Session 5 Cancer, acute cardiovascular effects and COPD

Association between chronic obstructive pulmonary disease (COPD) and indoor air pollution: a review of literature.

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ABSTRACT

COPD is a chronic respiratory disorder responsible for a major burden to the society worldwide. Although the majority of COPD occurs in current or former smokers, a not negligible proportion of the disease also occurs in persons who have never smoked. Available data in the literature indicate that indoor pollution exposure largely affects respiratory health worldwide. Conservative estimates show that between 1.5 million and 2 million deaths per year could be attributed to indoor air pollution, with a significant proportion of deaths due to COPD. In this review of scientific literature, we will describe relevant findings on the association of non-smoking related COPD with the exposure to more common indoor air pollutants, in adults. **Results**: Most of the findings relate to the association of COPD with passive smoke and, in developing countries, biomass combustion exposure. Both these exposures prove to be risk factors for non-smoking related COPD. Mold/dampness exposure results associated to symptoms/signs, who may be related to the presence of COPD or its development. **Conclusion**: In spite of an increased COPD prevalence (predicted to further increase in the next years), and the evidence that other risk factors than smoking may be associated to COPD development, we found relatively few studies that assessed the association between COPD and common indoor air pollution in adult general population. It would be important to improve awareness on adverse health effects possibly associated with biomass combustion-related air pollution in developed countries among others because of the increasing interest for wood and other biomasses as potential alternative energy sources.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD)

Currently, approximate estimates indicate COPD as the fifth leading cause of global morbidity (1). In 2010 the disease is expected to rank as number three (2, 3).

According to World Health Organization (WHO) estimates, 80 million people have moderate to severe COPD. More than 3 million people died of COPD in 2005, which corresponds to 5% of all deaths globally. Total deaths from COPD are projected to increase by more than 30% in the next 10 years, unless urgent action is taken to reduce the underlying risk factors. WHO predicts that COPD will become the third leading cause of death worldwide by 2020 (1).

Several different definitions have been used for COPD. Historically, it has been defined symptomatically as chronic bronchitis, anatomically as emphysema, or, most recently, physiologically as airway obstruction (4, 5). ATS and ERS (6) have defined COPD as “a preventable and treatable disease state characterised by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking. Although COPD affects the lungs, it also produces significant systemic consequences”.

Objective demonstration of airflow obstruction by spirometry is mandatory for a diagnosis of COPD. Individuals with chronic cough/sputum production can be at risk for developing airflow obstruction (6). These symptoms, as well as progressive dyspnoea, are common among COPD patients, and they may precede the development of airflow limitation by many years. Thus, all adult individuals (age >40 yrs) with chronic cough/phlegm/progressive dyspnoea, especially if smokers, should be carefully evaluated (6).

Variable definitions and lung function criteria for COPD have made it difficult to quantify the prevalence of the disease around the world (7-9). In addition, a large proportion of patients with COPD in the community remain undiagnosed. In US, about 90% of subjects with undiagnosed airflow obstruction had mild impairment and 10% moderate to severe impairment (10). In Spain, among the subjects with airflow obstruction, previous diagnosis of COPD had been made in only 21.7% of cases (11), and in UK 18.8% of COPD people were undiagnosed (12). The under-recognition and under-diagnosis of COPD lead to significant under-reporting.

Halbert et al (4) have recently published a quantitative summary of the world literature on COPD prevalence, with estimates for COPD in important subgroups defined by age, smoking status, sex, WHO region, study setting (urban or rural), and quality study. It was not possible to locate any spirometric studies reporting COPD prevalence in the African or Eastern Mediterranean regions. The pooled prevalence has been valuated 7.6%, 4.5% in Americas, 11.4% in South-East Asia, 9.0% in Western Pacific, and 7.4% in Europe. The European Lung White Book (13) reports the prevalence of clinically relevant COPD varies in Europe from 4-10% of the adult population.

Active smoking is the most important risk factor for COPD. It has been estimated that about 70% of COPD related mortality is attributable to cigarette smoking (14). Other risk factors than smoking may play an important role in pathogenesis and development of chronic bronchitis and COPD (15). There is enough evidence that poverty, nutritional factors, age, familial and genetic factors, airway hyperresponsiveness, childhood infections, passive smoking, specific occupational exposure, outdoor and indoor air pollution, are risk factors that increase the probability of developing airway obstruction, independent from smoking status (16).

