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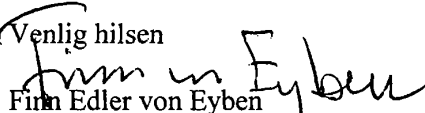
På vegne af Den Medicinske Forskningsenhed, Ringkøbing Amt, medvirkede jeg i planlægningen og gennemførelsen af en konference

Smoke Free Workplaces – improving the health and well-being of people

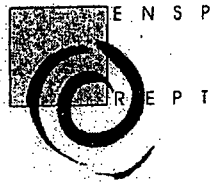
som European Network for Smoking Prevention med støtte fra Den Europæiske Kommission afholdt i Berlin 10-11 maj 1999.

Jeg sender bilagt mit kapitel i det hæfte, som blev publiceret i forbindelse med konferencen. Hele publikationen findes tilgængelig på ENSP hjemmeside, både på engelsk og på en række andre europæiske sprog. Således er mit kapitel senere blevet trykt i et spansk—sproget tidsskrift for sundhedsfremme.

Venlig hilsen


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DEUTSCHE
KREBSGESELLSCHAFT E.V.

EUROPEAN CONFERENCE

SMOKE FREE WORKPLACES

IMPROVING THE HEALTH AND WELL-BEING OF PEOPLE

BERLIN

MAY 10-11, 2001

UNDER THE PATRONAGE OF MR. DAVID BYRNE, MEMBER OF THE EUROPEAN COMMISSION

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Europa gegen den Krebs

European Status Report

**Working document for the European conference
“Smoke Free Workplaces:
Improving the health and well-being of people at work”**

Berlin, Germany – 10/11 May 2001

The final version of the report will be ready by September 15th 2001 and will be available in 10 EU-languages and Romanian from the ENSP-website (www.ensp.org)

SMOKE FREE WORKPLACES

Improving the health and well-being of people at work

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Executive Summary

Since 1986, 14 authoritative reports from respected medical and environmental institutions and government agencies in Europe, North America and Australia have established that passive smoking constitutes a public health hazard. Some Institutions, such as the USA National Institute on Occupational Safety and Health (NIOSH) have classified tobacco smoke as a recognized occupational carcinogen.

Environmental tobacco smoke (ETS) contains many chemicals, more than forty of which cause cancer in animals and humans. ETS also contains chemicals that cause irritation to the eyes and upper respiratory tract. The 'side-stream smoke', which is given off by a burning cigarette, contains higher concentrations of these chemicals than does the mainstream smoke' which is inhaled directly by the smoker.

Children are the most vulnerable victims of passive smoking. Exposure to ETS increases their risk of sudden infant death syndrome (SIDS), upper respiratory tract infections, bronchial asthma, and middle ear infections.

For adults, passive smoking increases the risk of lung cancer and coronary heart disease and cerebrovascular disease (stroke) in non-smokers.

The implications of long term exposure to ETS in the workplace have been increasingly recognised in Europe as in much of the rest of the world. All 15 Member States of the EU currently have some legislation on smoking in public places but few regulations specifically refer to workplaces. Regulations also refer to smoking restrictions in health premises, public buildings and public transport; of course, all of these places are also workplaces.

At the European level, two Directives deal directly, but very narrowly with workplace smoking. For example, a Directive of 1983 (83/477) stated that smoking should be banned in areas where the workers are exposed to asbestos dust. A Directive of 1989 (89/654) dealt with air in enclosed workplaces. In particular, it ruled that appropriate measures should be taken for the protection of non-smokers, for example in places where workers are allowed to rest.

Eight out of the 15 EU Member States have had litigation to protect non-smokers' rights: Denmark, France, Germany, Ireland, Italy, the Netherlands, Sweden and the United Kingdom. These cases often use existing legislation that obliges employers to provide a safe working environment to argue that forcing employees to work in smokey atmospheres constitutes a workplace health hazard.

No-smoking policies have been successfully implemented in workplaces of different sorts and sizes in many countries in Europe, North America and elsewhere. More than 20 years' experience with framing and implementing such policies has clearly highlighted the key principles for a successful policy. They include:

- o Focus on the nuisance of the smoke, not the smoker.
- o Focus on individuals' rights to a safe environment
- o Get the commitment and support of management
- o Provide opportunities for employees' participation in planning and implementing the policy
- o Provide training to managers to communicate and enforce the policy
- o Educate all staff about the hazards of workplace ETS
- o Ensure the restrictions and enforcement are equitable across all levels of the organisation
- o Allow plenty of time to go through the process of announcing, planning and implementing the policy
- o Offer smoking cessation support to employees and their families before and after the policy change
- o Continue to monitor the policy

Workplace smoking policies are a health benefit to smokers and non-smokers alike. Studies have shown that workplace smoking policies decrease workday cigarette consumption among smokers. Smokers employed in smoke free workplaces are also more likely to quit smoking than those working in places without a policy.

Absence from work is higher among smokers than non-smokers. Smokers miss work not only because of major diseases, such as heart or respiratory disease, but also because they are more liable to coughs, colds and 'flu.

A worker on sick leave costs employers the equivalent of double his salary. The costs of absenteeism include sick pay as well as loss of efficiency and production through understaffing or use of inexperienced temporary staff.

Other costs of workplace smoking to employers include extra payments for insurance, decorating and equipment that may be harmed by ETS. A workplace smoking policy can also give the company an improved image and decrease conflicts between smoking and non-smoking employees.

Planning and implementing a workplace smoking policy can in itself entail costs such as the expense of building and ventilating smoking rooms, buying in cessation programmes, and so on. However analyses of the long term benefits of such policies show that they are very sound economic investments and save money in the long run as well as provide other less tangible benefits such as improving employee morale and enhancing the company image.

1. Health risks from active and passive smoking

Finn Edler von Eyben, Medical Research Unit of Ringkoebing County (Denmark)

1.1 Introduction

Smoking in workplaces causes illness and death, is a nuisance for non-smokers, leads to lost productivity and causes extra costs. Smoke free workplaces can save money, time and life. These facts are the background for tobacco control at workplaces.

Tobacco smoke is the source for passive smoking. Epidemiological studies have shown that smoking causes illness that disable and kill. Other studies showed that passive smoking increases the risk of tobacco-caused illness. The National Institute for Occupational Safety and Health in the USA, the US General Surgeon, the National Research Council in the USA, the California Environmental Protection Agency and the American College of Occupational and Environmental Medicine reviewed the literature and concluded that passive smoking in workplaces is a health risk.¹⁻⁵ In 1999, the French Comité National Contre le Tabagisme made a review of the health effects of passive smoking.⁶

So there is a scientific base for action to reduce environmental tobacco smoke. Countries, however, vary in acting to protect non-smokers from being exposed to passive smoking against their will. The tobacco industry has opposed laws banning passive smoking because it fears that these laws reduce the social acceptability of smoking.

