

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ



# COPD in Non-Smokers

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The WHO Global Burden of Disease Project estimated that COPD was the fifth leading cause of death worldwide in 2001 and will be the third leading cause by 2020

The prevalence of physiologically defined chronic obstructive pulmonary disease in adults aged  $\geq 40$  yrs is approximately 9–10%

(Eur Respir J 2006; 28: 523–532)

**An estimated 25–45% of patients with COPD  
have never smoked**

**the burden of non-smoking COPD is therefore  
much higher than previously believed**

*(Lancet 2009; 374: 733–43)*


Evidence suggests that rates of disease are generally underestimated

PLATINO study:

- Among 5,315 subjects studied, 2278 were never smokers and 3036 were ever smokers
- COPD was observed in 3.5% of never smokers and in 7.5% of ever smokers
- Never smokers comprised **26%** of all individuals with airflow obstruction

## *Other studies:*

Report from  
the Obstructive  
Lung Disease in  
Northern  
Sweden (**OLIN**)  
studies:



- Both among subjects with airway obstruction and among subjects with GOLD stage II, the proportions of nonsmokers were **20%**

On a global scale, the majority of chronic non-reversible airway obstruction occurs in current or former smokers

But

Results from the Austrian BOLD study showed that:

Approximately **every third subject** with non-reversible airway obstruction has never smoked, yet still demonstrates a substantial burden of symptoms and impairment of quality of life

*Respiratory Medicine (2008) 102, 1833e1838*



# RISK FACTORS

- Active and passive smoking

(The risk attributable to active smoking in COPD is thought to vary from 40% to 70% according to the country)

- Genetic factors

(At present, only a severe deficit in  $\alpha$ 1-antitrypsin, responsible for the PiZZ phenotype, is a proven genetic causal factor)

- Occupational exposure

(The risk attributable to occupational exposure in COPD has been estimated at 19%, and for nonsmokers at 31%)

- Air pollution

(The role of air pollution in terms of risk factors is not well known)

Its impact as an aggravating factor has been shown in patients with the most severe forms of COPD during peaks in air pollution)

# RISK FACTORS

- Air pollution (Indoor)

(Exposure to pollution inside the home, in particular in developing countries, is an important risk for COPD)

- Age

(In the course of life, there is a physiological decline in respiratory function which begins around the age of 30–40 yrs)

- Sex

(From an epidemiological point of view, males were classically more at risk of developing COPD in comparison with females because of their smoking habits  
Whether females are more at risk of developing COPD when they undertake similar smoking habits to their male counterparts is still under debate)

# RISK FACTORS

- Infections

(Infections seem to play an important role in the occurrence of COPD

some authors have suggested that the onset of COPD might be facilitated by a *latent adenoviral infection*)

- Bronchial hyper reactivity

(Whether bronchial hyper reactivity is a factor in the development of COPD is still under debate)

(Eur Respir Rev 2009; 18: 114, 213–221)

# Tuberculosis

It is now also becoming clear that TB, like tobacco smoke, besides its known consequences of bronchiectasis and other pulmonary morbidity, is also a significant risk factor for the development of COPD

(Eur Respir J 2010; 35: 27–33)

# RISK FACTORS

## Non-smoking risk factors associated with chronic obstructive pulmonary disease

### Indoor air pollution

- Smoke from biomass fuel: plant residues (wood, charcoal, crops, twigs, dried grass) animal residues (dung)
- Smoke from coal

### Occupational exposures

- Crop farming: grain dust, organic dust, inorganic dust
- Animal farming: organic dust, ammonia, hydrogen sulphide
- Dust exposures: coal mining, hard-rock mining, tunnelling, concrete manufacturing, construction, brick manufacturing, gold mining, iron and steel founding
- Chemical exposures: plastic, textile, rubber industries, leather manufacturing, manufacturing of food products
- Pollutant exposure: transportation and trucking, automotive repair

# RISK FACTORS (Cont...)

Treated pulmonary tuberculosis

Lower-respiratory-tract infections during childhood

Chronic asthma

Outdoor air pollution

- Particulate matter (<10  $\mu\text{m}$  or <2.5  $\mu\text{m}$  diameter)
- Nitrogen dioxide
- Carbon monoxide