Although the majority of COPD occurs in current or former smokers, a not negligible proportion of the disease also occurs in persons who have never smoked.

Halbert et al estimated a pooled prevalence of COPD diagnosis of 9.2%, in adults over 40 years and of 4.3% (95% Confidence Interval, CI 3.2-5.7) in never-smoker subjects (4). Recent analyses on the Third National Health and Nutrition Examination Survey (NHANES III) data reveal that never smokers represent a significant proportion of airway obstruction in US adults (23% of obstructed subjects), and only one fifth of the obstruction in this group is explained by presence of asthma (17). Results by a recent Japanese Study indicate airflow obstruction in 5.8% of never-smokers (18). Also in Europe it has been observed a sizeable proportion of never-smoker people with COPD, defined by either airflow obstruction or presence of chronic bronchitis/emphysema. In Spain, the prevalence of COPD in never-smoker people resulted 4.1% (19), and 23% of COPD subjects had never smoked (11). The prevalence of obstruction in lifelong nonsmoking subjects was 8.7% in UK (12), 12% in Poland (20), and even 20.4% in Austria (21). In Italy, COPD in never-smokers of a general population ranged from 10.4% to up 38.8%, when different spirometric criteria for defining COPD were used (22). In non-smoker adult Swedes, the prevalence of COPD varied from 3.4 to 24.5%, according to different spirometric cut-off points for COPD (23).

Chronic cough/phlegm was present in 16% of never-smoker Italian women selected by a general population sample (24). Other Italian Study on young adults of the general population showed that 30% of the subjects with chronic cough/phlegm were never-smokers (25). In Finland, about 50% of the women with chronic bronchitis/emphysema had never regularly smoked (26). In Sweden, chronic bronchitis/emphysema was present in about 10% of the general never-smoking population (27).

Indoor air pollution

Indoor exposure more frequently occurs at home, in social private/public settings, or in workplaces. Indoor environments contribute significantly to human exposure to pollutants, because people spend most of their time indoors. Today, indoor air pollution is globally ranked tenth among the preventable risk factors causing burden of disease (28).

Common indoor pollutants and related sources are summarized in Table 1.
Available data in the literature indicate that indoor pollution exposure largely affects respiratory health worldwide. Conservative estimates show that between 1.5 million and 2 million deaths per year could be attributed to indoor air pollution, with a significant proportion of deaths due to COPD (28).

Table 1. Main indoor pollutants and related sources (28)

Type	Pollutant	Typical sources
Combustion products:	*Carbon monoxide (CO)*	Gas ranges and pilot lights, unvented kerosene and gas heaters, wood and coal combustion, tobacco smoke
	Nitrogen dioxide (NO_2)	Gas ranges and pilot lights, unvented kerosene and gas heaters
	Respirable Particulate Matter (PM)	tobacco smoke, wood and coal combustion, fireplaces
	Environmental Tobacco Smoke (ETS)	Tobacco cigarettes and cigars, pipes
Volatile organic compounds (VOCs)	*- Aldehyde (formaldehyde)* *- Aliphatic halogenated hydrocarbons* *- Aromatic hydrocarbons* *- Terpenes*	Furniture, solvents, paints, adhesives, cleaning products, tobacco smoke, insulation materials
Major indoor allergens	*Acarids*	
	House dust mites	Dust, bedding, carpeting
	Pets:	
	Cats or Dogs	Dandruff
	Birds	Feathers
	Insects:	
	Cockroaches	Floors
	Fungi (moulds)	Dampness
	Pollens	Plants
	Rodents	Mice

The aim of this paper is to describe relevant findings, available in scientific literature, on the association of non-smoking related COPD with the exposure to more common indoor air pollutants, in adults.

METHODS

We performed a review of the literature by focusing on COPD, defined as either airflow obstruction or chronic bronchitis/emphysema. Chronic cough or phlegm and dyspnoea have been also considered as health outcomes. Longitudinal studies have confirmed that cough/phlegm are associated to higher risk for COPD development. In the European Community Respiratory Health Survey (ECRHS), in subjects with chronic cough/phlegm both at baseline and at 8-years follow-up, the incidence of COPD was four-fold higher than in subjects who had never reported these symptoms at baseline (29). Lindberg et al, who prospectively studied the incidence of COPD in people with normal lung function (FEV_1 (forced expiratory volume in one second)/FVC (forced vital capacity) ratio $\geq$ 70%) at baseline, concluded that bronchitic symptoms and dyspnoea were significant risk factors for developing COPD, and they persisted after adjustment for possible confounders (30).