This chapter reviews and updates the health aspects from smoking using examples from Europe and workplaces. The review addressed the health risks from smoking in both men and women, the impact on median life expectancy, the gains from smoking cessation, the social pattern of smoking in Western societies and the social differences in smoking caused illness. Furthermore, the chapter describes measurements of Environmental Tobacco Smoke (ETS; see 1.4), immediate and long-term effects of passive smoking and detailed consequences of passive smoking in workplaces.

1.2 Effects of active smoking

1.2.1 Tobacco-caused illness

Smoking is the main cause of lung cancer, chronic obstructive pulmonary disease, and peripheral arteriosclerosis, and one of the most important causes of cardiovascular and cerebrovascular disease. Hospitals cure only few of these cases. Smoking also leads to other chronic diseases and raises the risk for illness over that of a non-smoker, especially at a young age. A current smoker aged 35 to 64 years has a higher risk of illness than a non-smoker (Table 1).⁷

Table 1. Risk of illness caused by tobacco.

Diagnosis	Relative risk at 35 – 64 years of age		Risk attributed to current smoking (%)	
	Current smoking	Previous smoking	Men	Women
Lip/mouth/pharynx cancer	2.9	2.0	50	48
Esophagus cancer	3.4	2.0	54	52
Larynx cancer	5.4	2.5	67	66
Lung cancer	11.5	5.0	83	83
Pancreas cancer	1.6	1.0	17	17
Kidney cancer	1.5	1.0	15	15
Urinary bladder cancer	2.5	1.5	41	29
Chronic obstructive pulmonary disease, bronchitis/emphysema	8.7	4.0	79	78
Other heart diseases	1.6	1.2	21	20
Cerebrovascular disease	2.4	1.3	37	36
Hypertension	1.6	1.2	21	20
Atherosclerosis	4.1	1.0	52	52
Pneumonia	1.8	1.3	27	26
Bronchial asthma	1.8	1.3	27	26

Source: Kjørstad: Liability of tobacco industry, Norway 2000.⁷

Current smoking increases the risk of getting lip, mouth and pharynx cancer with 2.9. Some 50% of the risk of lip, mouth and pharynx cancer in men is ascribed to smoking. About 30% of all deaths before the age of 70 in Western Europe is caused by smoking.⁹ Smokers have a 11 times higher risk of getting lung cancer than non-smokers and smoking causes 83% of all cases of lung cancer in men. Before 1920, lung cancer was a rare disease whereas now it is the cause of most cancer deaths in men in Western countries. Three European studies from 1940-1955 pointed out that smoking causes lung cancer.⁹⁻¹¹ Many other studies have confirmed this link. In Denmark, the rise in use of cigarettes from 1950 to 1980 was followed by a rise in the number of lung cancers fifteen years later.¹² The link between consumption of cigarettes and number of lung cancers explains why more Danes had lung cancer in the period 1950 - 2000 despite lower current smoking rates. In the USA, changes in lung cancer incidence followed regional changes in number of smokers.¹³

Table 2. Smoking prevalence in women (1980) and lung cancer incidence in women (1995).

	Smoking prevalence in 1980 (%)	Lung cancer incidence in 1995*
Austria	14	14
Belgium	28	14
Denmark	44	41
Finland	18	10
France	24	8
Germany	21	13
Greece	25	11
Ireland	32	26
Italy	17	12
The Netherlands	34	19
Norway	25	18
Portugal	12	7
Spain	17	6
Sweden	23	17
United Kingdom	37	31

Source: Joossens and Sasco: Smoking and women. ENSP conference report, 1998.¹⁴

* Lung cancer incidence is an age-adjusted incidence per 100,000 women per year.¹⁵

Women's smoking is also closely linked to lung cancer. Comparing all Western European countries, in 1980 the number of smokers among women in a country was followed in 1995 by the number of lung cancers in women in the country (Table 2).^{14,15} The prevalence of smoking in women correlated with the incidences of lung cancer. The Spearman rank correlation coefficient for the link between smoking prevalence and lung cancer incidence in women was 0.78 ($p = 0.001$). So the difference in the prevalence of smoking between the countries caused 61% of the difference in number of lung cancer in women. Danish women smoked more than women in other Western European countries in 1980 and had lung cancer more often in 1995. Countries with low smoking prevalence among women had few cases of female lung cancer.

Smoking also increases the risk for chronic obstructive pulmonary disease and bronchitis (with or without emphysema) 8.7 times for smokers and causes 79% of all cases. The death rate from chronic obstructive pulmonary disease and bronchial asthma is higher in Denmark - both for men and women - than in other Western European countries.¹⁶ In recent years, clinicians found disability due to poor lung function both in old patients and in middle aged women. Some 5% of adult Danes are disabled due to chronic obstructive pulmonary disease.

Smoking is also the main cause of poor circulation of blood to arms and legs, peripheral arteriosclerosis. This disease leads more often to disability than to death of the patients.

Smoking is one of several major risk factors for cardiovascular and cerebrovascular disease. The information on risk factors is based on studies that followed groups of individuals over years. The Seven Countries Study studied more than 12,500 men aged 40 - 59 years in the late 1940s for risk factors for heart disease in Finland, Greece, Italy, the Netherlands, Yugoslavia, the USA and Japan.¹⁷ In a follow-up, smoking and other risk factors such as high blood pressure and high cholesterol, increased the risk of later cardiovascular disease (coronary heart disease). Other life style risk factors are dietary habits with a high consumption of saturated fat and lack of physical activity. Half the patients with cardiovascular disease had an acute myocardial infarction (heart attack). The impact of smoking on cardiovascular disease depends on how much the smoker has smoked and on whether the smoker inhaled the smoke.^{18,19} Overall, 30% of all cases of acute myocardial infarction are caused by smoking. The

group under 40 years of age had the highest risk of acute myocardial infarction from smoking.²⁰⁻²² For this age group, smoking caused more than 50% of the cases. Heavy smokers may have the first acute myocardial infarction as young as 25 years of age. Smoking also increases the risk of suicide.^{23,24} It also increases the risk of non-fatal diseases, such as goiter,^{25,26} and osteoporosis.²⁷ Smoking increases the risk of peptic ulcers to 3 times that of non-smokers.²⁸ Several of these diseases cause serious disability.

1.2.2 Gender differences in tobacco-caused illness

Smoking impairs the health of both men and women. Early in the 20th century, more men than women smoked and men had lung cancer and cardiovascular disease more often than women. A recent Danish thesis however, showed that smoking causes a higher risk of lung cancer and acute myocardial infarction in women than in men.²⁹

1.2.3 Tobacco-caused decrease in median life expectancy

The tobacco-caused diseases have a large impact on the life expectancy in Western societies. The four leading causes of death are cardiovascular disease, cancer, cerebrovascular disease and chronic obstructive pulmonary disease. 85% - 95% of patients with lung cancer die from lung cancer. Worldwide, smoking causes 3 million deaths per year. This number will increase to 10 million per year within the next 30 years.

