEF levels decreased in 80% of participants from the during- to the post-Olympic periods. These studies have demonstrated that $PM_{2.5}$ and other components or the air pollution mixture (haze) can retard the development of children's lung function and impair adults' lung function.

Haze and prevalence of COPD

The relationship between COPD and air pollution has been assessed in the developed countries (23-26). In China, most of the studies focused on the effect of haze on the respiratory symptoms and diseases. Few studies have

examined the long-term effect of haze on the prevalence of COPD. Lam *et al.* (27) have reported that COPD was associated with high exposure to dust or gas/fume [adjusted odds ratio (OR): 1.41; 95% CI: 1.06-1.87]. Ma *et al.* (28) conducted an epidemiological study of the effects of air pollution on the occurrence of respiratory diseases in Tianjin and found that air pollution was a risk factor for emphysema and chronic bronchitis. A 100 μg/m^3 increase of TSP was associated with an increased odds ratio for emphysema (1.29) and chronic bronchitis (1.59), respectively. Jin *et al.* (29) conducted a cross-sectional epidemiological study of the effects of air pollution on the occurrence of respiratory symptoms and diseases in adults in Taiyuan city of Shanxi province. They reported that with a unit increase in logarithm of the concentration SO_2, TSP, PM_{10}, $PM_{2.5}$, the odds ratio of COPD increased to 1.31, 1.53, 1.51, and 1.68, respectively. Another study by Jin *et al.* (30) reported that the occurrence risk of respiratory symptoms and COPD of adults in a heavily polluted area was 1.7 and 1.5 times that in a relatively clean area respectively; and FVC and FEF_{50} of pupils decreased by 194 and 172 mL, respectively, with increasing of every concentration unit of the Ln(PM_{10}). FVC and FEF_{50} of pupils decreased by 69 and 119 mL, respectively, with the increase of every unit of the Ln(SO_2). However, there were several issues of these studies. Firstly, most studies used only questionnaire-based definitions (e.g., chronic symptoms or "doctor-diagnosed COPD', etc.) of COPD as the diagnostic criteria. Secondly, pollutant data were obtained from one or several monitoring stations with limited geographic coverage, assuming these data to represent the average exposure for people living in a large area. Thirdly, there is no long-term study to assess the effect of haze on COPD. So, evidence of chronic effects of air pollution on the prevalence and incidence of COPD among adults was suggestive but not conclusive.

Haze and acute exacerbation of COPD (AECOPD)

To assess the association between haze and AECOPD is to evaluate the role of air pollution in triggering symptoms and exacerbations, i.e. the acute effects of short-term exposure to air pollution. As in a review article by Sunyer *et al.* (31), the role of air pollution in the acute exacerbation of COPD has been generally recognized for many years, and has been confirmed in recent studies (32,33). Some research from China also supports this conclusion (34-37). However, most of the studies have focused on respiratory diseases, not just COPD. Zhang *et al.* (37) reported that a 10 μg/m^3 increase in nitrogen dioxide (NO_2) concentration was associated with a 1.94 (95 % CI: 0.50-3.40) increase in excessive risk of respiratory illnesses at lag0, especially among COPD. Haze (at lag1) and air pollution (for NO_2 at lag5 and for SO_2 at lag3) both presented more drastic effects on the 19 to 64 years old and in the females. NO_2 was the sole pollutant with the largest risk of hospital admissions for total and respiratory diseases in single- and multi-pollutant models. A study (30) conducted in Lanzhou showed that PM_{10} and NO_2 were significantly associated with female COPD hospitalizations; that PM_{10} was associated with male COPD hospitalizations; and that NO_2 was associated with hospitalizations of COPD patients aged >65 years. A study by Liu *et al.* (35) found that the onset of AECOPD was associated with air quality and temperature, especially in severe COPD patient. A study (36) by Ko *et al.* in Hong Kong showed that air pollution was associated with a significantly increased risk of hospitalizations for patients with acute exacerbations of COPD. A time series study conducted by Qiu *et al.* (38) examined whether the effects of air pollution on emergency COPD hospital admissions in Hong Kong varied across seasons and relative humidity levels. They observed an increase in the detrimental effects of air pollution in the cool season and on low humidity days. On the cool and dry days, a 10 μg/m^3 increment of lag3 exposure was associated with an increase in emergency COPD admissions by 1.76% (95% CI: 1.19-2.34%), 3.43% (95 % CI: 2.80-4.07%), and 1.99% (95% CI: 0.90-3.09%) for NO_2, ozone (O_3), and SO_2, respectively, all of which were statistically significantly higher than those on the other days. Chiu *et al.* (39) assessed the possible associations of Asian dust storm events with hospital admissions for COPD in Taipei, Taiwan, during the period 1996-2001. They identified 54 dust storm episodes that were classified as index days. Daily COPD admissions on the index days were compared with admissions on the non-index days. They also selected 2 non-index days for each index day, 7 days before the index days and 7 days after the index days. They found that the effects of dust storms on hospital admissions for COPD were prominent 3 days after the event [relative risk (RR): 1.057; 95% CI: 0.982-1.138]. In another study, Tsai *et al.* (40) found that COPD admissions were significantly and positively associated with higher $PM_{2.5}$ levels during both warm days (>23 ℃) and cool days (<23 ℃), with an interquartile range increase of 12% (95% CI: 8-16%) and 3% (95% CI: 0-7%) in COPD admissions, respectively. Furthermore, two other studies (41,42) also

found that higher levels of ambient air pollutants increased the risk of hospital admissions for COPD in Taiwan.

