Silica dust and COPD, is there an association?

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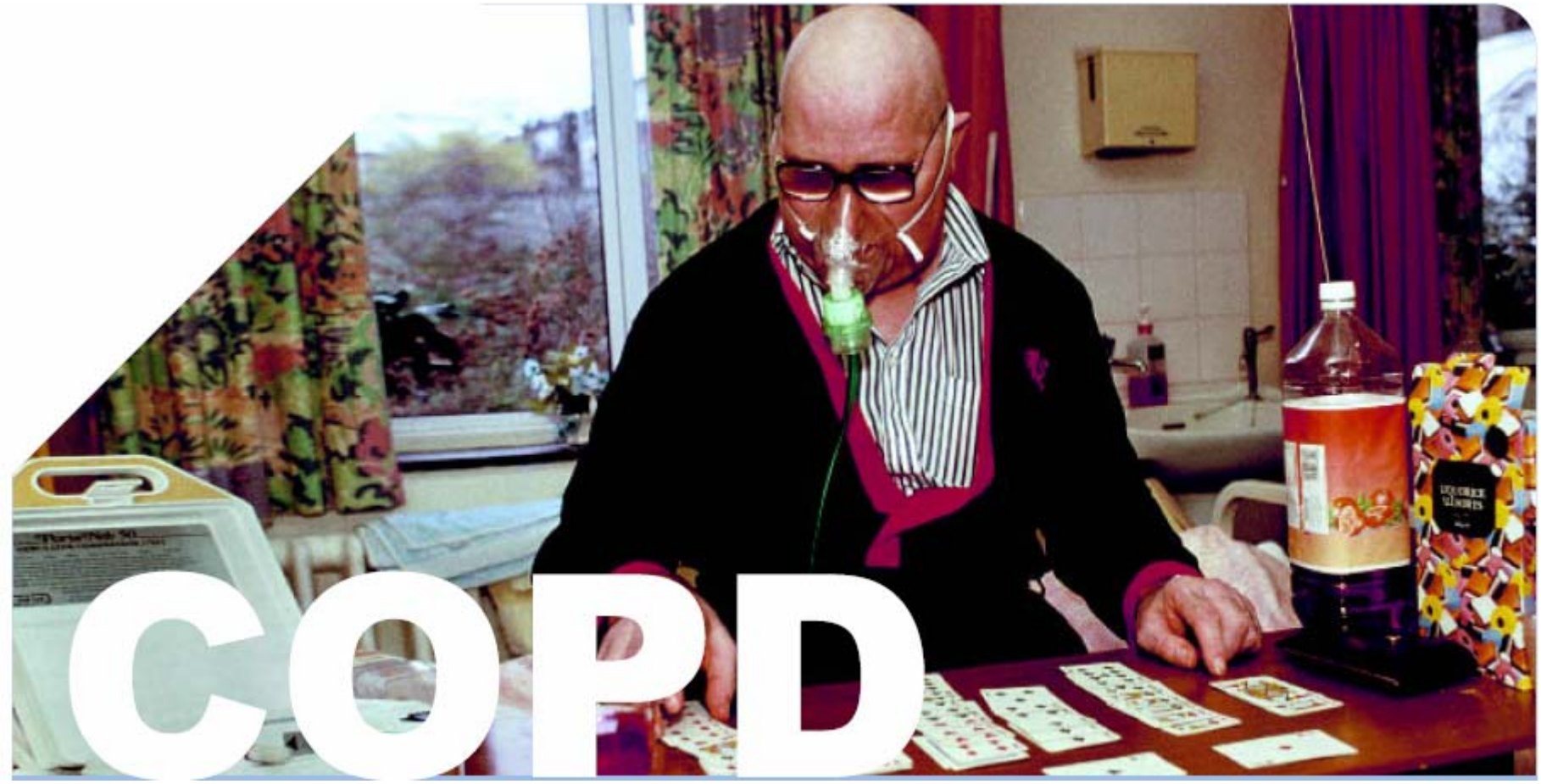
Outline:

- what is COPD?
- some fact about COPD
- what causes COPD?
- role of silica dust
- areas for actions
- summary

what is COPD?



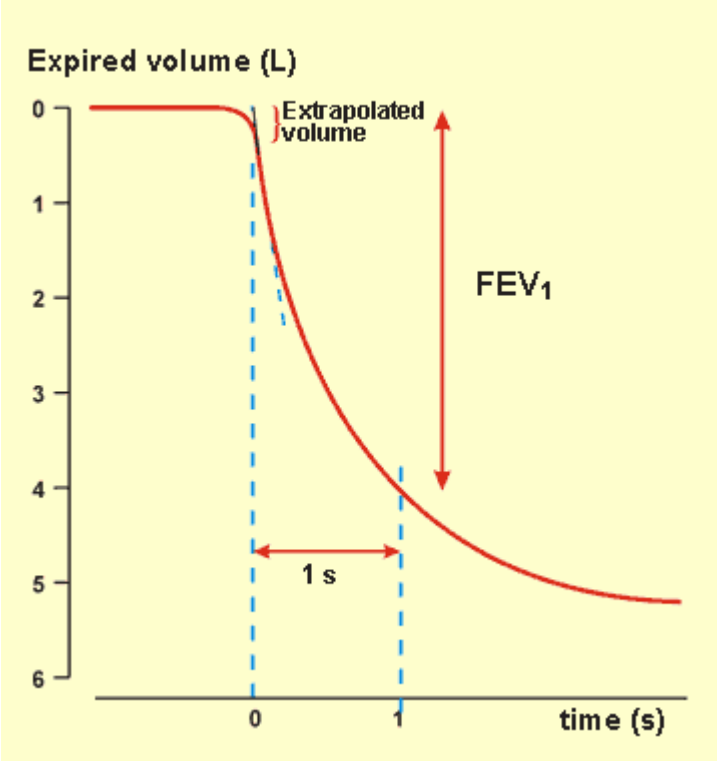
COPD = chronic obstructive pulmonary disease

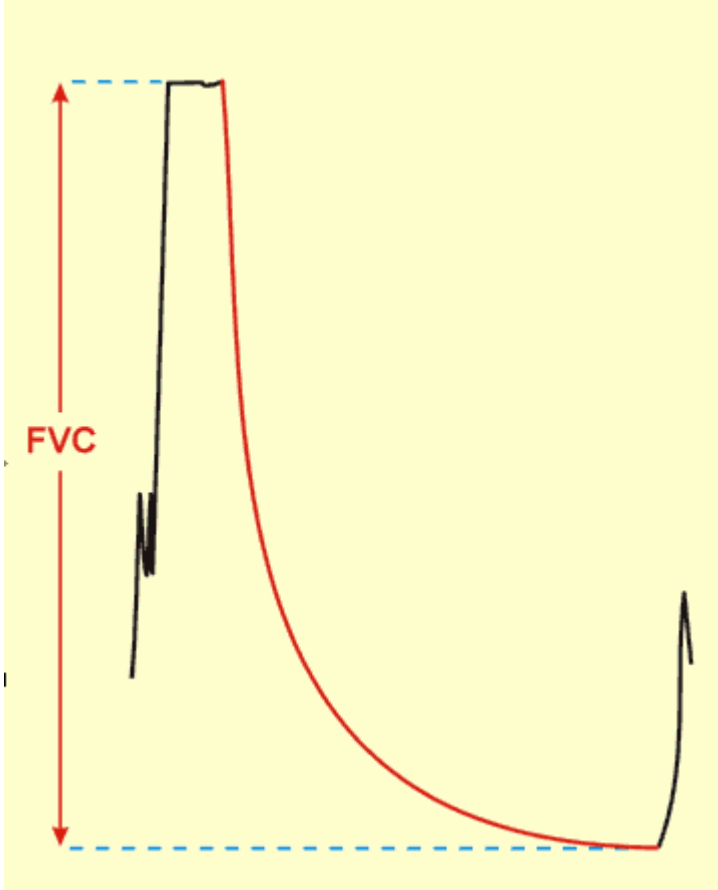
Global Initiative for Chronic Obstructive Lung Disease

Figure 1-2. Spirometric Classification of COPD Severity Based on Post-Bronchodilator FEV_1

Stage I: Mild	$FEV_1/FVC < 0.70$ $FEV_1 \geq 80\%$ predicted
Stage II: Moderate	$FEV_1/FVC < 0.70$ $50\% \leq FEV_1 < 80\%$ predicted
Stage III: Severe	$FEV_1/FVC < 0.70$ $30\% \leq FEV_1 < 50\%$ predicted
Stage IV: Very Severe	$FEV_1/FVC < 0.70$ $FEV_1 < 30\%$ predicted or $FEV_1 < 50\%$ predicted plus chronic respiratory failure

FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; respiratory failure: arterial partial pressure of oxygen (PaO_2) less than 8.0 kPa (60 mm Hg) with or without arterial partial pressure of CO_2 ($PaCO_2$) greater than 6.7 kPa (50 mm Hg) while breathing air at sea level.