A study of nearly 35,000 British male physicians showed the link between smoking, illness and death.³⁰ Smokers lived eight years less than non-smokers and the difference was largest for heavy smokers. The same differences were found in a Danish population followed for 30 years.³¹ Thus the finding is repeated in different countries and different groups in society.

Danes smoke more than Swedes, and Denmark has a higher death rate from tobacco-caused diseases (2,4/1000 persons) than Norway and Sweden (0,9/1000 persons). Smoking in the past effects the death rate, so smoking prevalence is only in part related to the death rate the same year. In EU countries in 1995, the proportion of men from 35 - 69 years of age who died of smoking-caused illness linked only weakly to the consumption of cigarettes per person per year (Table 3).^{8,32,33}

Table 3. Consumption of cigarettes in Western European countries in 1995 and fraction of deaths of smoking-caused disease in men 35 - 69 years.

Country	Cigarettes per person per year	Fraction of smoking-caused deaths (%)
Austria	2073	26
Belgium	2428	40
Denmark	1919	31
Finland	1351	21
France	2058	33
Germany	1702	31
Greece	4313	31
Ireland	2238	30
Italy	1813	36
The Netherlands	2088	36
Norway	725	20
Portugal	2079	21
Spain	2364	34
Sweden	1202	16
Switzerland	2932	31
United Kingdom	1733	30

Source: Reference books.^{8,32,33}

In Austria, 26% of the deaths in men 35 - 69 years of age was due to smoking. The weak relation, however, does not preclude gaining years of life expectancy from lower cigarette consumption. On the contrary, follow-up studies of countries showed that changes in cigarette consumption over the years had an impact on smoking-caused deaths in middle-aged men. In the period 1970 - 1995 in the Western European countries, the change in number of cigarettes smoked per person per year closely related to the change of smoking-related deaths in men from 35 - 69 years of age (Table 4).^{8,32,33,34}

Table 4. Change in consumption of cigarettes in Western European countries 1970 - 1995 and in deaths of smoking-caused illness of men aged 35 - 69 years

Country	Change in consumption of cigarettes per person per year (%)	Change in number of smoking-caused deaths (%)
Austria	88.8	66.1
Belgium	78.3	77
Denmark	101.7	100
Finland	68.8	46.7
France	112.8	110.3
Germany	72.2	90.3
Greece	152.3	160
Ireland	101.4	76.2
Italy	100.3	101.4
The Netherlands	100.7	86.8
Norway	93.6	108.3
Portugal	152.5	195
Spain	108.2	175
Sweden	68.7	81.8
Switzerland	82.3	85.7
United Kingdom	59.0	43.1

Source: The table shows the status in 1995 as percentage of the status in 1970.^{8,32-34}

Consumption of cigarettes per person a year decreased 11.2% in Austria from 1970 to 1995, and the number of deaths in men aged 35 - 69 years decreased 33.9%. The rate of smoking-caused deaths in a country rose as the use of cigarettes increased. There were fewer tobacco-caused deaths in middle aged individuals as the use of cigarettes in countries declined.³⁴ The Spearman rank correlation coefficient was high, 0.79 ($p < 0.0005$). So the change in consumption of cigarettes in a country caused 62% of the change seen in tobacco-caused deaths for young adult men.

During the period 1970 - 1990, the median life expectancy in Denmark increased less than in other OECD countries. Median life expectancy in Denmark remained short in

the 1990s. This period also showed that smoking played an increasing role for the short median life expectancy of the Danes. Sweden had the lowest proportion of smokers in the EU and a longer median life expectancy (Table 5).

Table 5. Median life expectancy in men and women in Sweden and in the EU

	Median life expectancy (years)			
	Men		Women	
	Sweden	EU	Sweden	EU
1979	72.6	70.6	78.3	77.3
1983	74.6	72.9	80.6	79.6
1996	76.7	74.4	81.0	81.0

Source: Public reviews of the Swedish state.¹³⁹

Tobacco-caused mortality for women aged 55 - 84 years increased from 2% in 1955 to 30% in 1995, whereas the standardized death rate for all other causes decreased.³⁵ Because of that, the total death rate increased after 1976 in Denmark. This development differed from that of the EU overall.³⁶ In 1996, the death rate of Danish women was 48% higher than the average in the EU.

The difference between Denmark and the other countries in the European Union reflects the Danish life style where many adults, especially women, smoke. Lung cancer and chronic obstructive pulmonary disease caused one quarter to a third of the extra deaths in Denmark in the age group 35 - 70 years compared with those of the other Scandinavian countries.³⁷

The development of the tobacco epidemic differs by gender. Men began to smoke cigarettes early in the 20th century, whereas smoking became a fashion in women in Western Europe later than in men. Similarly, the number of men who smoked started to decline around 1950, many years before the number of smoking women declined. Therefore, while the number of tobacco-caused deaths in men decreases in a few Western countries, women have ever more smoking-caused deaths. So the two genders differ in the impact from smoking on median life expectancy.

In the USA in recent decades, the death rate for lung cancer increased while the death rate for all other cancers decreased.³⁸ The overall death rate of cancer in the USA remained unchanged. Lung cancer is the leading cause of cancer death of men and women in the USA, and the leading cause of cancer death in men in most Western countries. In these countries the incidence of lung cancer in women increases and may surpass breast cancer as the most important cause of cancer deaths in women within the next twenty years. The lung cancer became a major cause of death in the twentieth century. Prior to that, lung cancer was a rare disease.

Half the active smokers die of smoke-caused diseases and a quarter die at 35 - 69 years of age. So tobacco-caused illness has a high impact on median life expectancy for men in Western countries and an increasing impact for women.

1.2.4 Gains from smoking cessation

Former smokers have a lower risk of illness than current smokers (Table 1).⁷ Quitting smoking reduces the health risks of smoking compared with that of continued smoking. From the first day, smoking cessation decreases the cardiovascular risk and it normalizes in two years. Smoking cessation also reduces the risk of lung cancer. Cohort studies showed that former smokers had a lower risk of lung cancer than active smokers. In the study of British male physicians, the former smokers lived longer than the current smokers and those who stopped smoking before they were 40 years of age gained most. Smokers who quit smoking normally have side effects, such as withdrawal symptoms and weight gain. Physical activity and a low fat diet may reduce the weight gain and add to the gains in health. The main gain from smoking cessation is the increase in median life expectancy. This gain is caused by the cessation itself and is not impaired by other changes in life style.