Haze and the mortality of COPD

Both short-term and long-term exposure to air pollution can influence the mortality of COPD. However, most of the studies focused on the short-term exposure effect; and. most of which reported the short-term effects of air pollution on respiratory mortality, but not specifically on COPD mortality. Zhao *et al.* (43) have conducted quantitative assessment of the impact of $PM_{2.5}$ on daily disease mortality of residents in urban area in Xi'an. They found that when $PM_{2.5}$ concentration increased by 100 μg/m^3, the mortality of COPD increased by 7.25%. Ju *et al.* (44) found that there was an association between COPD mortality and PM_{10} concentration and CO concentration, respectively, in Suzhou. In their study, a 10 μg/m^3 increase in PM_{10} and CO concentration would lead to the RR of mortality of 1.38 (95% CI: 1.069-1.702) and 1.042 (95% CI: 1.029-1.056), respectively. Chang *et al.* (45) reported that a 100 μg/m^3 increase in SO_2 concentration was associated with a19.22% increase in COPD mortality. A study (46) in Shanghai reported that an increase in 10 μg/m^3 of PM_{10}, SO_2 and NO_2 corresponded to 0.5% (95% CI: 0.1-1.1%), 3.5% (95% CI: 1.5-5.4%) and 3.2% (95% CI: 0.9-5.6%) increase in COPD mortality. A meta-analysis (47) showed that Air pollution (PM_{10}, SO_2, and NO_2) was found to be associated with increased risk of COPD mortality. Using the random-effects model, an increase of 10 μg/m^3 of 2-day moving average concentrations of PM_{10}, SO_2 and NO_2 corresponded to a 0.78% (95% CI: 0.13-1.42), 1.30% (95% CI: 0.61-1.99), and 1.78% (95% CI: 1.10-2.46) increase of COPD mortality, respectively. Chen *et al.* (48) reported that population-wide COPD mortality decreased when air quality improved and increased with increasing air pollution in Hong Kong. There have been no studies, to our best knowledge, that evaluated the effect of long-term exposure to air pollution on mortality of COPD.

Biomass smoke exposure and COPD

Biomass smoke has been reported to be associated with an increased risk of COPD, respiratory symptoms, and impaired lung function (10-14), mainly in cross-sectional and case-control studies (13,14). Among the two studies that evaluated the effect of long-term biomass smoke exposure on COPD (10,11), one is a retrospective cohort study (11) and the other is a prospective study (10).

To assess the effect of reduction of biomass smoke exposure on COPD, Zhou *et al.* (10) conducted a 9-year prospective cohort study among 996 eligible participants aged at least 40 years from November 1, 2002, through November 30, 2011, in 12 villages in southern China. The interventions involved improving kitchen ventilation and using cleaner fuels (i.e., biogas) instead of biomass for cooking .Indoor air pollutants in a randomly selected subset of the participants' homes were measured. This study found that the use of clean fuels and improved ventilation were associated with a reduced decline in FEV_1: the decline in FEV_1 was reduced by 12 mL/year (95% CI: 4-20 mL/year) and 13 mL/year (95% CI: 4-23 mL/year) in those who used clean fuels and those with improved ventilation, respectively, compared to those who used neither intervention, after adjustment for confounders. The combined improvements of use of clean fuels and improved ventilation had the largest reduction in FEV_1 decline: 16 mL/year (95% CI: 9-23 mL/year). The study further found that the longer the duration of improved fuel use and ventilation, the greater the benefits in slowing the decline of FEV_1. The reduction in the risk of COPD was unequivocal after the fuel and ventilation improvements, with an odds ratio of 0.28 (95% CI: 0.11-0.73) for both improvements.

Chapman *et al.* (11) performed a retrospective cohort study (follow-up 1976-1992) to test whether improvement in household coal stoves affected the incidence of COPD in Xuanwei County, China. They found that installation of a chimney was associated with distinct reduction in the incidence of COPD. Compared with people who did not have chimneys, the Cox-modelled RR was 0.58 (95% CI: 0.49-0.70; P<0.001) in men and 0.75 (95% CI: 0.62-0.92; P=0.005) in women.

Liu *et al.* (12) measured concentrations of carbon monoxide, PM_{10}, SO_2 and NO_2 in the kitchen during biomass fuel combustion to assess the effect of biomass smoke exposure on COPD. The use of biomass fuel was higher in rural Yunyan than in urban Liwang (88.1% *vs.* 0.7%). They found that concentrations of carbon monoxide, PM_{10} in the kitchen during biomass fuel combustion were significantly higher than those during LPG combustion. Furthermore, this study found that the prevalence of COPD in rural Yunyan was significantly higher than in urban Liwan (12.0% *vs.* 7.4% for whole population, and 7.2% *vs.* 2.5% for subpopulation of non-smoking women, respectively). Univariate and multivariate analysis showed a significant association between COPD and exposure to

biomass fuel for cooking.