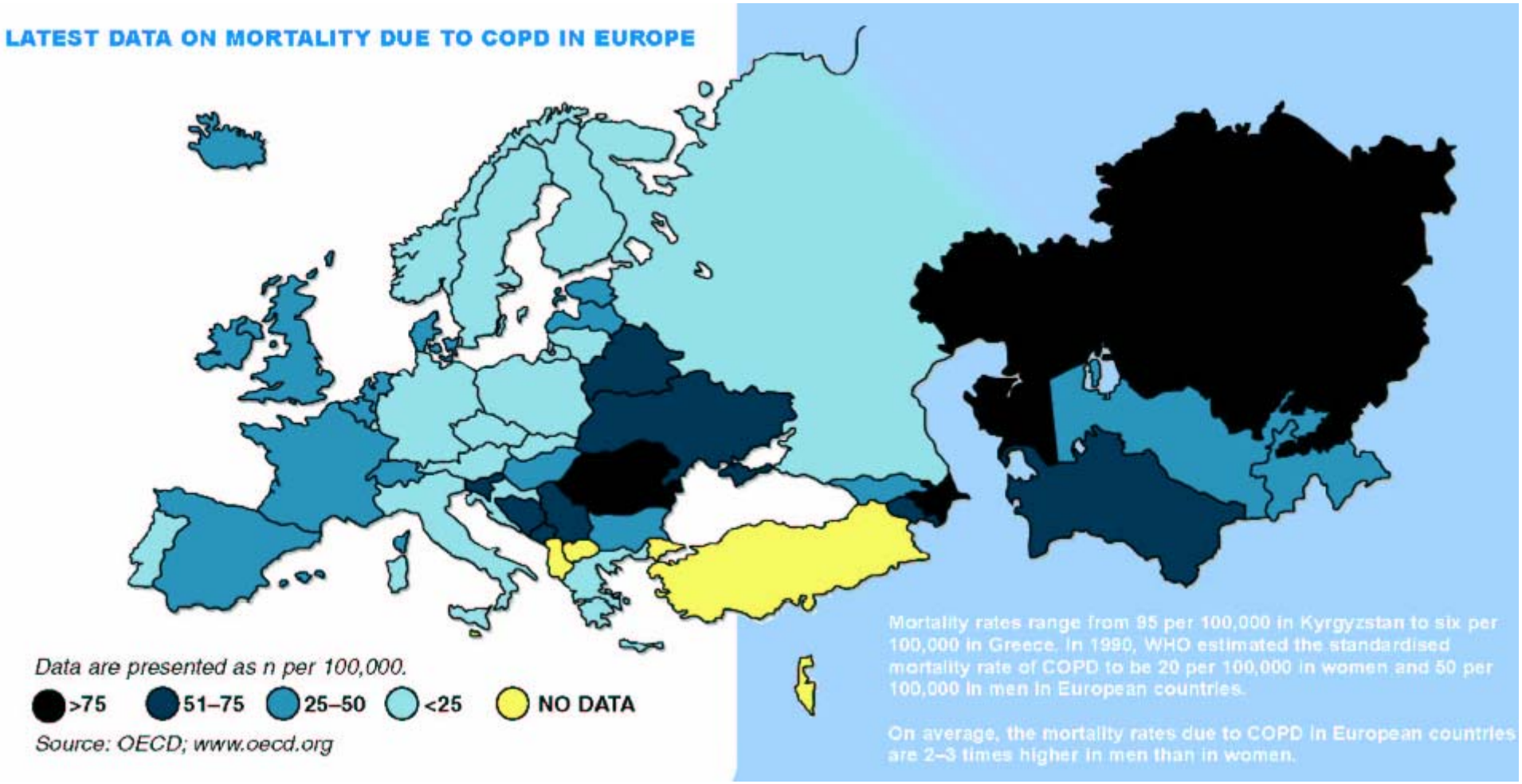




FEV1 is the volume exhaled during the first second of a forced expiratory maneuver started from the level of total lung capacity.

FVC - forced expiratory vital capacity
The volume change of the lung between a full inspiration to total lung capacity and a maximal expiration to residual volume.

some fact about COPD



Burden in Europe

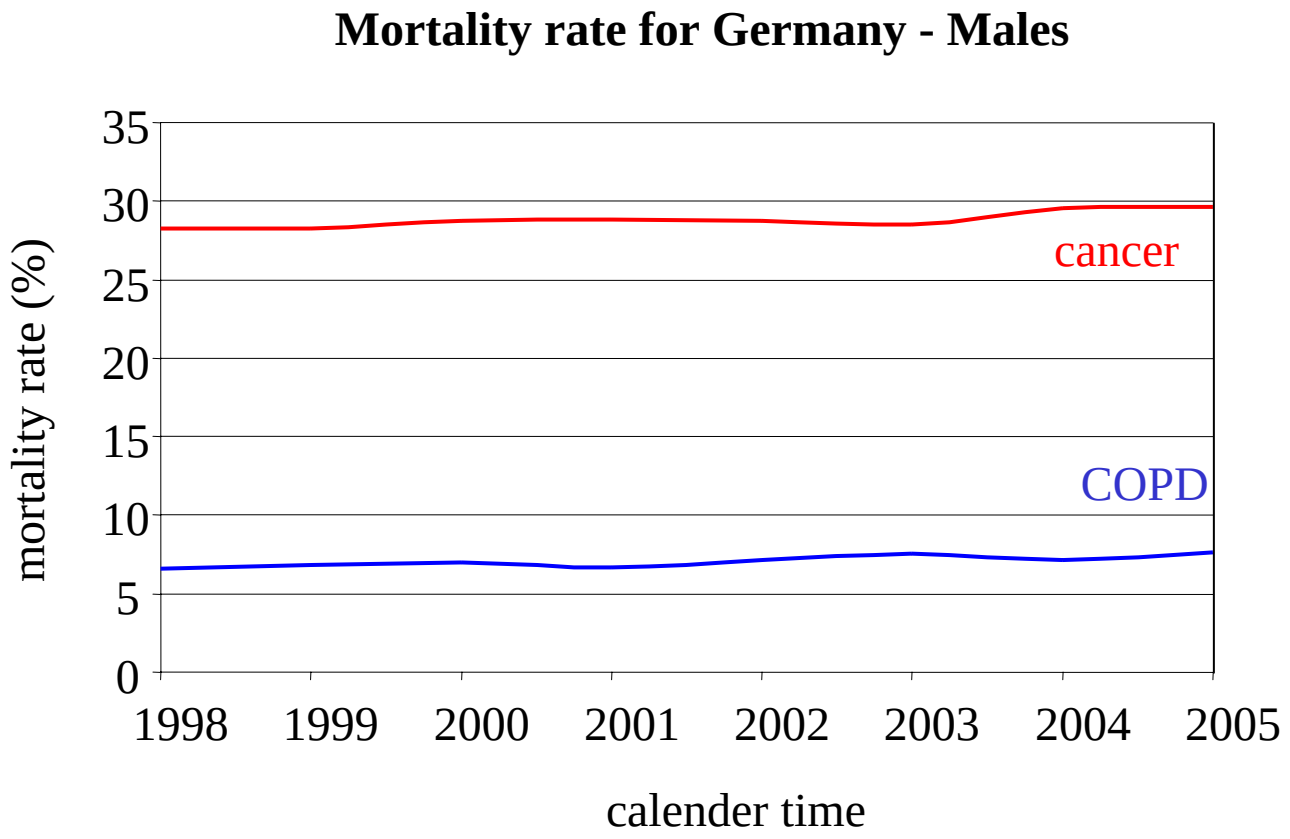
There are approximately 44 million cases of COPD worldwide. In developed countries, COPD is seen in late-middle and old age, usually after 45 years.

Occurrence in Europe

The frequency of clinically relevant COPD varies in European countries from 4–10% of the adult population. Data on the frequency of COPD in Central and Eastern Europe are very limited.

Deaths from COPD

Approximately 200,000–300,000 people die each year in Europe because of COPD. Data provided by the World Health Organization (WHO) in 1997 showed that COPD was the cause of death in 4.1% of men and 2.4% of women in Europe.


Mortality rate for Germany - Males
mortality rate (%)
35
30
25
20
15
10
5
0
cancer
COPD
1998
1999
2000
2001
2002
2003
2004
2005
calender time