In Finland, the decrease in use of cigarettes was followed by a declining number of patients with lung cancer, cardiovascular disease, and cerebrovascular disease.³⁹

The Ministry of Health in Denmark estimated that a 50% decrease in smoking in Denmark would prolong the median life expectancy in the country by three years.¹² Other countries have made similar estimates.

1.2.5 Smoking in different countries

From the 1950s, as smokers in Western countries became increasingly aware of the health risks, fewer men have smoked, mainly because moderate smokers gave up smoking. In contrast, more male smokers began to smoke heavily (20 or more cigarettes a day), and more women began to smoke. Therefore, total cigarette consumption in a country may rise though fewer men are smoking.

In Denmark, the number of smokers among Danish men fell from 80% in 1950 to 35% in 1995 whereas the consumption of cigarettes rose from 2×10^9 cigarettes a year to 8×10^9 cigarettes a year.^{12,40}

Western European countries differ in trends in cigarette consumption in recent decades (see table 3).⁸ Consumption increased 50% in Portugal and Greece whereas it fell with one third in the UK and Finland.^{8,32,33}

In Catalonia in 1982, adult men smoked more than women: 58% versus 20%.⁴¹ The difference between gender in the youngest age group however, differed less: 59% of men smoked and 48% of women. In 1987, the proportion of smokers among young Spanish men and women was grossly similar, and young women smoked heavily more often than older women.

In Italy, smoking decreased in adult men from 72% to 46% in the period 1949 - 1983.⁴² Meanwhile, smoking rose from 9,7% to 17,7% in adult women. In this period, the number of heavy smokers and the total consumption of cigarettes rose and so did the number of lung cancers.⁴³

During the 1979 - 1991 period, more French women smoked.⁴⁴ The use of cigarettes and other tobacco products finally fell in France after the Evin tobacco law in 1991.⁴⁵

Laws limiting marketing and sponsoring of tobacco products, and restricting smoking in public places and workplaces may influence the consumption of cigarettes more than information to the public on the health hazards from smoking.

1.2.6 Social class and use of tobacco

Social classes differ in the use of tobacco. Smoking began as a fashion for upper-class men. Later, other groups in society took up smoking, while it became less trendy for upper-class men. This pattern is similar in most Western countries.

Initially in 1956, Norwegian men with high and medium income smoked more often than those with low-income. Women smoked less often and consumed less than men.⁷ 38% of high-income women, 30% of medium-income women and 15% of low-income women smoked. After 1975, the social pattern changed. Norwegian men with low-income smoked more often than other male social groups. In recent decades, Norwegian men with higher education smoked less often whereas more men with low educational attainments smoked. Overall, the number of smokers decreased in recent years.⁴⁷ Denmark had a similar development.^{48,49}

Since the 1970s, Great Britain also had these social changes. While tobacco use halved among high-income families, those with low-income smoke as often as they did thirty years ago. Today, unskilled workers smoke more than skilled workers and they are more likely to be heavy smokers than other social groups. They also differ in smoking habits in other ways. The low-income group uses hand-rolled cigarettes more than the high-income groups. Hand-rolled cigarettes increase the risk of health hazards from smoking. Unskilled workers also have a higher burden of other risk factors in life style: they are twice as likely to eat high-fat diets and have little physical activity in their leisure-time compared to skilled workers.

In the late part of the 20th century, Southern Europe had a pattern of smokers in social groups corresponding to that seen earlier in Northern Europe. In France, between 1979 – 1991 the number of smokers fell in most male socio-economic groups, whereas it rose in female workers.⁵⁰ In Spain in 1987, women with a university education and women with a high income smoked more often than other women. This is like the smoking pattern in Norway in 1956.

In most Southern European countries, women repeat the changes in smoking habits among women in Northern Europe: an increase in proportion of smokers to the level found in men, and an uptake of smoking by women with few educational qualifications and in lower social class.

1.2.7 Social gradient in tobacco-caused illness and death

The social groups that smoke most are the social groups with the highest numbers of tobacco-caused illness and death. In Finland, professions vary in the number of lung

cancers. Physicians had an incidence of lung cancer that is 0,2 times the national average whereas the number of lung cancers for employees in hotels and restaurants was twice the national average. In Sweden, workers had more illness than public employees (Table 6).

Table 6. Risk for workers in relation to that of public employees in Sweden.

	Relative risk	
	Male workers	Female workers
Lung cancer	1.66	1.59
Cardiovascular disease	1.56	1.81
Suicide	2.50	1.87
Total burden of diseases and accidents	1.45	1.34

Source: Public reviews of the Swedish state.¹³⁹

Male workers had a risk of lung cancer that was 66% higher than that of public employees. Tobacco-caused diseases such as lung cancer, cardiovascular disease and suicide caused most of the difference.

The median life expectancy of the social groups differed mainly due to the diversity in cardiovascular disease. Social class I is the highest social class and social class V the lowest. In Denmark, persons in social class I have the longest life and those in social class V the shortest.⁵¹ The population in the USA showed the same difference.^{52,53} For all social classes in Denmark, smokers had coronary heart disease more often than non-smokers (Table 7).⁵⁴ The extra number of patients with cardiovascular diseases from smoking rose from social class I to social class V. Many studies linked chronic obstructive pulmonary disease to low socio-economic status.

In recent years, the social gradient for tobacco-caused illness and death increased in Western countries.

Table 7. Total number of coronary heart disease in the Copenhagen male study

Social class	Total number of coronary heart diseases	
	Non-smokers	Smokers
I	3.8	7.3
II	3.4	12.8
III	4.9	14.7
IV	14.2	17.2
V	18.4	26.9
All	9.2	16.0

Source: Hein et al.⁵⁴ I = highest social class, V = lowest social class.

1.2.8 Tobacco-caused illness in workplaces

Some of the differences between social groups might be due to interactions of tobacco smoke with exposure to other compounds at the workplace. Exposure to asbestos increases the risk of lung cancer from smoking and smoking increases the risk from exposure to asbestos.⁵⁵ Asbestos workers have a five-fold increased risk of lung cancer compared with the non-exposed workers but asbestos workers who smoke have a fifty-fold increased risk. Thus smoking and exposure to asbestos act together in causing lung cancer by multiplying the risk of the two factors. Smoking also increases the health risks from exposure to other compounds, such as ethanol, silica and radiation.⁵⁶

Smoking in workplaces may cause absence due to sick leave, extra medical costs and insurance costs.⁵⁷ The working environment may accelerate the impact from smoking on health in many ways.

1.3 Nicotine addiction

Nicotine addiction is the greatest factor that contributes to the fact that a smoker continues to smoke. The definition of addiction has changed during the last part of the 20th century. Today addiction is defined as presence of three or more of the following findings:

- tolerance
- abstinence
- intake of the agent in larger amount or for a longer period than intended
- persisting ambition and not fulfilled attempts to reduce or control use of the agent
- large amount of time used for activities to obtain the agent
- important social, working time or spare time activities are reduced or prohibited due to the use of the agent
- use of the agent continues despite knowledge of a continuous or recurrent physical or psychological problem likely caused by or enhanced by the agent.