We mainly considered studies on the health effects of indoor air pollution to which the general population may be commonly exposed. Specific indoor occupational exposures, that regard only some groups of workers, have been considered only marginally.

RESULTS

In general, we found that few studies investigated the association of non-smoking related COPD with indoor air exposure. Most studies assessed the relationship between COPD and specific occupational exposure, or the health effects of ETS exposure. Biomass combustion was widely investigated as risk factor for COPD, in developing countries. Few studies evaluated the effects by directly measuring levels of pollutants. Information on such exposure has been more likely collected by interview with questions on the presence of known sources of indoor pollution.

Environmental tobacco smoke (ETS)

ETS is produced by tobacco combustion and contains over 4,500 compounds in both vapour and particle phases, many of them being known carcinogens and irritants. ETS is a common major source of indoor PM. Significantly higher concentration of PM has been measured in indoor places where people smoke than in smoking-free indoor environments. The effects of passive smoking have been widely investigated (28). Based on the evidence by literature, the US Environmental Protection Agency (US EPA) (31) concluded that ETS exposure may increase the frequency of respiratory symptoms in adults, and that these effects are estimated to be 30-60% higher in ETS exposed compared to unexposed nonsmokers. Between 10 and 50% of European adults are exposed to ETS (32, 33). Preventable policy legislation has been applied in several countries to reduce ETS exposure at work and in public settings, but no legislative intervention has so far been made in dwellings. In addition, a study performed in some European cities shows that, even in places where smoking is prohibited, the concentration of nicotine indicate that some residuals of tobacco smoke can still be found (34).

Table 2 shows recent studies on the relation of ETS with COPD in never smokers. Chronic bronchitis was the diagnosis more frequently linked to ETS exposure, and the highest risk was reported for never smoking Chinese women exposed to ETS both in childhood and adulthood.

Table 2. Association between ETS and COPD in never smoker adults (OR=odds ratio, CI=Confidence Interval)

Author, ***Source*** **(sample)**	**Country**	**Exposure**	**Disorder**	**OR**	**95% CI**
Simoni M, *Respir Med 2007* (women)(24)	Italy	at home and work	Dyspnea CB/emphysema Cough/Phlegm	1.61 2.24 1.52	1.20-2.16 1.40-3.58 1.07-2.15
Jindal SK, Indian J Chest Dis Allied Sci 2006 *(35)*	India	any	CB	1.40	1.21-1.61
David GL, *Thorax 2005* (women)(36)	China	in childhood and adulthood	CB Chronic Phlegm Chronic Cough	2.87 2.38 2.80	1.58-5.22 1.82-3.12 1.61-4.87
Larsson ML, Eur Respir J 2003*(33)*	Estonia	outside home	Dyspnoea CB/Emphysema	1.65 1.54	1.20-2.27 1.13-3.00
Radon K , *Chest 2002* (37)	German	at work	CB	1.90	1.16-3.11
Iribarren C, J Epidemiol Community Health 2001 *(men)(38)*	US	at home or in other places	Chronic Cough Emphysema	1.60 3.02	1.22-2.10 1.22-7.34
Jedrychowski W, *Int J Occup Environ Health 1995* (39) (elderly women)	Poland	any	Dyspnoea	2.23	1.45-3.44
Leuenberger P, *Am J Respir Crit Care Med 1994* (40)	Swiss	any	Dyspnoea CB	1.45 1.65	1.20-1.76 1.28-2.16
Dayal HH Environ Res 1994 *(41)*	US	at home	ORD	1.86	1.21-2.86

CB=Chronic Bronchitis; ORD=obstructive respiratory diseases.