In a nested case-control study using data collected in a large community survey (n=29,319) conducted between October 2000 and March 2001 in Nanjing, China, Xu *et al.* (13) compared exposure to indoor air pollutants from cooking and heating in patients diagnosed with COPD (n=1,743) and in non-COPD controls matched for age, gender and residence (n=1,743). They found that there were no significant associations between COPD and domestic fuels used, kitchen ventilation. However, as discussed in the article, the study has some limitations: first, spirometry tests were not examined, and hence there may have been misclassification bias in case and control selection; second, indoor air pollutant concentrations were not measured. To our best knowledge, there have been no studies of the effect of biomass smoke exposure on AECOPD, and the mortality of COPD in China.

The interaction of haze with cigarette smoking in relation to COPD

Some studies (49,50) have examined whether there is an interaction between haze and cigarette smoking in relation to COPD. In an extended follow-up of the Harvard Six Cities study (49), using 20 years of survival follow-up of a random sample of adults living in six cities in the East and Midwest USA, the authors found a positive but not statistically significant risk of death due to COPD. In former smokers, a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a RR of 1.64 (95% CI: 0.92-2.93), in current smokers of 1.10 (95% CI: 0.74-1.62) and in never-smokers of 0.85 (95% CI: 0.36-2.02). However, another study (50) reported that mortality due to COPD was negatively associated with $PM_{2.5}$ concentrations (RR: 0.84; 95% CI: 0.77-0.93), especially in current and former smokers, but the association was not significant in never smokers (RR: 0.96; 95% CI: 0.73-1.24). In China, Lam *et al.* (27) reported that they did not find evidence of an additive or multiplicative interaction between air pollution exposure and smoking. In a subgroup analyses comparing the during- to pre-Olympic time points, Mu *et al.* (22) found a larger percentage change in PEF (+17%) among female, younger and non-smoking participants than among male, elderly and smoking participants (+12%). Xiao *et al.* (51) have evaluated the effect of air pollution on chronic bronchitis in three polluted areas and found that there was an interaction between air pollution and cigarette smoking (for example the RR of chronic bronchitis for cigarette smoke in mild pollution area were 7.96, while the RR of chronic bronchitis for cigarette non-smoke in mild pollution area were 3.04). Hu *et al.* (52) conducted a meta-analysis to assess the effect of biomass smoke exposure on COPD and found that there is an interaction between cigarette and biomass smoke (compared with biomass smoke unexposed group, OR: 4.39; 95% CI: 3.38-5.70 for biomass and cigarette smoke exposure group, and OR: 2.55; 95% CI: 3.38-5.70 for biomass smoke exposure without cigarette smoke exposure).

Summary and recommendations

Among studies of air pollution and COPD, the vast majority have focused on the role of outdoor air pollution in triggering symptoms and exacerbations of COPD. In contrast, few studies have examined the effect of long-term exposure on COPD. General limitations of the studies include the following (53). First, most studies used only questionnaire-based definitions (e.g., chronic symptoms or "doctor-diagnosed COPD" etc.) of COPD as the diagnostic criteria. These definitions lack standardisation and, most importantly, the concepts and terms used both by doctors and patients have changed over time (e.g., "chronic bronchitis", "emphysema", "chronic lung disease", etc.). Secondly, in almost all the Chinese studies, measurement error was listed to be one of the study limitations (54). Namely, data from one or several monitoring stations with limited geographic coverage were used to represent the average exposure for people living in a large area. Thirdly, there is no long-term study to assess the effect of haze on COPD. Despite these limitations, the studies conducted to date provide evidence that air pollution affects lung function development during the childhood, damages lung function in the adulthood, exacerbates symptoms in COPD patients, and is a risk factor for the mortality of COPD. In contrast, there is weaker evidence concerning the chronic effect of outdoor air pollution on the prevalence and incidence of COPD.

A number of epidemiologic studies showed that biomass smoke exposure is a risk factor for COPD. A few long-term prospective and retrospective cohort studies have proved that the reducing exposure of biomass smoke resulted in slowing of FEV_1 decline. There have been no studies of the short-term effect of biomass smoke exposure on AECOPD and the mortality of COPD. To fully assess the role of air pollution in the development of COPD, larger studies with longer follow-up periods are needed. These studies need to use improved or more standardized definitions of COPD and more refined and source-specific exposure assessments (53).

Acknowledgements

Funding: Supported by Chinese Central Government Key Research Projects of the 10th National 5-year Development Plan Grants 2001BA703B03(A) (P.R.), The National Key Technology R&D Program of the 12th National Five-year Development Plan 2012BAI05B01 (P.R.).
Disclosure: The authors declare no conflict of interest.

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Cite this article as: Hu G, Zhong N, Ran P. Air pollution and COPD in China. J Thorac Dis 2015;7(1):59-66. doi: 10.3978/j.issn.2072-1439.2014.12.47