Smoking fulfils most criteria. Addiction to smoking is due to nicotine. Smokers who fail to quit smoking tend to blame themselves for lack of will power instead of blaming nicotine for their addiction. Those who are very addicted to nicotine are those who have the smallest chance of stopping. Nicotine addiction changes the smokers social behavior and attitude towards the health risks from smoking. Nicotine addiction is increasingly important for the social pattern of smoking and for the tobacco epidemic.

When smokers try to quit smoking, they get withdrawal symptoms. This also may occur when waking up in the morning. One of the first things many smokers do after waking up is smoking. Most smokers have tried to quit smoking in vain. Most current smokers who try to stop fail, and most former smokers tried to quit smoking several times before they succeed. Most current smokers know the health risks from smoking but are not able to quit smoking despite this knowledge. Some even continue to smoke after they have had lung cancer. Nicotine replacement therapy doubles the quit rate over that of smoking cessation without medical help. Bupropion also doubles the quit rate by acting on the centers in the brain for nicotine addiction. Recently, the tenth revision of the International Classification of Disease took up nicotine addiction as a diagnosis.

In a survey of the Norwegian workers, 68% of the smokers had previously tried to stop smoking. 3% planned to stop smoking within one week, 10% within one month, 21% within six months, and 16% later on, so overall 50% of the smokers intended to stop smoking. About 60% of the smokers considered it difficult to stop smoking. Of the smokers, 38% would take part in a smoking cessation course if offered by their employer. Some 47% thought that the labour union should help in securing smoke free workplaces. ⁵⁸

1.4 Exposure to environmental tobacco smoke

1.4.1 What is Environmental tobacco smoke?

Sharing a space with someone who is smoking exposes non-smokers to smoke. Ambient tobacco smoke is called environmental tobacco smoke (ETS), passive smoke or second-hand smoke. The act of breathing environmental tobacco smoke is called involuntary or passive smoking.

A burning cigarette produces two types of smoke: mainstream smoke - the smoke that smokers inhale and exhale from cigarettes - and sidestream smoke - the smoke that goes directly into the environment as tobacco burns. Environmental tobacco smoke consists of exhaled mainstream and sidestream smoke. Sidestream smoke comprises more than 75% of the overall smoke, and has higher levels of many noxious constituents than mainstream smoke (Table 8).

Environmental tobacco smoke contains many chemicals that irritate the eye and upper respiratory tract. The irritating constituents include particulates, acrolein, formaldehyde, ammonia, carbon monoxide, hydrogen cyanide, oxides of nitrogen and sulphur oxide. Environmental tobacco smoke also contains more than forty chemicals that cause cancer in animals and man. These compounds include:

- organic chemicals like aromatic polycyclic hydrocarbons, aromatic amines, nitrosamines, hydrazines, benzene and vinyl chloride
- inorganic compounds like arsenic, cadmium, chromium
- radio-nucleotides like polonium-210.

Finland, Germany and the US National Toxicology Program have listed environmental tobacco smoke as a workplace carcinogen.

Table 8. Constituents of fresh, undiluted mainstream smoke from nonfilter cigarettes and ratio for the constituents of diluted sidestream smoke.

	Mainstream smoke	Sidestream smoke/Mainstream smoke
<u>Gas phase</u>		
Carbon monoxide	10 - 23 mg	2.5 - 4.7
Carbon dioxide	20 - 40 mg	8 - 11
Formaldehyde	70 - 100 µg	5.6 - 8.3
Acetone	100 - 250 µg	2 - 5
Ammonia	50 - 130 µg	40 - 170
Hydrogen cyanide	400 - 500 µg	0.1 - 0.25
N-Nitrosodimethylamine	10 - 40 µg	20 - 100
N-Nitrosodiethylamine	ND - 25 ng	<40
Acrolein	60 - 100 µg	8 - 15
Hydrazine	32 ng	3
Benzene	12 - 48 µg	5 - 10
<u>Particulate phase</u>		
Particulate matter	10 - 40 mg	1.3 - 1.9
Nicotine	1 - 2.5 mg	2.6 - 3.3
2-Toluidine	160 ng	19
Phenol	60 - 140 µg	1.6 - 3.0
Aniline	360 ng	30
Benzo[a]pyrene	20 - 40 ng	2.5 - 3.5
4-Aminobiphenyl	4.6 ng	31
N-Nitrosodiethanolamine	20 - 70 ng	1.2
Cadmium	100 ng	7.2
Nickel	20 - 80 ng	13 - 30
Polonium-210	0.04 - 0.1 pCi	1.0 - 4.0

Source: Committee on Passive Smoking of the National Research Council, USA, 1986.¹

1.4.2 Procedures

Many procedures are used in measuring environmental tobacco smoke and its impact on exposed non-smokers. The procedures measure the exposure in the environment or the compounds absorbed by non-smokers.

Indirect procedures measure the exposure in the environment by the concentration of one or more of the compounds of ETS. They may measure the concentrations in specific rooms by sampling air over hours and days. This gives a time-weighted average level, f.e. in enclosed workplaces. Most reports deal with nicotine levels and the concentration of respirable suspended particulate matter. Exposure may also be calculated from the number of smoked cigarettes, the size of the room and the air renewal in the room.⁵⁹

Direct procedures measure the compounds absorbed in the exposed persons. The compounds may be measured as the part of the compounds that the exposed person absorbs. Most studies measured the short-term exposure. Another direct method is to measure biomarkers of tobacco smoke, either compounds of the smoke or metabolites of the compounds, in exposed persons.⁶⁰ Cotinine is a main metabolite of nicotine and widely used as a biomarker. Cotinine is metabolized in the body of the exposed person with a half-life-time of 17 hours. So the cotinine levels show how much non-smokers were exposed to nicotine for two days before the sampling.

An unexposed non-smoker has a low cotinine level. Exposure for 8-hours to a nicotine concentration in the air of $20 \mu\text{g}/\text{m}^3$ nicotine leads to absorption of $112 \mu\text{g}$ nicotine through the lungs. This corresponds to the nicotine content of a tenth of a cigarette. This dose of nicotine raises the blood cotinine level $1 \mu\text{g}/\text{L}$.

When non-smokers are heavily exposed to ETS, they have high levels of a lung cancer carcinogen found only in tobacco smoke (4-(methylnitroso-amino)-1-(3-pyridyl)-1-butanone) and other carcinogens.^{62,63}

Comparing the measurements in current smokers and non-smokers exposed to ETS, measurements of nicotine and cotinine in non-smokers underestimate the risk of illness compared with the information regarding the risk of epidemiologic studies.