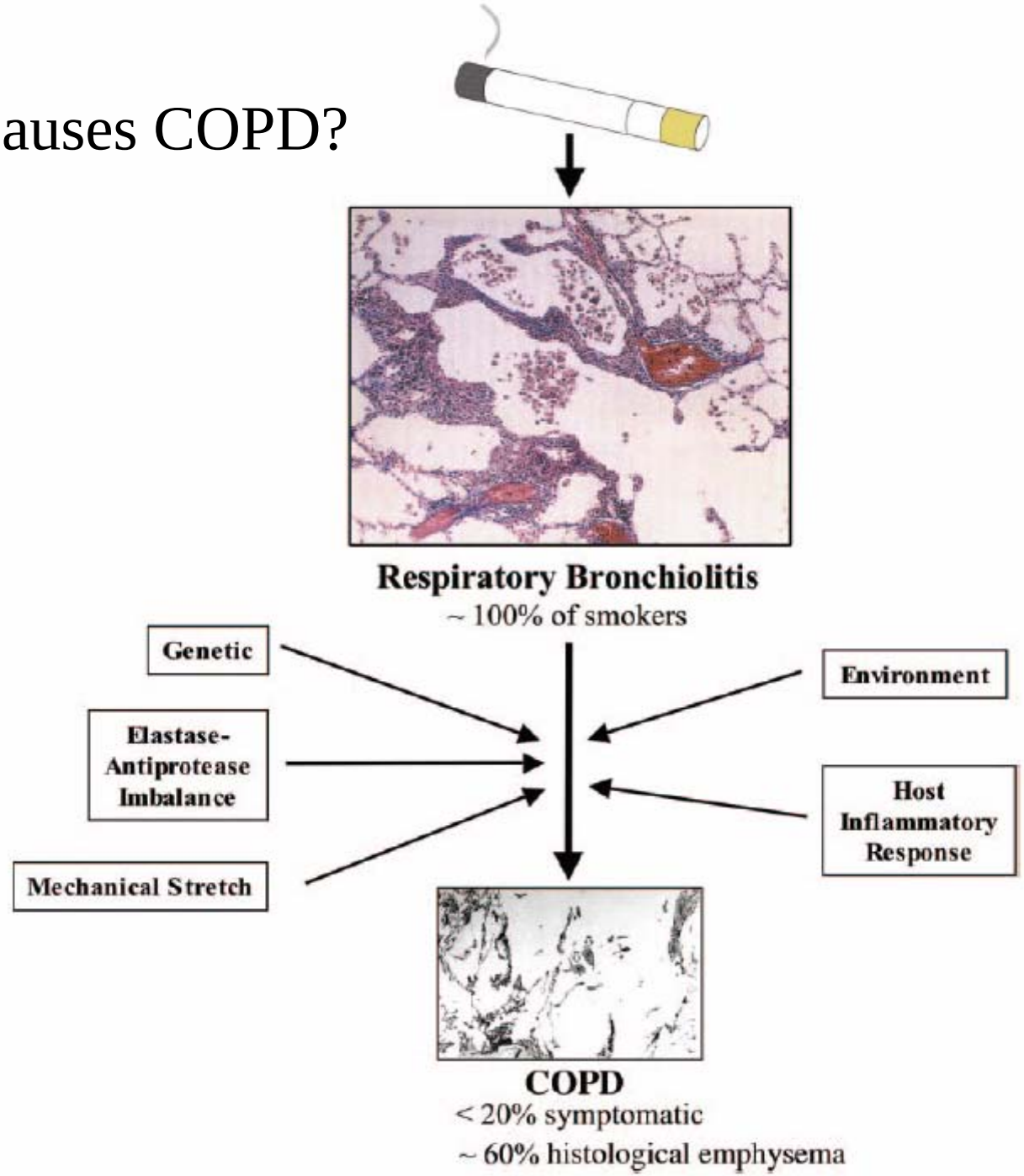
The cost of COPD

Among respiratory diseases, COPD is the leading cause of lost work days. In the EU, approximately 41,300 lost work days per 100,000 people are due to COPD every year. In Europe, productivity losses due to COPD amount to a total of €28.5 billion annually.

The future?

By 2020, COPD is likely to account for over 6 million deaths worldwide every year, making it the third leading cause of death.

what causes COPD?



effect of COPD on total mortality

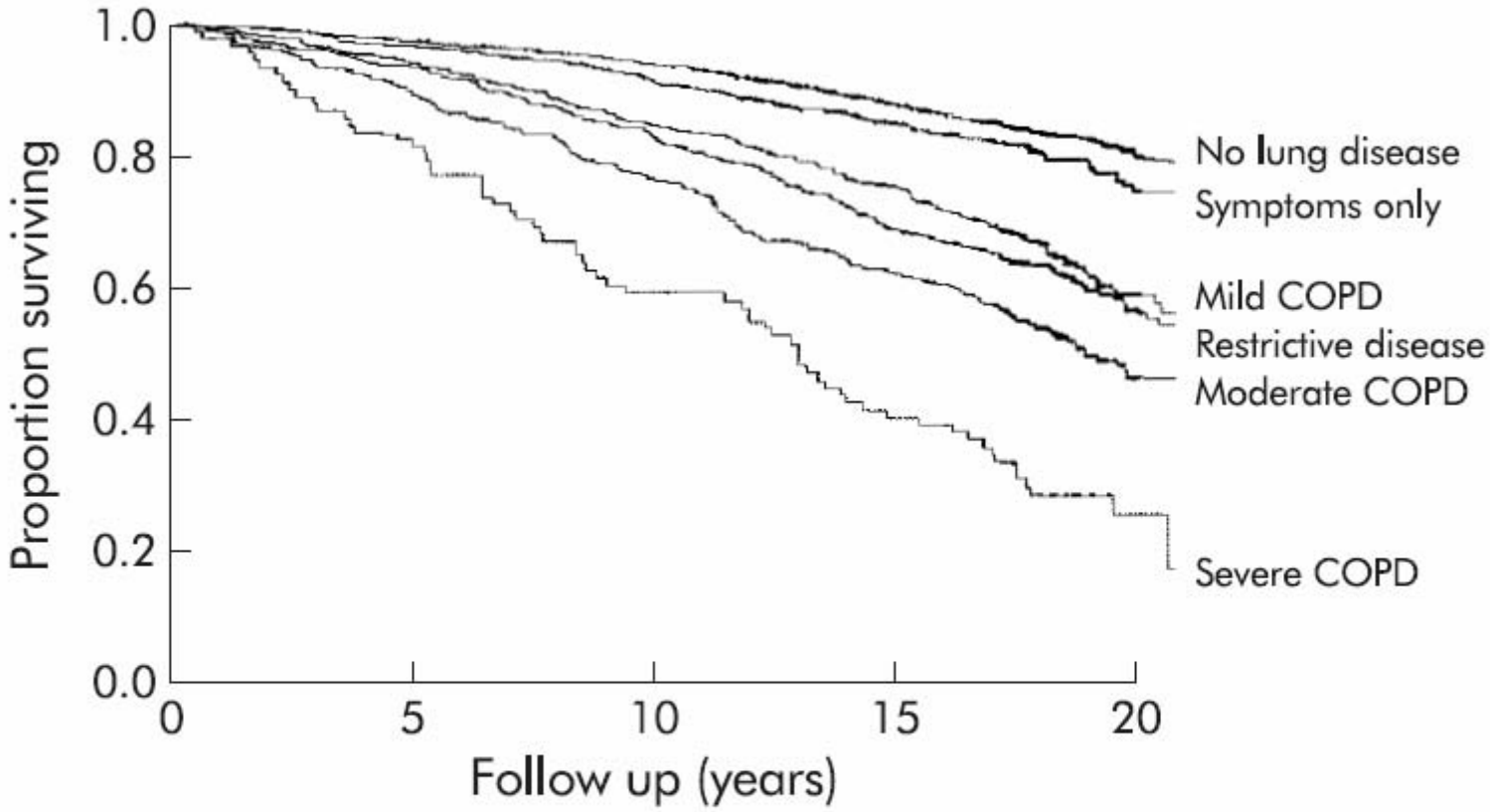


Figure 1 Kaplan-Meier curve for death among 5542 participants stratified by degree of lung function impairment (number available for follow up at each time interval is shown in table 4). From the National Health and Nutrition Examination Survey 1971–5 and follow up to 1992.

What causes COPD?

The most important cause of COPD (both bronchitis and emphysema), in about 80% - 90% of cases, is cigarette smoking.

Smoking and genetics

Not all smokers develop COPD, suggesting that genetic factors also have an influence on each individual's risk. The only proven genetic risk factor for COPD is hereditary deficiency of a protein termed α1-antitrypsin. People with this deficiency who smoke may develop COPD in early adult life.

Pollution

The role of outdoor air pollution as a cause of COPD is unclear, but urban air pollution is harmful to individuals with this condition.
Occupational factors that cause intense or prolonged exposure to dust, chemicals and vapours, etc., can result in COPD, whether a person smokes or not, and increase the risk of the disease in smokers. Indoor air pollution from biomass fuel has also been implicated as a risk factor for the development of COPD.

Passive smoke

Passive exposure to cigarette smoke also contributes to respiratory symptoms and reduced lung function in schoolchildren. In later life, this may lead to COPD.

Chronic obstructive pulmonary disease (COPD) and occupational exposures

Journal of Occupational Medicine and Toxicology 2006,

Abstract

Chronic obstructive pulmonary disease (COPD) is one of the leading causes of morbidity and mortality in both industrialized and developing countries.

Cigarette smoking is the major risk factor for COPD. However, relevant information from the literature published within the last years, either on general population samples or on workplaces, indicate that about 15% of all cases of COPD is work-related.

Specific settings and agents are quoted which have been indicated or confirmed as linked to COPD. Coal miners, hard-rock miners, tunnel workers, concrete-manufacturing workers, nonmining industrial workers have been shown to be at highest risk for developing COPD.

American Thoracic Society

Am J Respir Crit Care Med Vol 167. pp 787–797, 2003

TABLE 3. LUNG FUNCTION IMPAIRMENT:

Reference Number	Type of Study	Age Range	Sex	Number of Subjects/ Number of Cases
(108)	Population study of six cities in the United States	25–74	M/F	8515/137
			M/F	8515/135
(118)	Population study of Po Delta area in North Italy	18–64	M	763/180
(149)	Population study of four areas in New Zealand (phase of ECRHS survey)	20–44	M/F	1132/24
(113)	Population study of five Spanish areas (phase of ECRHS survey)	20–44	M/F	1735/34
(156)	Population study of Tucson area	>18	M	1195/96

Definition of abbreviations: ECRHS = European Community Respiratory Health Survey;

POPULATION ATTRIBUTABLE RISK CAUSED BY OCCUPATION

Lung Function	Type of Exposure	PAR% Reported	PAR% Calculated 1	PAR% Calculated 2
$FEV_1/FVC < 60\%$	Dusts	34	14	14
	Gases, fumes	12	NS	
$FEV_1/FVC < 70\%$ or $FEV_1 < 70\%$	Dusts, gases, fumes		9	12
$FEV_1/FVC < 75\%$ and chronic bronchitis symptoms	Dusts, gases, fumes	19	56	55
$FEV_1/FVC < 70\%$	High mineral dusts		19	35
FVC < 75% predicted or FEV1/FVC ratio < 80% predicted	Dusts, gases, fumes		19	19

NS = not significant; PAR% = magnitude of the population attributable risk.