A recent review of the literature estimated the pooled risk for chronic cough in never smokers heavily exposed to ETS: Odds Ratios (ORs) were similar in both men (1.60, 95%CI 1.22-2.10) and women (1.68, 1.17-2.34) (42). Significant relations between ETS exposure and COPD development have been found in the elderly, too, with an OR range of 1.68-5.63 (43). A French study on never smoker adults found a significant inverse association between ETS exposure and both FVC and FEV_1, with a decrement of 3.16% and 2.90%, respectively, in exposed subjects. To be exposed to ETS at home or at work represented an increased risk for abnormal low FVC (OR 2.71, 1.09-6.75) (44). Also in Scotland there was evident decrement of FVC and FEV_1 in non smoker subjects exposed to ETS, when compared to unexposed ones (45). A dose-response effect was reported by Eisner et al, in a study on the general population in US: chronic bronchitis/emphysema/COPD resulted associated with higher ETS lifetime exposure at home (OR 1.55, 1.09-2.21) or at work (1.46, 1.08-1.96), after controlling for smoking history and sociodemographic characteristics; the association was significant also after discarding from the analyses the subjects who reported chronic bronchitis alone (2.38, 1.42-3.90; and 1.79, 1.21-2.65, respectively (46). A significant dose related increase in the risk for developing dyspnoea has been observed in young adults for an average exposure of 10 cigarettes/day (OR 2.37, 1.25-4.51) (47).

Biomass fuels

Indoor air pollution from biomass (wood/coal) use for either cooking or heating is an important risk factor for COPD, especially in women. About 50% of world's households burn these products for cooking in open fire or with inefficient stoves in poorly ventilated rooms (48). It occurs especially in developing countries, where the production of PM and CO (a proxy for $PM_{2.5}$) by biomass combustion is dramatically high (49). Through an extensive review of epidemiological studies around the world, the estimation of the risk by biomass use for COPD results in ORs of 1.8 (1.0-2.8) in males and 3.2 (2.3-4.8) in females (50). In US, the presence of coal stove increased the risk for chronic inflammatory and obstructive respiratory symptoms, in non smoker adults, with OR ranging from 1.8 to 3.3 (C.I. 1.0-5.9) (51). A selection of studies concerning the association of biomass fuel use with COPD is reported in Table 3.

Table 3. Association between biomass fuel and COPD in adults (OR=odds ratio, CI=Confidence Interval).

Author - ***Source*** **(sample)**	**Country**	**Exposure**	**Health outcome**	**OR**	**95% CI**
Shengming L, *Thorax 2007* (never smoker women)(52)	China	biomass fuel	$FEV_1/FVC<0.70$	3.11	1.63-5.94
Orozco-Levy M, *Eur Respir J 2006* (women)(53)	Spain	wood and charcoal smoke	Dyspnoea CB	1.45 1.65	1.20-1.76 1.28-2.16
Ekici A, Environ Res 2005 *(women) (54)*	Turkey	biomass vs GPL	$FEV_1/FVC<0.70$ or CB	2.5	1.5-4.0
Golshan M, *Respir Med 2002* (never smoker women)(55)	Iran	biomass fuel	CB	2.91	2.08-4.40
Dennis RJ, *Chest 1996* (women)(56)	Columbia	wood-smoke	$FEV_1/FVC<0.70$	3.9	1.7-9.1
Xu X, *Rev Respir Dis 1993* (non smokers)(51)	US	Coal stove use: for both cooking and heating	Chronic Cough Chronic Phlegm	1.8 2.0	1.0-3.3 1.2-3.4

CB=Chronic Bronchitis; FEV_1=forced expiratory volume in one second; FVC=forced vital capacity.

Most studies have been performed in developing countries, mainly on women. Biomass fuel use resulted associated with airflow obstruction in women living in Turkey, Columbia, and China. In Turkey, after adjusting for possible confounding factors, the risk for COPD, defined either as $FEV_1/FVC<0.70$ or chronic bronchitis, was higher in women who used biomass fuel than in those who used GPL (liquid petroleum gas), and the attributable fraction of COPD to biomass smoke was 23.1% (54).

In addition (not shown in Table 4), other two studies performed in Turkey found: 1) women exposed to biomass fumes more likely to suffer from chronic bronchitis and COPD than those unexposed, even though the prevalence of current smoking was higher among the latter (57); 2) never smoker housewives exposed for 30+ years to biomass fuel to be at higher risk for developing COPD than those never exposed (OR 6.61, 2.17-20.18) (58). In Mexico, the exposure to biomass smoke has been associated with chronic bronchitis and chronic airflow obstruction, in adults. Among never smoker women, those exposed to wood smoke had a five-fold risk as compared to the unexposed (59), and women exposed domestically to biomass developed COPD with clinical characteristics, quality of life, and increased mortality similar in degree to that of tobacco smokers (60). In China, coal smoke derived from home heating was associated with high reporting of persistent cough and phlegm (61).