Passive smoking gives higher levels of toxic compounds in relation to the level of nicotine than mainstream smoke (Table 8). Therefore, measurements of nicotine in the air or cotinine in the exposed underestimate the risk from passive smoking.

1.4.3 Exposure

From workplaces, bars and restaurants have the highest nicotine levels. Nicotine levels are lowest in offices (Table 9).

Table 9. Measurements of environmental tobacco smoke

Area	Nicotine in the air ($\mu\text{g}/\text{m}^3$)	Reference	Cotinine in the exposed ($\mu\text{g}/\text{L}$)	Reference
Nightclubs	37.1 (28 - 50)	⁶⁴	3.4 (1.7 - 5.0)	⁷
Services	3.0	⁶⁸		
Industry workplaces	2.7	⁶⁸		
Offices	0.6	⁶⁸		
Workplaces with a ban on smoking	0 - 0.39	⁷³		
Workplaces with restrictions on smoking	1.3 - 5.9	⁷³		
Workplaces with smoking allowed	8.6 - 10	⁷³		
Homes with two non- smoking parents	0.15	⁷	0.29	⁶¹
Homes with a smoking father	2.5		1.17	
Homes with a smoking mother	5.5		2.20	
Homes with two smoking parents	12.1		4.1	

Nightclubs had an average nicotine level of $37 \mu\text{g}/\text{m}^3$.⁶⁴ Non-smoking musicians who worked in nightclubs had a mean cotinine concentration of $3.4 \mu\text{g}/\text{L}$ (range 1.7 - 5.0) and non-smokers working in bars had a mean level of $7.9 \mu\text{g}/\text{L}$.⁶⁵

The nicotine levels in a room reflect the number of cigarettes smoked in the room. Rooms designated for smokers had nicotine levels as high as 77 $\mu\text{g}/\text{m}^3$, even if the room had good ventilation.

A large series of US offices had a mean of 4.3 $\mu\text{g}/\text{m}^3$, and 4.3 $\mu\text{g}/\text{m}^3$ in homes.^{66,67} Recently in Finland, mean nicotine concentration was 2.7 $\mu\text{g}/\text{m}^3$ in industry workplaces, 3.0 $\mu\text{g}/\text{m}^3$ in services, and 0.6 $\mu\text{g}/\text{m}^3$ in offices.⁶⁸

Non-smokers who were exposed to smoke both at home and at work had a mean cotinine level of 0.926 $\mu\text{g}/\text{L}$, and those who were exposed to smoke at the work but not at home had a cotinine level of 0.318 $\mu\text{g}/\text{L}$.⁶⁹ The mean cotinine level was 0.651 $\mu\text{g}/\text{L}$ in those exposed to smoke at home but not at work, and 0.132 $\mu\text{g}/\text{L}$ for those not exposed to smoke at home or at work. Non-smokers who lived with a smoker and those who worked with smokers have similar median levels of cotinine in saliva, 1 $\mu\text{g}/\text{L}$ versus 0.8 $\mu\text{g}/\text{L}$.⁷⁰

In a recent study, 88% of all American adult non-smokers had measurable serum cotinine levels.⁶⁹ In the EU, 79% of Europeans over 15 years of age are exposed to ETS.^{71,72} The levels of nicotine in the workplaces corresponded with the cotinine levels in the exposed.

1.4.4 Restrictions to environmental tobacco smoke

Restrictions of smoking at workplaces lowers the exposure to ETS. The levels were median 0.10 - 10.0 $\mu\text{g}/\text{m}^3$ in offices where they allowed smoking, <0.05 to 5.85 $\mu\text{g}/\text{m}^3$ in offices where they restricted smoking, and <0.05 to 0.39 $\mu\text{g}/\text{m}^3$ in offices where they banned smoking (see table 8).^{73,74}

The smoking section of a USA cafeteria had a four-day mean nicotine level of 47.9 $\mu\text{g}/\text{m}^3$; it was 3.4 $\mu\text{g}/\text{m}^3$ in the non-smoking section of the cafeteria, 2 to 8 m away from the smoking section and 0.5 $\mu\text{g}/\text{m}^3$ more than 9 m away from the smoking section.⁷⁵ Restrictions on smoking in workplaces in Finland by law in 1995 lead to less exposure to ETS for the smokers and the non-smokers.

German surveys showed that smoking was a cause of conflict between smokers and non-smokers in a third of the workplaces.⁷⁶ This is because 80% of the non-smokers

preferred a ban of smoking in workplaces whereas only 35% of the smokers had the same opinion.

The Norwegian Council on Smoking and Health made a survey of Norwegian working persons.⁵⁸ Norway banned smoking in workplaces by law in 1988. After the ban, the Norwegians found that smoking in the workplace was of little nuisance. One sixth of non-smokers avoids smoky rooms. Even many smokers prefer smoke free rooms when they are not smoking. In the Netherlands, 35% of non-smoking employees felt annoyed or very annoyed to inhale ETS.⁷⁷ Some 78% thought that a smoky workplace was harmful to their health. The US Occupational Safety and Health Administration proposed as a norm for clean indoor air that it should be a nuisance for less than 20% of the exposed.

1.5 Immediate effects of environmental tobacco smoke

Most non-smokers feel uncomfortable when they are exposed to smoke. They may have irritation of eyes, headache, dizziness, tiredness, sore throat, nausea, coughing, or shortness of breath. Whereas smokers get used to the smoke, non-smokers remain uncomfortable being exposed to smoke. Even short-term exposure to ETS reduces the function of the body of the exposed. Passive smoking may impair lung function of non-smokers with 8%, even at low levels of exposure.⁷⁸ In a Swiss study, exposure to smoke gave non-smokers acute symptoms of the lungs.⁷⁹ Control of bronchial asthma is more complex, and the illness is worse, in adult patients who are exposed to ETS at home or at work.⁸⁰

Exposure to smoke reduces the oxygen uptake and exercise capacity.⁸¹ Patients with coronary heart disease may exercise 8% shorter and at a 10% lower intensity when they breathe ETS than when they breathe clean air.⁸²⁻⁸⁴ Passive smoking may give rise to cardiac arrhythmias in patients with heart disease. In the vulnerable, the smoke may provoke acute myocardial infarction.

ETS also causes adverse responses in the neuroendocrine and immune systems and a series of biochemical abnormalities. Passive smoking raises the blood levels of carboxyhemoglobin and carbon monoxide, changes thought to contribute to the

development of coronary heart disease.⁸⁴ Short-term exposure makes the platelets 80% more sensitive to prostacyclin and increases the clotting of the platelets.⁸⁵ ETS also raises the level of fibrinogen in the blood.⁸⁶ These changes increase the risk of a blood clot in the arteries of the heart and the brain and by that the risk for acute myocardial infarction and stroke.