Nonetheless, a value of 15% is a reasonable estimate of the occupational contribution to the population burden of COPD.

TABLE 3. COPD* by industry, Third National Health and Nutrition Examination Survey subjects aged 30–75 years, United States, 1988–1994 *Am J Epidemiol* 2002;156:738–746

Industry	All subjects (n = 9,823)†					
	*N**	P* (%) (SE*)	*n**	COPD§	OR*,¶	95% CI*
Rubber, plastics, leather manufacturing	9.0	14.8 (3.9)	71	9	2.5	1.4, 4.4
Utilities	12.0	16.7 (6.0)	94	12	2.4	0.7, 7.7
Office building services (males)	5.4	13.6 (6.6)	84	12	2.4	0.7, 7.8
Textile mill products manufacturing	15.4	15.3 (4.6)	163	20	2.2	1.1, 4.2
Armed forces	14.0	13.3 (3.5)	119	16	2.2	1.2, 3.9
Food products manufacturing	21.0	13.9 (3.0)	267	27	2.1	1.1, 4.1
Repair service, gas station	29.6	12.0 (3.0)	256	22	1.5	0.8, 2.9
Chemicals, petroleum, coal manufacturing	13.7	11.6 (4.1)	107	10	1.5	0.5, 4.4
Agriculture	39.0	10.4 (1.9)	598	60	1.5	0.8, 2.7
Sales	136.5	8.3 (1.2)	1,012	83	1.4	0.9, 2.2
Construction	54.3	8.7 (2.0)	493	46	1.3	0.8, 2.3
Transportation and trucking	35.9	8.9 (2.2)	325	32	1.2	0.8, 2.0
Personal services	40.3	6.1 (1.6)	543	41	1.1	0.6, 2.0
Health care	74.4	4.4 (1.3)	653	27	1.1	0.6, 2.1
Other industries#	201.5	6.1 (0.8)	2,090	124	0.8	0.6, 1.2
Office workers	401.0	4.7 (0.4)	2,653	133	1.0	
Total working population**	1,103	6.9 (0.4)	9,528	674		

The fraction of COPD attributable to work was estimated as 19.2% overall

change of FEV_1 over time

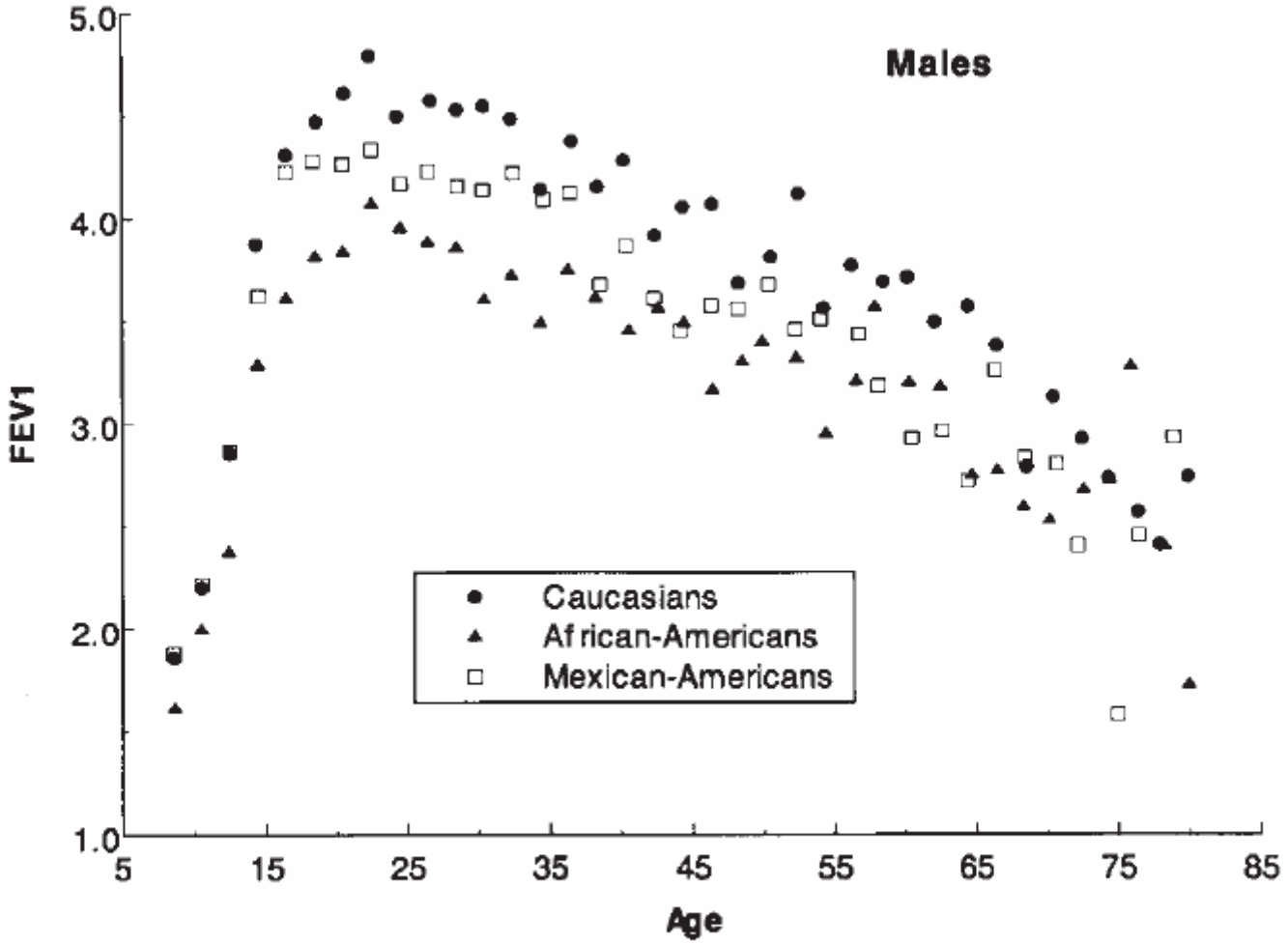


Figure 1. Mean FEV_1 versus age (2-yr increments) for male subjects.