Recently, Orozco-Levy et al have evidenced that biomass fuel may be a risk factor for COPD also in Europe. In their Spanish case-control study in women, exposure to wood or charcoal smoke was associated with COPD after adjusting for age and smoking. Wood or charcoal alone independently increased the risk of COPD (OR 1.8 and 1.5, respectively), but only the combination of both was statistically significant. The association between length of exposure and COPD suggested a dose-response pattern (53).

Mould/dampness

Building dampness may lead to emission of odorous or irritation compounds from microorganisms or chemical degradation of building materials, such as formaldehyde (VOC).

Reported prevalence rates of home mould/dampness range widely around the world: from 10 to up 50% (62).

There is evidence that long-term exposure to mould/dampness is linked to higher risk for cough, phlegm, or dyspnoea, in adults. Dales et al, in Canadian adults, found that dampness/moulds were associated with respiratory symptoms, including cough, phlegm, or dyspnoea, with an OR of 1.62 (1.48-1.68)(63).

The Institute of Medicine (IOM) of the National Academy of Sciences has published, in 2004, a critical review of the scientific literature pertaining to the association of indoor dampness and mould contamination with adverse health effect (64). Recently, through a quantitative meta-analysis of the studies reviewed by IOM, Fisk et al estimated the pooled OR for cough in adults to be 2.10 (1.27-3.47) (65).

Table 4 reports details of the studies considered in the meta-analysis. All the studies have been performed in European Nordic countries. Another study performed on Swedish adults found, by meta-analysis, that an exposure of at least 3 years to damp or mouldy odour at home was associated with persistent cough with OR ranging from 1.32 to 5.86 (95%CI from 1.22 to 6.19)(70).

Table 4. Association between mould/dampness at home and cough/phlegm in adults (OR=odds ratio, CI=Confidence Interval).

Author - *Source* (sample)	**Country**	**Exposure**	**Disorder**	**OR**	**95% CI**
Brunekreef B, *Allergy 1992* (66)	Netherlands	damp	Chronic Cough: men	2.56	1.94-3.38
			women	1.75	1.30-2.36
			Chronic Phlegm: men	2.56	1.94-3.38
			women	1.66	1.16-2.38
Gunnbjornsdottir MI *Respir Med 2003* (youg adults)(67)	Sweden	visible mould/ water damage	Chronic Cough	2.23	1.24-4.00
Koskinen OM, Eur Respir J 1999 *(68)*	Finland	mould	Cough	1.60	1.01-4.01
Pirhonen I, *Eur Respir J 1996* (69)	Finland	mould/damp	CB	1.51	0.96-1.35
			Cough	1.37	0.99-1.88
			Phlegm	1.36	1.01-1.85

CB=Chronic Bronchitis.

Gas/kerosene fuels

Some studies have evidenced associations of COPD with gas/kerosene fuel use for both heating or cooking. Gas/kerosene combustion mainly produce nitrogen dioxide and carbon monoxide. In UK, decrements in FEV_1 (-70mL) and in FVC (-35mL) have been observed in young adults using gas fuel when compared to those using electricity for cooking (71). In Poland, never smoker elderly women exposed to high gas cooking showed an elevated risk for dyspnoea (OR 7.16, 5.02-10.2) (39). In US, kerosene heaters use in never smoking women living in nonsmoking households was associated with increased cough (OR 1.05, 1.01-1.09)(72). In Italy, in a rural general population sample, the use of bottled gas for cooking, resulted related to higher risk of chronic cough in males (OR 1.66, 1.12-2.46) and dyspnoea in males (OR 1.81, 1.15-2.85) and females (OR 1.45, 1.00-2.10)(73). The association between chronic cough and use of bottled gas, instead of natural gas, was also confirmed in Italian male nonsmokers of a urban general population (OR 2.82, 1.12-7.10); the presence of stoves for heating (mainly non-natural gas stoves) inside the home resulted a risk factor for attacks of shortness of breath in nonsmoker women, when compared to those who lived in dwellings with central heating (stoves outside the home)(OR 1.72, 1.11-2.65)(74).