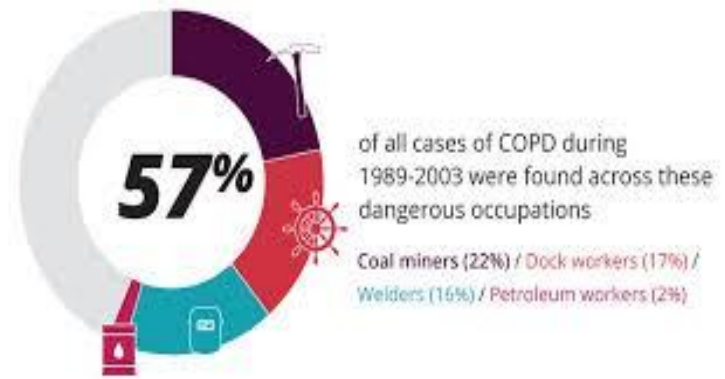
Poor socioeconomic status

Low educational attainment

Poor nutrition

(Lancet 2009; 374: 733–43)

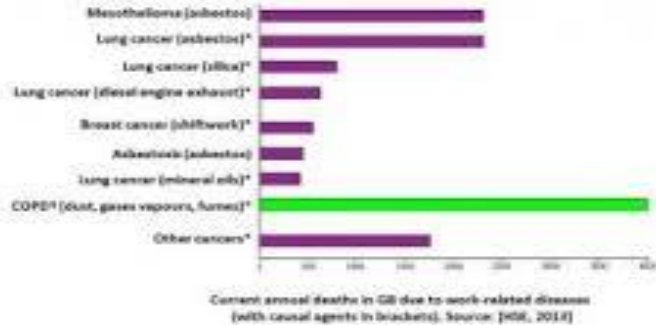
# Occupational exposures and COPD



analysis of international data showed that the prevalence of exposures predicted COPD prevalence: 0.8% increase in COPD prevalence per 10% increase in exposure prevalence

By comparison, for every 10% increase in the proportion of the ever-smoking population, the prevalence of COPD GOLD stage II or above increased by 1.3%

(Eur Respir J 2009; 33: 298–304)



Given the observed median population COPD prevalence of 3.4%, the model predicted that:

a **20%** relative reduction in the disease burden (i.e. to a COPD prevalence of 2.7%) could be achieved by a **5.4% reduction in overall smoking rates** or an **8.8% reduction in the prevalence of occupational exposures**

*(Eur Respir J 2009; 33: 298–304)*



## HSE says...

- Work related COPD is a priority because of the human costs in terms of suffering, its effects on the quality of life and the financial costs due to working days lost and medical treatment.
  - Around 15% of COPD may be caused or made worse by dusts, fumes and irritating gases
  - 4,000 COPD deaths every year may be related to work exposures
  - 40% of COPD patients are below retirement age
  - A quarter of those with COPD below retirement age are unable to work at all


ردیف	ضایعات انسدادی راههای هوایی (شامل آسم، COPD و ...)	درصد از کارافتادگی
۱	CLASS I $FEV_1 / FVC \geq 70$ , $FVC \geq 80$ , $FEV_1 \geq 80$	%۰
۲	CLASS II $(FEV_1 / FVC) (79-55)$ , $(FVC) (79-55)$ $FEV_1 / FVC (79-55)$	%۱۰-۳۰
۳	CLASS III $(FEV_1 / FVC) (55-41)$ , $(FEV_1) (55-41)$	%۳۰-۵۰
۴	CLASS IV $FEV_1 < 40$	%۵۰-۶۶
۵	$PEFR < 100$ L/min یا $PEFR < 20$ % $FEV_1 < 1$ Lit یا $FEV_1 < 25$ % که پس از داروهای گشادکننده برونش پاسخ ناچیز داشته	%۶۶
۶	افرادی که همکاری جهت اسپرومتری ندارند براساس معاینات بالینی X-Ray ، ABG (هیپوکسی، هیپرکاپنی) درصد داده می شوند. - اگر $PO_2 \downarrow (60-55)$ و $PCO_2 \uparrow (45-55)$ باشد - اگر COPD همراه با RHf (نارسایی قلب راست باشد)	%۶۶ %۵۰-۶۶

♦ مطابقت اسپرومتری با علائم کلینیکی و دیگر آزمایشات پاراکلینیک در تقسیم بندی بالا باید همیشه مدنظر باشد.