maximum of about 5 l
between 20 – 25 years

decline of 30 ml per year

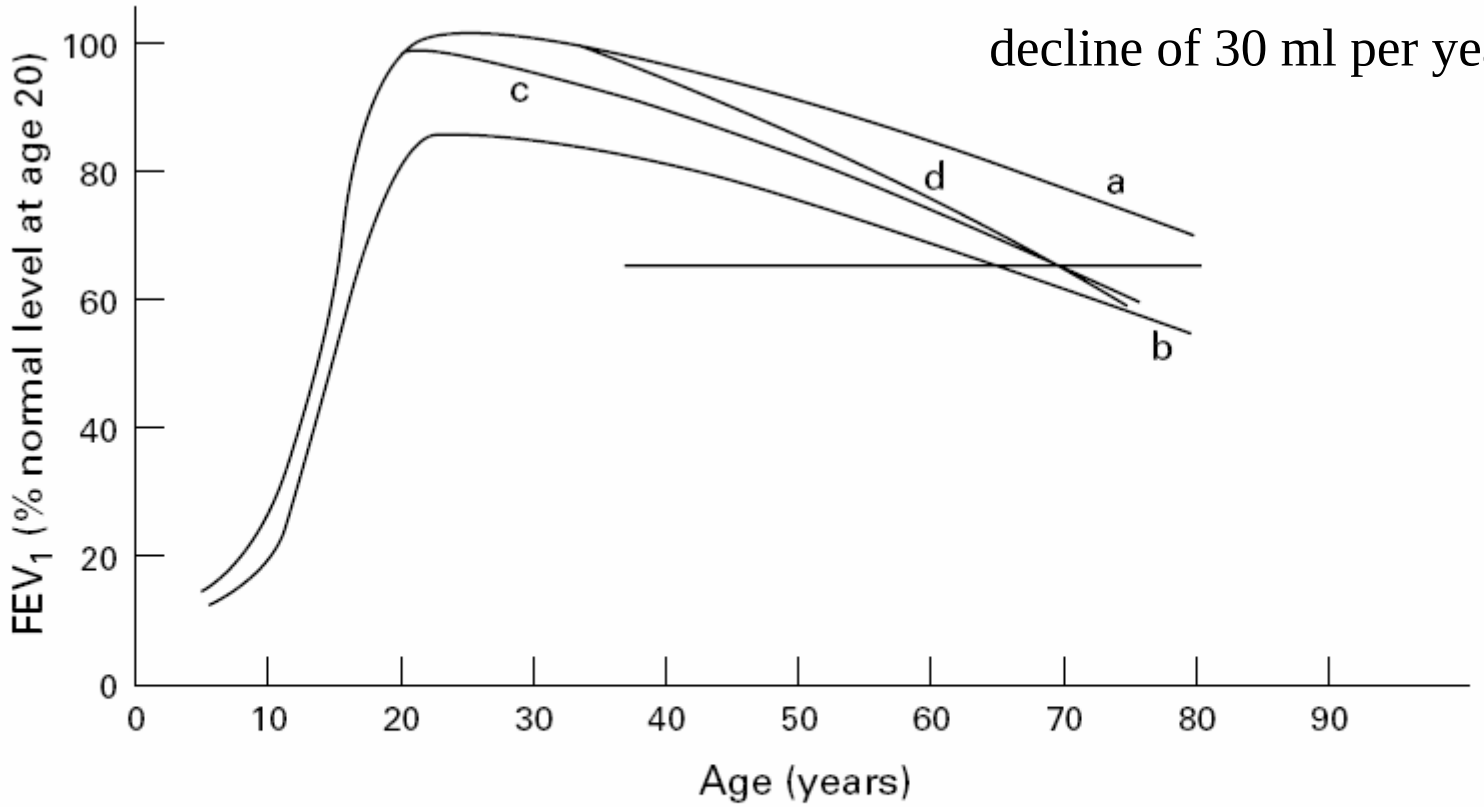
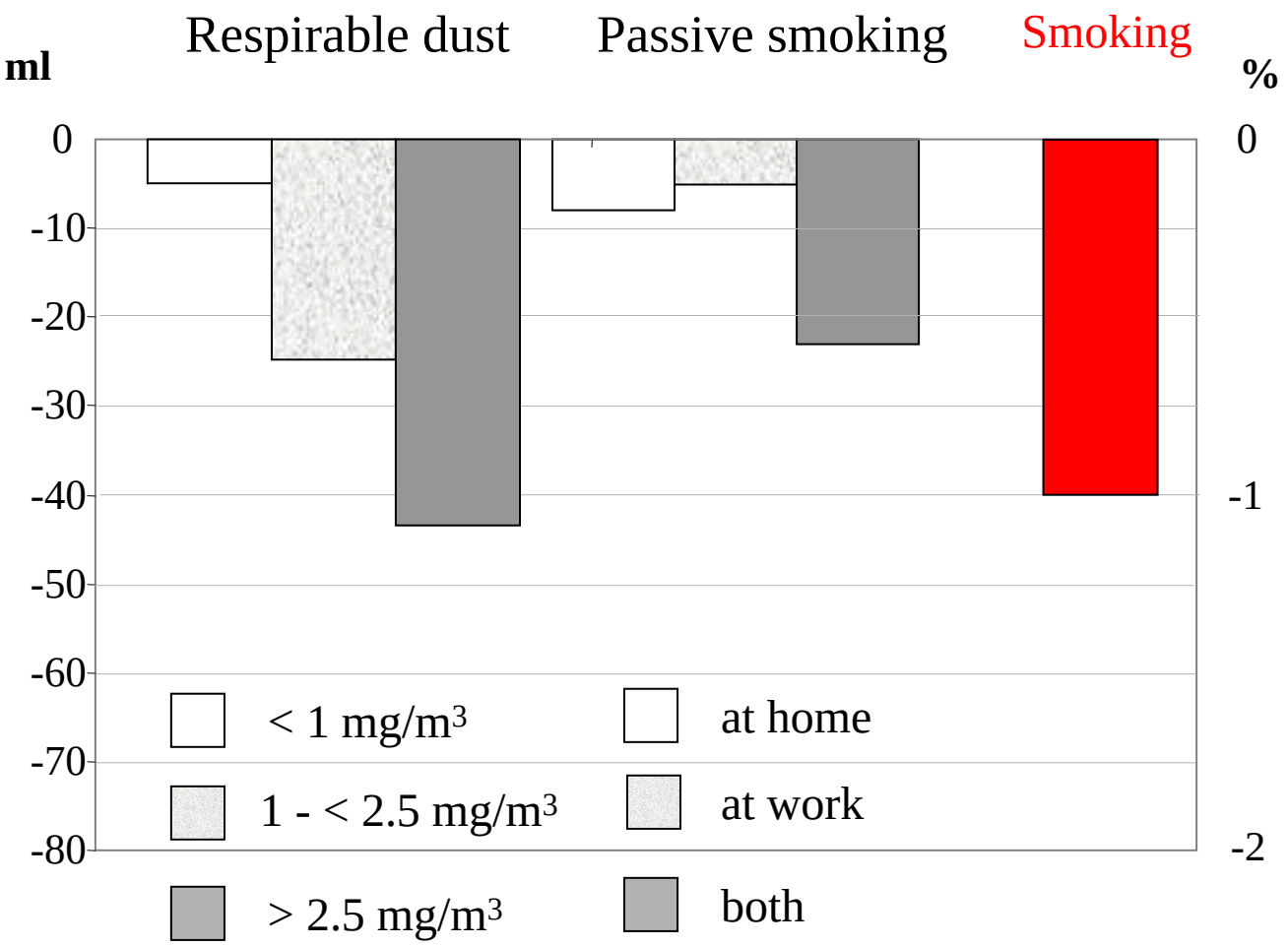


Figure 1 FEV_1 plotted as a percentage of maximal at age 20 years against age. Line a = healthy normal subjects, line b = submaximal growth but normal decline, line c = premature or early decline, line d = an accelerated decline in lung function compared with normal subjects (line a). In real life more than one mechanism for a low level of FEV_1 in adult life can be operating in any one individual. Figure reproduced with permission from Weiss and Ware.[13]