Objectively measured indoor pollutants and COPD

As above reported, few studies on the relationships between COPD and indoor pollution were based on direct measurements of pollutants concentrations, except for specific occupational exposure. In Mexico, non smoking rural women, long-term exposed, when cooking, to peaks of PM_{10} > 2.6mg/m^3 , showed a borderline significantly higher risk for having FEV_1/FVC <70% and FEV_1<80% predicted (OR 3.5, 0.94-16.3) than those exposed to lower concentration (75). In China, in those exposed to elevated indoor PM_{10} level, higher prevalence of chronic cough and phlegm (76) and adverse effects on the lung function have been observed (77). Effects on lung function have been found in Italy, too. In a general population of adults, the exposure to high concentration of $PM_{2.5}$ resulted in both increased maximum amplitude (OR 1.38, 1.24-1.54) and diurnal variation (1.37, 1.23-1.53) of peak expiratory flow (78).

Occupational exposure

A brief comment has to be devoted to specific occupational exposure. Even if it involves only specific groups of persons and can not be defined as common indoor air exposure for the general population, it often occurs in indoor environments. Occupational exposure is an important risk factor for COPD independently of tobacco smoke, and several studies report a causal association between specific work-related exposures and COPD. Dust or chemical agents to whom some categories of workers are exposed result in inflammation, a key factor in the pathogenesis of COPD. Chronic inflammation throughout the airways, parenchyma, and pulmonary vasculature are hallmarks of the disease process and lead to the pathologic changes characteristic of COPD (79). Blanc and Toren (80), in their recent review, estimated a population attributable risk (PAR) of 15% due to occupational factors, when the outcome analyzed was either chronic bronchitis or airflow obstruction. Thus, they have confirmed the figures previously published by the ad hoc committee of the ATS (81). In Spain, among the workers in the textile industry, lung function impairment resulted related to exposure duration, being independent of the effect of smoking. (82). Data from the ECRHS Study showed that occupational exposures to dust/fumes, vapours, or gas were risk factors for chronic cough/phlegm (relative risk ratio (RRR) 1.47, 1.31-1.65) and for COPD (by GOLD criteria, 1.62, 1.24-2.12), also after adjustment for sex, ETS exposure, smoking status, socio-economic status, and respiratory infection (83). The European Farmer Study (Denmark, Germany, Switzerland, and Spain), found a COPD prevalence of 17% in never-smoker farmers working inside animal confinements buildings, and higher risk for having COPD in subjects highly exposed to indoor dust (OR 6.6, 1.1-39.5) (84). Finally, in Poland, among the workers in a pesticide producing factory, chronic bronchitis/emphysema or COPD (by GOLD criteria) was more prevalent in exposed to pesticides than in unexposed ones (19.3 vs 3%)(85).

COPD Exacerbations

Acute exacerbations that increase both morbidity and mortality are common among the subjects with diagnosis of COPD. Exacerbations are accompanied by increased specific symptoms (i.e. dyspnoea or phlegm), and frequently require medical intervention or hospitalization. Studies report significant relationships between acute exacerbations of COPD and exposure to increased levels of outdoor particulate pollution. Considering that indoor levels of PM may be several fold higher than outdoors, high indoor levels of PM might be associated with COPD exacerbations, too. Indeed, among Spanish COPD patients, ETS - one of the major sources of indoor PM - resulted associated with increased hospital readmission for COPD exacerbations (OR 1.63, 1.04-2.57)(86). A similar result was found in a study on US never smoker adults, who were more likely to report exacerbation of chronic respiratory disease (including chronic bronchitis and emphysema) when they were exposed to ETS (OR 1.44, 1.07-1.95) (87).

CONCLUSION

In spite of increased COPD prevalence (and its predicted increasing in the next years), and the evidence that other risk factors than smoking may be associated to COPD development, we found relatively few studies that assessed the association between COPD and common indoor air pollution in adult general population, except for studies on ETS and, in developing countries, biomass combustion exposure. Both these exposures prove to be risk factors for non-smoking related COPD. It would be important to improve awareness on adverse health effects possibly associated with biomass combustion-related air pollution in developed countries among others because of the increasing interest for wood and other biomasses as potential alternative energy sources.

Mold/dampness exposure results associated to symptoms/signs, which may be related to the presence of COPD or its development.

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