1.6 Long-term effects of environmental tobacco smoke

1.6.1 Long-term effects in children

For a pregnant woman, exposure to ETS retards the growth of the foetus and increases the risk of birth complications.⁸⁷⁻⁹⁰ For children, ETS increases the risk of sudden infant death syndrome, bronchitis and pneumonia, bronchial asthma and exacerbations of the asthma, middle ear infections and glue ear, the most common cause of deafness in children.^{91,92} The risk for the child is higher when the mother smokes than when the father smokes.⁹³ The difference corresponds with the nicotine and cotinine findings. Children with bronchial asthma have a reduced severity of the illness as the parents reduce their smoking.⁹⁴ Exposure to passive smoking impairs the development of the child.

1.6.2 Long-term effects in adults

For adults, ETS causes chronic illness and death in the pattern well known from active smoking. Passive smoking in the workplace increased the risk of lung cancer by 39%.⁹⁵ Wells only reviewed studies that reported the risk of passive smoking in never-smokers, excluded former smokers, and followed the reported groups for at least ten years.⁹⁶⁻¹⁰⁰ Another recent review included a Swedish, a German and a multicenter European study.¹⁰¹⁻¹⁰³ Passive smoking raises the risk of lung cancer with the extent of the exposure.¹⁰⁴ The risk of lung cancer from passive smoking at the workplace rises with the number of years of exposure and with the strength of the exposure.¹⁰⁵ The risk decreased as the exposure was stopped and the risk lowered with increasing time since last exposure. The findings correspond with the risk of lung cancer from ETS at home.¹⁰⁵⁻¹⁰⁸

Long-term exposure to passive smoking reduced lung function, increasingly with the extent of the exposure.^{79,109}

The risk of coronary heart disease from passive smoking related both to exposure at home and at the workplace.¹¹⁰ Both exposures at home and at workplaces increased the risk. Passive smoking increased the risk by 25% for a non-smoker compared with that of the unexposed.¹¹¹⁻¹¹⁹ Steenland analysed eight studies and reviewed the risk of heart disease from passive smoking at the workplace.¹²⁰ Passive smoking in workplaces increased the risk with 21%. This implies a rate of death of coronary heart disease at 65 years of age of 0.004, and a death rate at 70 years of age of 0.007. Another review of the link between ETS and heart attacks evaluated 19 studies and 6,600 cases.¹²¹ Passive smoking raised the risk of heart attack by 32% and the risk of death of heart attack by 14%.¹¹¹ A study of a large number of nurses showed that occasional exposure to ETS at home and at work increased the risk by 58% and regular exposure raised the risk by 91%.¹¹³ So the impact from passive smoking may be higher for women than for men. Non-smokers with increasing serum cotinine concentrations had a higher risk of coronary heart disease.¹²²

Passive smoking causes cerebrovascular disease (stroke).¹²³ It causes atherosclerosis in carotid arteries, the large blood vessels to the brain, and infarctions in the brain not causing symptoms.

Passive smoking also produces more arteriosclerosis in the arteries.¹²⁴

Exposure to smoke lowers the level of high-density lipoprotein cholesterol - the good cholesterol - in non-smokers.¹²⁵ Smoke also increases the ratio of serum cholesterol to high-density lipoprotein cholesterol, aggregation of platelets, and damage of endothelial cells in the arteries.¹²⁶ Even young persons have these changes in lipid profile.^{127,128} These changes raise the risk of cardiovascular and cerebrovascular disease and peripheral-arteriosclerosis.

Exposed to environmental tobacco smoke at home or at work, adult non-smokers have a 40% - 60% higher risk of developing bronchial asthma¹²⁹ and a reduced lung function.^{109,130-132} They also have a higher risk of pneumonia.¹³³

1.6.3 Risk assessment of long-term effects

The risk of illness from passive smoking on health is 1/50 to 1/10 that of the risk from active smoking. Active smoking causes one third of the deaths in men aged 35 - 69

years so the number of premature deaths from passive smoking is large. Environmental tobacco smoke, whether at home or at the workplace, is the third leading cause of poor health and premature death that can be prevented, next to that of active smoking and abuse of alcohol.¹³⁴

Most exposed non-smokers have an increase of saliva cotinine level to 0.4 µg/L. This level implies a lifetime risk of 1/1000 for death of lung cancer and a risk of 1/100 for death of heart disease.¹³⁵ A lifetime risk of 1/1000 at the workplace is a norm for society to take action to reduce the risk. Persons get a smaller risk of illness when they are less exposed to nicotine and have lower cotinine levels.

Passive smoking at workplaces causes 30.000 – 60.000 deaths per year in the USA and 90.000 – 180.000 cases of cardiovascular events that did not end the life of the patients.^{134,136} Likewise, passive smoking causes 500 deaths per year in Norway.^{137,138} Based on these estimates and as the EU is larger than the USA, the exposure to ETS may be estimated to cause 50.000 – 100.000 deaths a year in the EU and 200.000 – 400.000 cases of heart attacks that does not cause death.

1.7 Conclusions

Western European countries differ in the prevalence of smoking, in the consumption of cigarettes, in the change in use of tobacco and in the pattern of tobacco-caused disease. All countries may learn from the experience of smoking in other countries.

Finland and the UK had a fall in consumption of cigarettes and a fall in incidence of lung cancer and cardiovascular disease. In contrast, Denmark had the highest number of female smokers of the Western countries and the highest number of smoking-caused diseases and deaths in women. Today, smoking contributes to the social inequality in health. Unfortunately, other Western countries are beginning to see the disease pattern for chronic obstructive pulmonary disease that Denmark had in the seventies and eighties.

Denmark has been called the smokers paradise because Denmark has fewer restrictions on smoking than other Nordic countries. Denmark allows smoking in most public places. The smokers paradise may be the non-smokers hell.