Decline of FEV_1 per year



role of silica dust

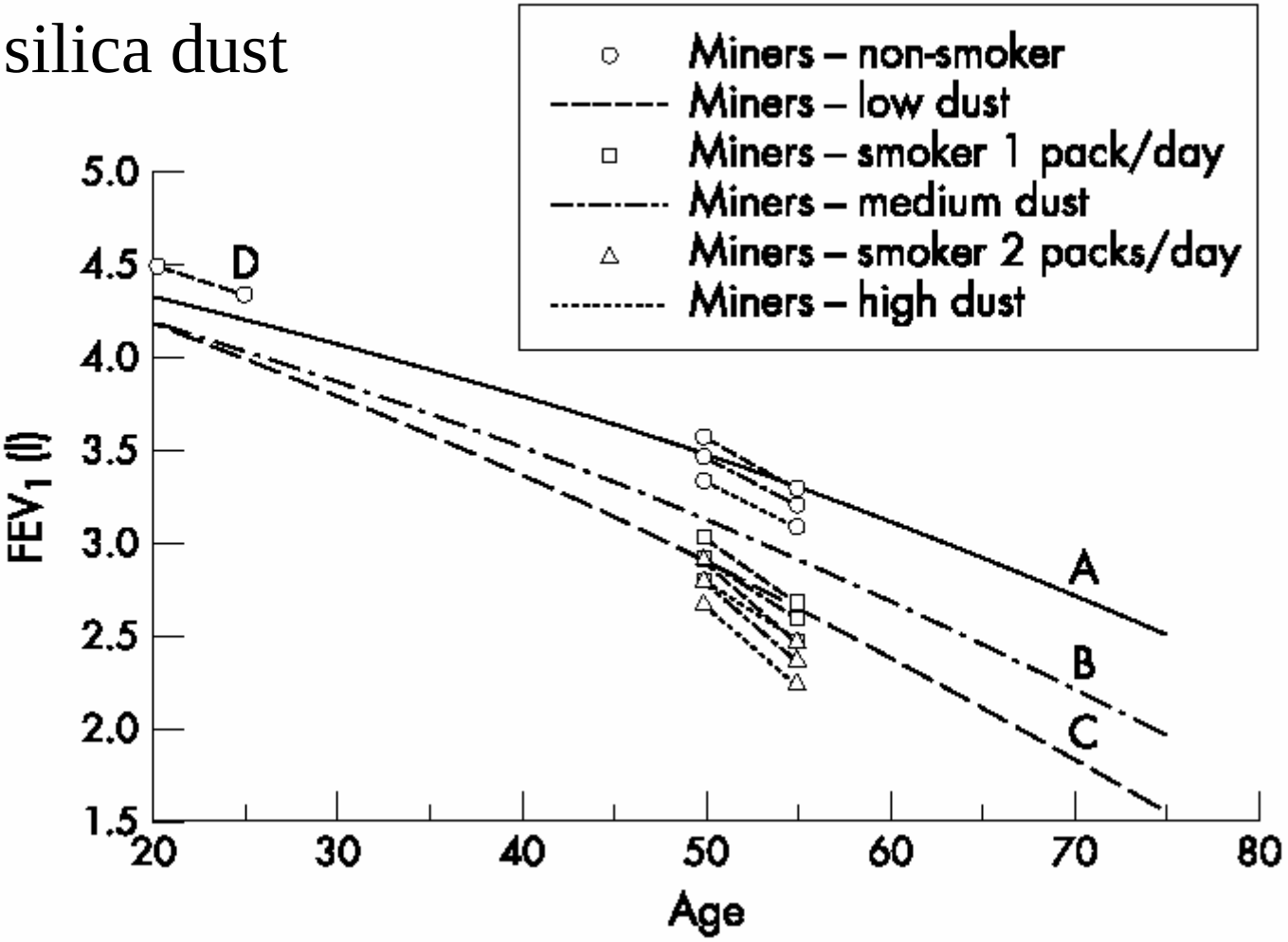
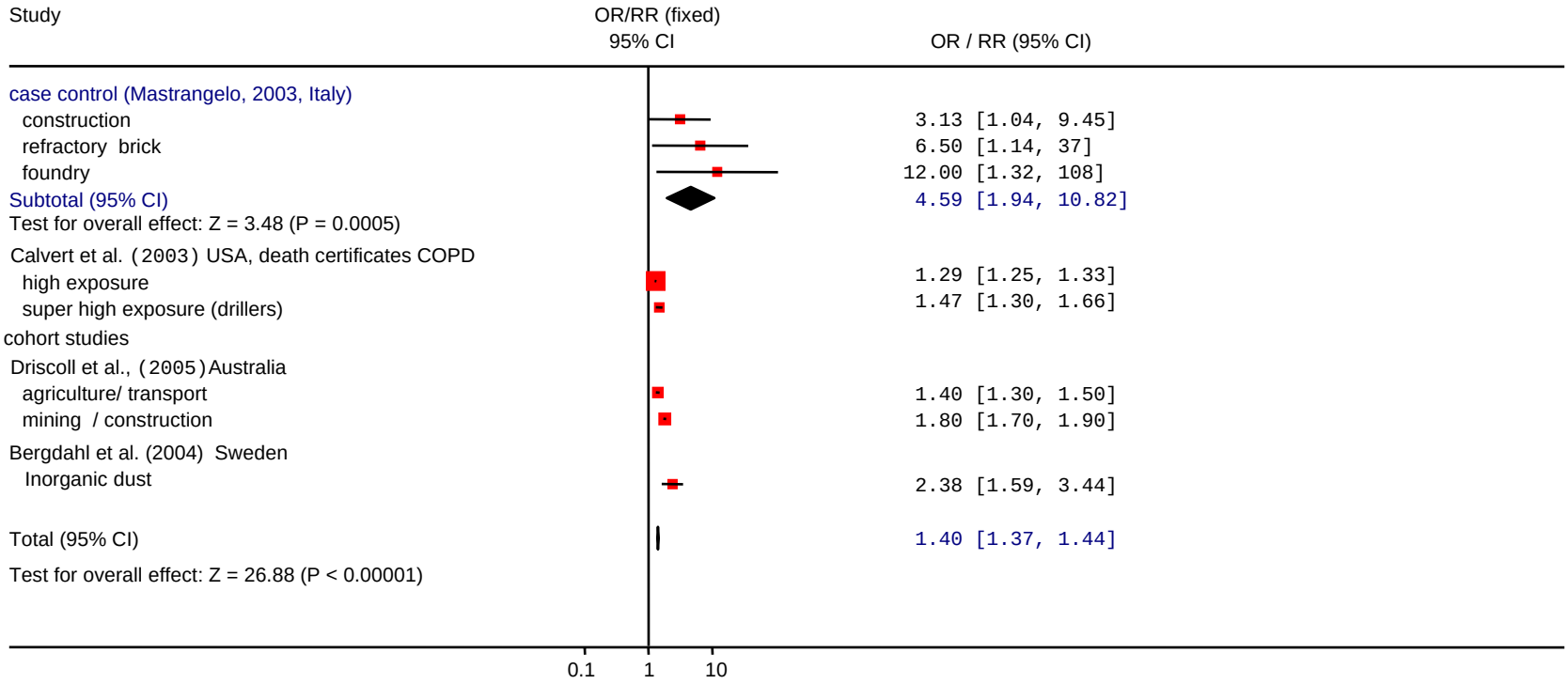
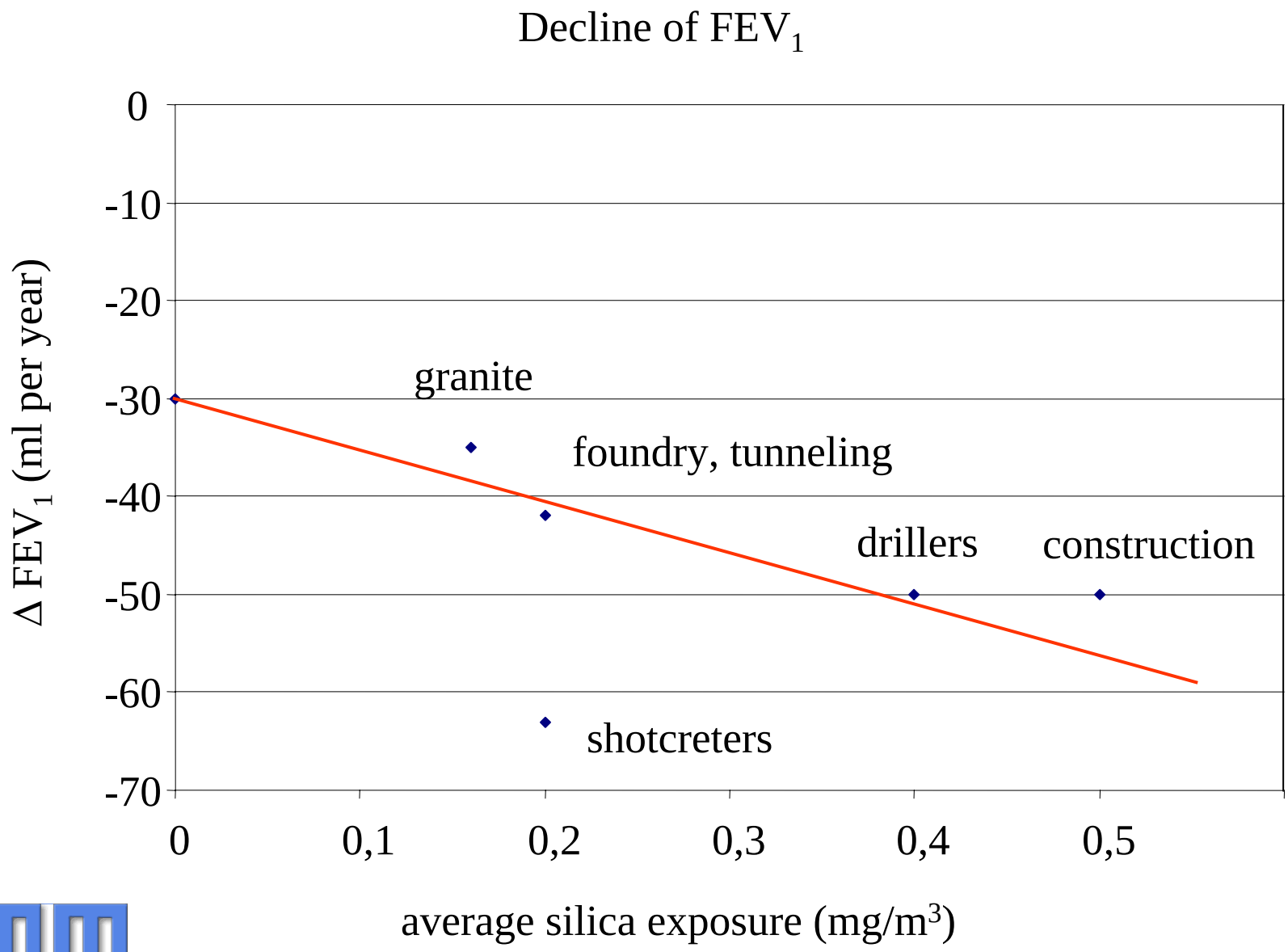


Figure 1 Predicted values for gold miners, according to levels of dust exposure and smoking habits, projected onto predicted curves for general white male population. A, non-smokers; B, current smokers of one pack a day; C, current smokers of two packs a day. The D curve shows the mean observed FEV_1 values for 20 and 30 year old white South African miners, smokers and non-smokers,

Review: COPD and occupation with silica dust



increased risk for silica dust


Decline of FEV1
0
-10
-20
-30
-40
-50
-60
-70
Δ FEV1 (ml per year)
granite
foundry, tunneling
drillers
construction
shotcreters
0
0,1
0,2
0,3
0,4
0,5
average silica exposure (mg/m3)

areas for actions: prevention

-primary prevention:

is designed to abate hazards before any damage or injury has occured.

Control of the exposure:

Reduction, elimination, personal protective equipment

Smoking is the major cause of COPD:

Smoking cessation at the workplace

and discourage workers from smoking also outside the workplace.

Particulate matter from tobacco versus diesel car exhaust: an educational perspective

G Invernizzi, A Ruprecht, R Mazza, E Rossetti, A Sasco, S Nardini and R Boffi

Tob. Control 2004;13;219-221

Background: Air pollution is a common alibi used by adolescents taking up smoking and by smokers uncertain about quitting. However, environmental tobacco smoke (ETS) causes fine particulate matter (PM) indoor pollution exceeding outdoor limits, while new engines and fuels have reduced particulate emissions by cars. Data comparing PM emission from ETS and a recently released diesel car are presented.
Methods: A 60 m^3 garage was chosen to assess PM emission from three smouldering cigarettes (lit sequentially for 30 minutes) and from a TDCi 2000cc, idling for 30 minutes.