ردیف	بیماریهای ریوی ناشی از محیط Environment Lung Disease	درصد از کارافتادگی
۱	تصمیم گیری براساس علائم بالینی، آزمایشات، گرافی سینه، اسپرومتری و ABG	براساس جدول صفحه قبل
۲	آزستوزیس به اضافه سرطان ریه	%۶۶
۳	آزستوزیس به اضافه مزوتلیوما ی پلور - صفاق	%۶۶
۴	سلیکوزیس به اضافه تومور ریه یا سرطان پلور	%۶۶



TABLE 5-4 Criteria for Rating Permanent Impairment due to Pulmonary Dysfunction<sup>a</sup>

 <b>Pulmonary Dysfunction</b>					
CLASS	CLASS 0	CLASS 1	CLASS 2	CLASS 3	CLASS 4
WHOLE PERSON IMPAIRMENT RATING (%)	0	2%-10%	11%-23%	24%-40%	45%-65%
SEVERITY GRADE (%)		2 4 6 8 10 (A B C D E) (Minimal)	11 14 17 20 23 (A B C D E) (Mild)	24 28 32 36 40 (A B C D E) (Moderate)	45 50 55 60 65 (A B C D E) (Severe)
HISTORY	No current symptoms  and/or intermittent Dyspnea that does not require treatment	Dyspnea controlled with intermittent or continuous treatment  or intermittent, mild Dyspnea despite continuous treatment	Constant mild Dyspnea despite continuous treatment  or intermittent, moderate Dyspnea despite continuous treatment	Constant moderate Dyspnea despite continuous treatment  or intermittent, severe Dyspnea despite continuous treatment	Constant severe Dyspnea despite continuous treatment  or intermittent, extreme Dyspnea despite continuous treatment
PHYSICAL FINDINGS	No current signs of disease	Physical findings not present with continuous treatment  or intermittent, mild physical findings	Constant mild physical findings despite continuous treatment  or intermittent, moderate findings	Constant moderate physical findings despite continuous treatment  or intermittent, severe findings	Constant severe physical findings despite continuous treatment  or intermittent, extreme findings
OBJECTIVE TESTS					
FVC	FVC $\geq$ 80% of predicted	FVC between 70% and 79% of predicted	FVC between 60% and 69% of predicted	FVC between 51% and 59% of predicted	FVC between 50% and 45% of predicted
FEV <sub>1</sub>	and FEV <sub>1</sub> $\geq$ 80% of predicted	or FEV <sub>1</sub> between 65% and 79% of predicted	or FEV <sub>1</sub> between 64% and 55% of predicted	or FEV <sub>1</sub> between 45% and 54% of predicted	or FEV <sub>1</sub> below 45% of predicted
FEV <sub>1</sub> /FVC (%)	and FEV <sub>1</sub> /FVC (%) lower limits of normal ( $>$ 75% of predicted)	or	or	or	or
DLco	and DLco $\geq$ 75% of predicted	DLco between 65% and 74% of predicted	DLco between 55% and 64% of predicted	DLco between 45% and 54% of predicted	DLco below 45% of predicted
Vo <sub>2</sub> max	or >25mL/(kg·min) or >7.1 METs	or between 22 and 25 mL/(kg·min) or 6.3–7.1 METs	or between 21 and 18 mL/(kg·min) or 5.1–6.0 METs	or between 17 and 15 mL/(kg·min) or 4.3–5.0 METs	or <15mL/(kg·min) or <4.3 METs
<sup>a</sup> FVC indicates forced vital capacity; FEV <sub>1</sub> , forced expiratory volume in the first second; DLco, diffusion capacity for carbon monoxide; Vo <sub>2</sub> max, maximum oxygen consumption; and METs, metabolic equivalents (multiples of resting oxygen uptake).					