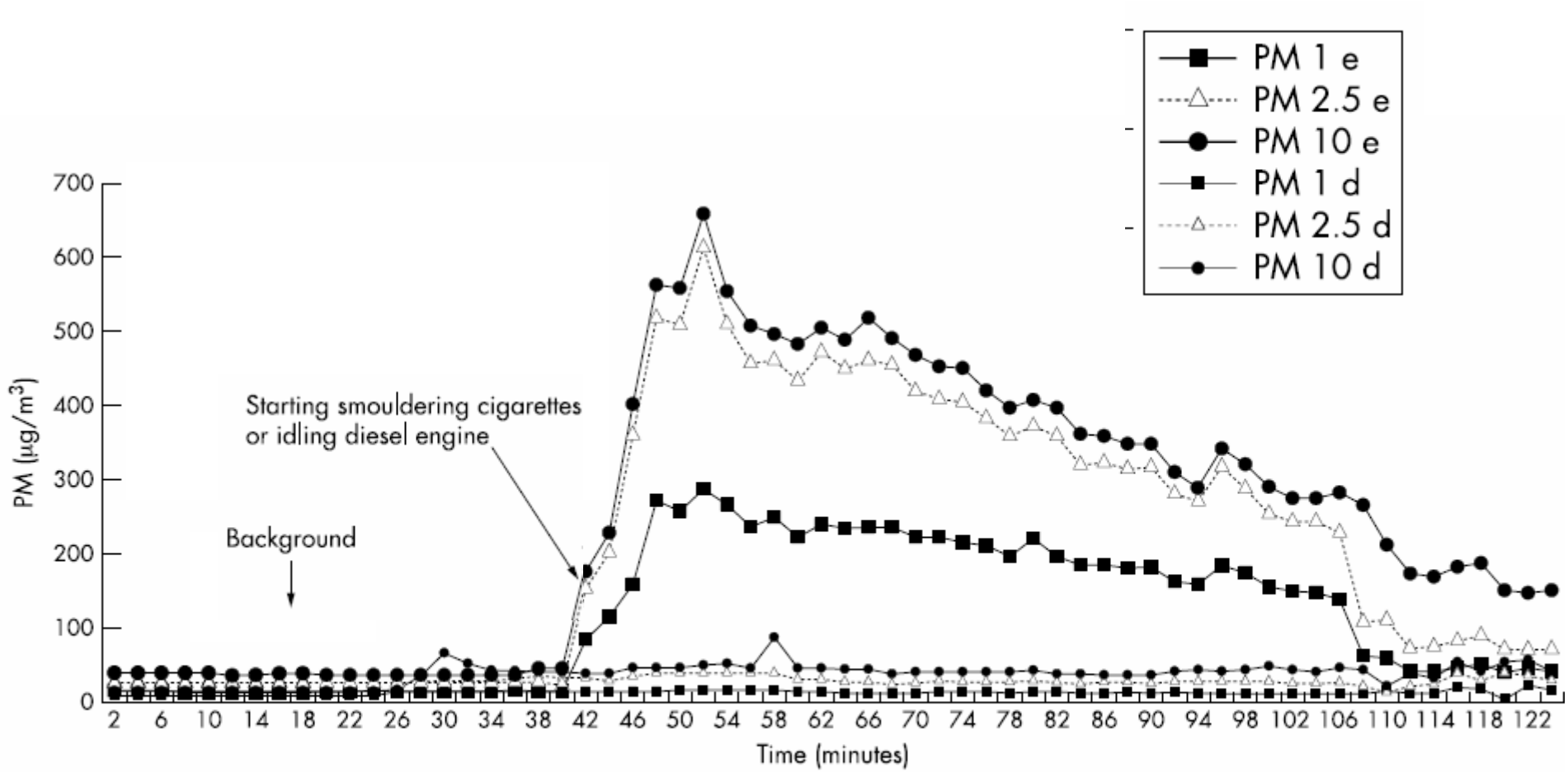


Figure 1 Particulate matter (PM) production from environmental tobacco smoke (e) and an ecodiesel engine (d) (three smouldering cigarettes or an idling engine for 30 minutes in a 60 m^3 garage).

-secondary prevention:

early detection of the disease, lung function tests,

proportion of undiagnosed COPD may be high

Early detection of COPD:

A case finding study in general practice

Respiratory Medicine (2007) 101, 525–530

Summary

Objectives: To estimate the prevalence of undiagnosed chronic obstructive pulmonary disease (COPD) in a population of general practice patients at risk for developing COPD. A further aim was to evaluate the presence of respiratory symptoms as a predictor for the diagnosis of COPD.

Methods: This study was conducted by eight general practitioners (GP) in six semi-rural general practices. During two consecutive months all patients attending their GP were included if they met the following criteria: current smokers between 40 and 70 yr of age, and a smoking history of at least 15 pack-years.

Table 2 Prevalence of COPD in a group of 146 general practice patients, aged 40–70 yr, with a smoking history of at least 15 pack-years.

Known COPD 25 (17.1%)	No previous diagnosis of COPD 121 (82.9%)						
	Newly detected COPD[a] 43 (29.5%)				Non COPD 78 (53.4%)		
	Stage I[a]	Stage II[a]	Stage III[a]	Stage IV[a]	Normal	Small airways disease	Restrictive pattern
	18 (12.3%)	21 (14.4%)	4 (2.7%)	0	45 (30.8%)	26 (17.8%)	7(4.8%)

[a]Diagnosis and assessment of severity of COPD according to the GOLD criteria.[2]

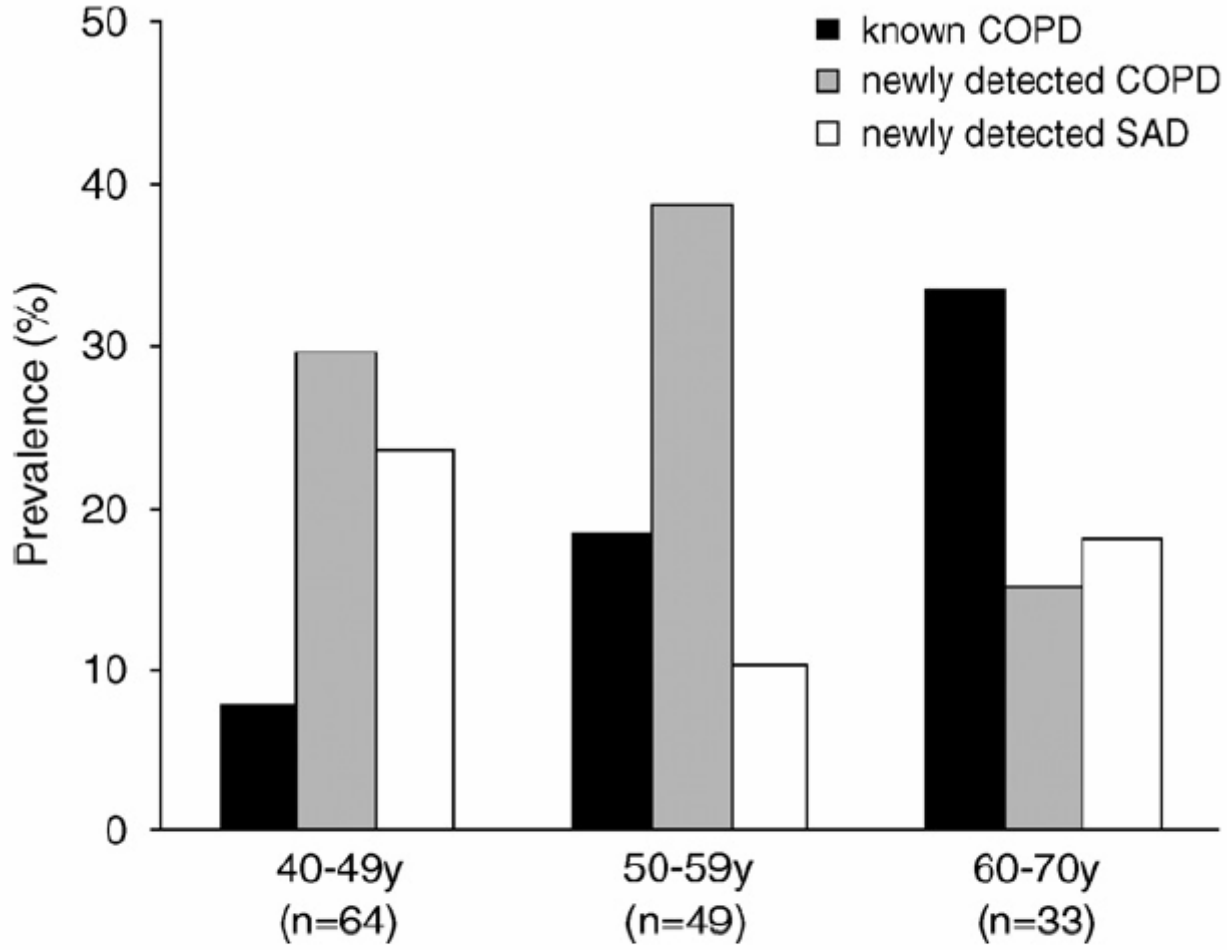


Figure 1 Prevalence of known chronic obstructive pulmonary disease (COPD), newly detected COPD and newly detected small airways disease (SAD) in a high-risk population (total $n = 146$): dependence on age category.

-tertiary prevention:
prevention of permanent COPD, appropriate health care, treatment

Salmeterol and Fluticasone Propionate and Survival in Chronic Obstructive Pulmonary Disease

N Engl J Med 2007;356:775-89.