No clinical data proves that COPD can derive from occupational exposure

The phenotype of patients with occupational exposure and those exposed to tobacco is similar

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)

scientific evidences prove that occupational exposure have a tobacco synergic effect on COPD patients increasing respiratory symptoms, emphysema, patients' mortality due to respiratory problems, and also reducing the pulmonary function

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)

- In the diagnosis of COPD due to occupational exposure, **two main factors** should be taken into account. On the one hand, it is necessary to achieve the **correct diagnosis** of the disease and, on the other, a **causal link** should be established so as to obtain a detailed occupational history of the patient in which the following items need to be included:
  - i. Chronological list of jobs
  - ii. Title, and description of the activity
  - iii. Description of possible exposures in each job
  - iv. Duration of the exposure, use of protection, ventilation

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)

Where necessary a  
hygienist should review the  
work place to determine  
the exposure risk

## **Agents and Professions Involved in Occupational COPD:**

### **1) Mineral Dust**

Coal

Silica and gold miner

Iron / Steel

Asbestos

Ceramic fibers

Construction workers

Cement

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)



## **2) Biological Dust:**

Farmers and agricultural workers

Workers in Swine operations

Cotton

Grain dust

Wood

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)

### **3) Gases-Fumes:**

Cadmium fume

Welding fumes

Painters

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)

- **Prevention** is the best strategy to prevent the impact of the exposure on respiratory health of the workers. This may be carried out at three levels:

1) **Primary**: this is based on developing strategies that control and inform of the level of personal exposure and protection (administrative controls, technical controls, protection equipment). Control of smoking (as a principal aetiological agent of COPD) in the workplace must also be a priority

- 2) **Secondary**: to detect the disease earlier. Medical follow-up programs that include periodic questionnaires on symptoms, and spirometry that could detect workers with occupational exposure and at risk of developing COPD earlier
- 3) **Tertiary**: treatment of diagnosed patients in accordance with clinical practice guidelines and avoid or reduce exposure in the place of work

(Current Respiratory Medicine Reviews, 2012, 8, 436-440)

# Indoor solid fuel

About **3 billion** people worldwide are exposed to smoke from biomass fuel compared with **1.01 billion** people who smoke tobacco

About **50%** of deaths from COPD in developing countries are attributable to **biomass smoke**, of which about **75% are of women**

*(Lancet 2009; 374: 733–43)*

Nearly **2 billion kg** biomass are burnt everyday in developing countries alone, and in some developed and developing countries the decline in biomass use has slowed or even **reversed**, especially in poorer households

*(Lancet 2009; 374: 733–43)*

COPD and chronic  
bronchitis risk of indoor air  
pollution from solid fuel: a  
systematic review and  
meta-analysis (*Thorax*  
*2010;65:221e228.*  
*doi:10.1136/thx.2009.1246*  
*44*):

- There were positive associations between the use of solid fuels and COPD (OR 2.80, 95% CI 1.85 to 4.0) and chronic bronchitis (OR 2.32, 95% CI 1.92 to 2.80)
- exposure to **wood smoke** while performing domestic work presents a greater risk of development of COPD and chronic bronchitis than other fuels

Burden of disease due to indoor air pollution from solid fuel use (WHO/SDE/PHE/07.01 rev):



Estimate that percentage of population using solid fuels in Iran is less than 5%



And



COPD deaths attributable to solid fuel use ( 30 years) is 110



# Outdoor air pollution

The association between high concentrations of outdoor air pollutants and COPD exacerbations and worsening of pre-existing COPD is supported by strong evidence, but the evidence to support an association with **new cases** of COPD is **not** yet available

# Socioeconomic status

- Poor socioeconomic status is a risk factor independently associated with COPD
- is likely to be indicative of other factors such as intrauterine growth retardation, poor nutrition (low intake of antioxidants) and housing conditions, childhood respiratory-tract infections, and exposure to tobacco smoke, biomass smoke and other indoor air pollutants, and occupational risks

(Lancet 2009; 374: 733–43)

- Socioeconomic status has been shown to have a significant correlation with lung function, even after adjustment for smoking status, occupational exposures, and ethnic origin

(Lancet 2009; 374: 733–43)

**Thanks for your attention**