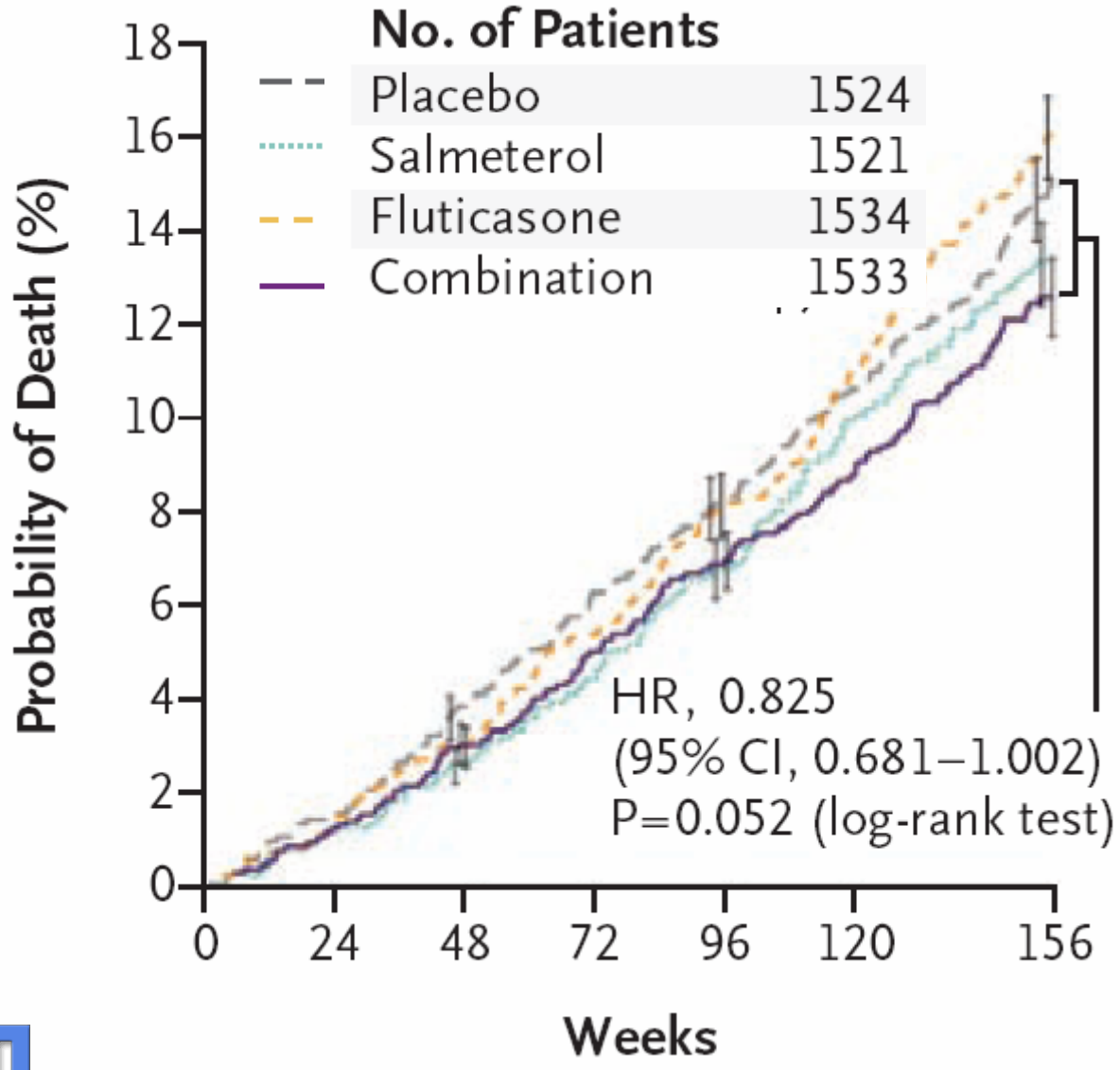
BACKGROUND

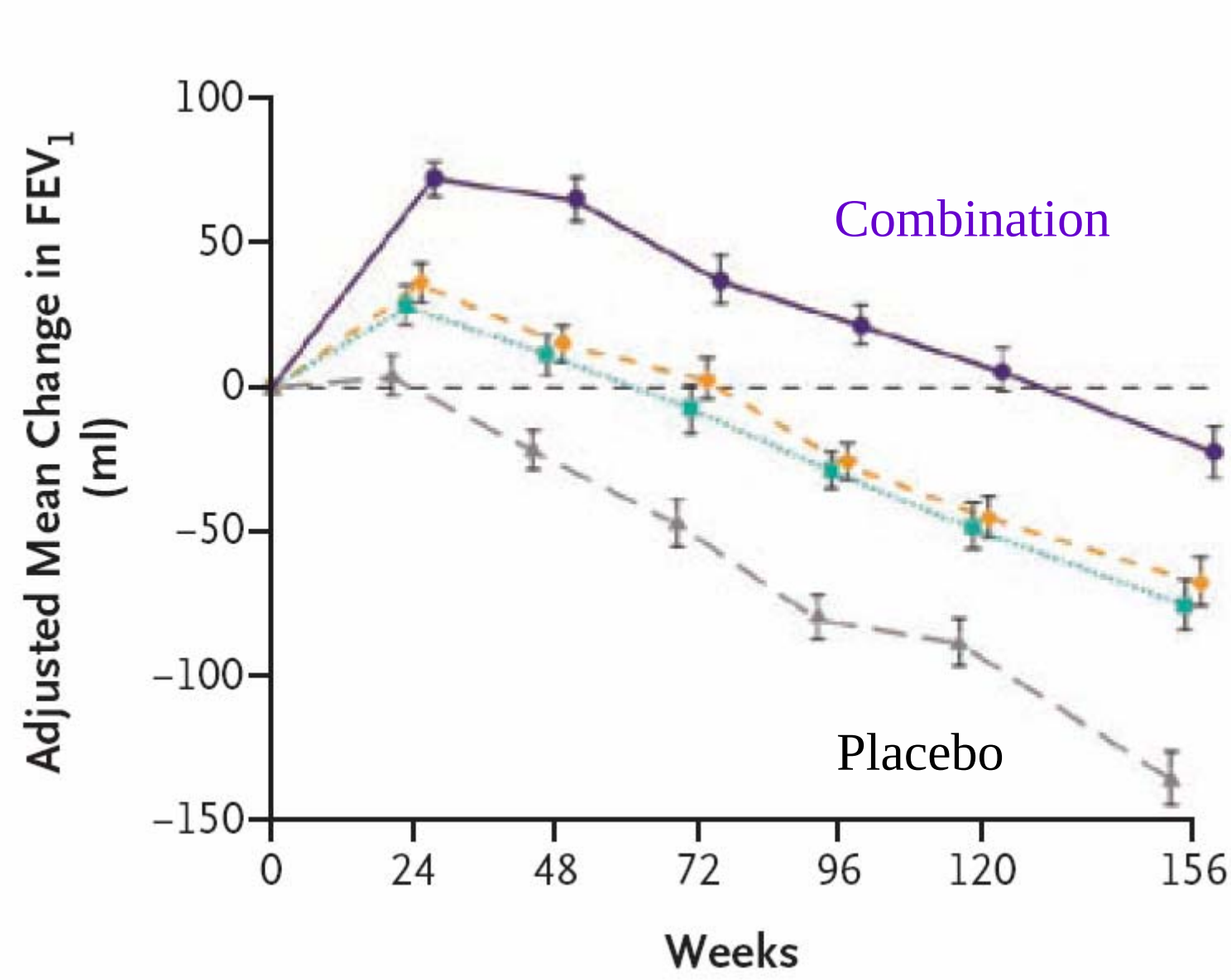
Long-acting beta-agonists and inhaled corticosteroids are used to treat chronic obstructive pulmonary disease (COPD), but their effect on survival is unknown.

METHODS

We conducted a randomized, double-blind trial comparing salmeterol at a dose of 50 μg plus fluticasone propionate at a dose of 500 μg twice daily (combination regimen), administered with a single inhaler, with placebo, salmeterol alone, or fluticasone propionate alone for a period of 3 years. The primary outcome was death from any cause for the comparison between the combination regimen and placebo; the frequency of exacerbations, health status, and spirometric values were also assessed.

Death from Any Cause




Adjusted Mean Change in FEV_1 (ml)
100
50
0
−50
−100
−150
0
24
48
72
96
120
156
Weeks
Combination
Placebo

RESULTS

Of 6112 patients in the efficacy population, 875 died within 3 years after the start of the study treatment. All-cause mortality rates were 12.6% in the combination-therapy group, 15.2% in the placebo group, 13.5% in the salmeterol group, and 16.0% in the fluticasone group. The hazard ratio for death in the combination-therapy group, as compared with the placebo group, was 0.825 (95% confidence interval [CI], 0.681 to 1.002; P=0.052, adjusted for the interim analyses), corresponding to a difference of 2.6 percentage points or a reduction in the risk of death of 17.5%.

CONCLUSIONS

The reduction in death from all causes among patients with COPD in the combination-therapy group did not reach the predetermined level of statistical significance. There were significant benefits in all other outcomes among these patients.

Silica dust and COPD, is there an association?

Conclusion:

Yes, there is an association.

There are no signs of a threshold value for dust in order to prevent COPD.

Primary goal:

Prevention

a) dust control

b) smoking cessation

c) increase of spirometric testing

Thank you for your attention