Continuing smoking of adult smokers is not solely the free choice of a mature individual knowing the health risks of smoking. Most smokers continue to smoke because they

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Continuing smoking of adult smokers is not solely the free choice of a mature individual knowing the health risks of smoking. Most smokers continue to smoke because they

are addicted to nicotine. More than half the smokers have tried to stop smoking. This large group may gain from more professional support for smoking cessation. Half of the smokers die of tobacco-caused diseases. Due to the low cure rate for the most common tobacco-caused diseases, the best option is to prevent them from occurring. *Quitting smoking reduces the health risk from smoking.*

ETS is one of the most widespread and harmful indoor air pollutants. Passive smoking causes the death of thousands of Europeans each year. Avoidance of passive smoking reduces the risk. Most non-smokers wish not to be exposed to tobacco smoke against their will. So a comfortable, cooperative and productive work environment respects the right for self-determination of the non-smokers with regard to involuntary exposure to ETS. *A ban on smoking in workplaces is an effective means to reduce exposure to ETS in workplaces. Most non-smoking employees and many smokers favour a ban.*

References

Bibliography Chapter 1

1. National Research Council. Environmental Tobacco Smoke. Measuring Exposures and Assessing Health Effects. Washington, District of Columbia, USA: National Academy Press, 1986; 1-337.
2. US Department of Health and Human Services. The health consequences of involuntary smoking: A report from the Surgeon General. Rockville, Maryland, USA, Office on Smoking and Health. Centers of Disease Control, Public Health Service, 1986.
3. National Institute for Occupational Safety and Health. Environmental tobacco smoke in the workplace: Lung cancer and other health effects. Washington, District of Columbia, USA: Department of Health and Human Services. National Institute of Occupational Safety and Health, 1991.
4. California Environmental Protection Agency. Health effects of exposure to Environmental Tobacco Smoke. Los Angeles, California: California Environmental Protection Agency, 1997.
5. Ducatman, Alan M. and McLellan, Robert K. Epidemiological basis for an occupational and environmental policy on environmental tobacco smoke. *The American College of Occupational and Environmental Medicine*, 2000; 1-11.
6. Sasco, AJ., Dubois, G, Ah-Song, R, Whidden, M. Passive smoking. The health impact. Sasco, AJ., Dubois, G, Ah-Song, R, Whidden, M. Paris, France, Comité National Contre le Tabagisme, 1999; 3-63.
7. Kjønstad A, Birkeland V, Dybing E, Lund KE, Sanner T, Skjerdal NV. Product liability of the tobacco industry. Review by a group of experts with mandate from the Ministry of Health and Social Affairs January 23, 1998 (Tobakksindustriens erstatningsansvar. Utredning fra en faggruppe med mandat fra Social- og helsedepartementet 23. januar 1998. Avgitt 28. juni 2000.). Oslo, Norway: 2000; 17-661.
8. Corrao MA, Guindon GE, Sharma N, Shokoohi DF. Tobacco Control Country Profiles. Atlanta, Georgia, USA: The American Cancer Society, 2000; 1-512.

9. Müller. Krebsforschung 1940; 49: 57
10. Shairer DE, Schoningen E. Zeitschrift für Krebsforschung 1941; 34: 261-269.
11. Hill A, Doll R. BMJ 1950;
12. Commission of median life expectancy - Ministry of Health, Danish Institute of Clinical Epidemiology. Mortality of the Danes in the 1990s. 1. report of the Commission of median life expectancy (Danskernes dødelighed i 1990erne. 1. delrapport fra Middellevetidsudvalget). Copenhagen: Ministry of Health, 1998; 1-96.
13. Devesa SS, Grauman DJ, Blot WJ, Fraumeni JF, Jr. Cancer surveillance series: Changing geographic patterns of lung cancer mortality in the United States, 1950 through 1994. J Natl Can Inst 1999; 91: 1040-9.
14. Joossens, L Sasco, A. *Some like it light. Women and smoking in the European Union.* Brussels, Belgium, European Network on Smoking Prevention, 1999: 5-65..
15. Council on Tobacco or Health. Lung cancer mortality in women in European countries. 1999. Copenhagen, Council on Smoking or Health.
16. Vestbo J, Juel K, Døssing M, Prescott EIB. The epidemy noone will acknowledge (Epidemien ingen tør se i øjnene). Ugeskr Laeg 2001; 163: 1870-2.
17. Kroumhout D, Menotti A, Blackburn H. The Seven Countries Study. Bilthoven, The Netherlands: RIVM, 1993; 7-219.
18. Jensen G, Nybroe J, Appleyard M, Schnohr P. Risk factors for acute myocardial infarction in Copenhagen II: smoking, alcohol intake, physical activity, obesity, oral contraception, diabetes, lipids, and blood pressure. Eur Heart J 1991; 12: 298-308.
19. Nyboe J, Jensen G, Appleyard M, Schnohr P. Smoking and the risk of first acute myocardial infarction. Am Heart J 1991; 112: 438-47.
20. Hansen EF, Andersen LT, von Eyben FE. Cigarette smoking and age at first acute myocardial infarction, and influence of gender and extent of smoking. Am J Cardiol 1993; 71: 1439-42.
21. von Eyben FE, Bech J, Madsen JK. High prevalence of smoking in young patients with acute myocardial infarction. J Roy Soc Health 1996; 116: 153-6.

22. von Eyben FE, von Eyben R. Smoking and other major coronary risk factors and acute myocardial infarction before 41 years of age: Two Danish case-control studies. *Scand Cardiovascular J* 2000; 35: 25-9.
23. Smith GD, Phillips AN, Neaton JD. Smoking as "independent" risk factor for suicide: illustration of an artifact from observational epidemiology? *Lancet* 1992; 340: 709-12.
24. Tverdal A, Thelle D, Stensvold I, Leren P, Bartveit K. Mortality in relation to smoking history: 13 years' follow-up of 68,000 Norwegian men and women. *J Clin Epidemiol* 1993; 46: 475
25. Knudsen NJ. Regional variations in goitre prevalence in Denmark - and the impact of tobacco smoking. *Ugeskr Laeg* 2001; 163: 1577-8.
26. Winsa B, Karlsson A. Rökning och giftstruma. *Läkartidningen* 1993; 90: 3051-2.
27. Hopper JL, Seeman E. The bone density of female twins discordant for tobacco use. *N Eng J Med* 1994; 330: 387-92.
28. Kato I, Nomura A, Stemmermann GN, Chyou P-H. A prospective study of gastric and duodenal ulcer and its relation to smoking, alcohol, and diet. *Am J Epidemiol* 1992; 135: 521-30.
29. Prescott, E. Tobacco-related diseases: the role of gender. An epidemiologic study based on data from the Copenhagen Center for Prospective Population Studies. Copenhagen, Lægeforeningens Forlag 2000; 1-63..
30. Doll R, Peto R, Wheatley KGR, Sutherland I. Mortality in relation to smoking: 40 years' observations on male British doctors. *BMJ* 1994; 309: 901-11.
31. Prescott E, Olsner M, Hein HO, Borch-johnsen K, Schnohr P, Vestbo J. Life expectancy in Danish women and men related to smoking habits: smoking may affect women more. *J Epidemiol Commun Health* 1998; 52: 131-2.
32. Peto R, Lopez AD, Borelam J, Thun M, Heath C, Jr. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet* 1992; 339: 1268-78.
33. Peto R, Lopez AD, Borelam J, Thun M, Heath C, Jr. Mortality from smoking in developed countries 1950-2000. Indirect estimates from National Vital statistics. Oxford: Oxford University Press, 1994.

34. von Eyben FE. Redefining goals for tobacco control. *Lancet* 2001;357:889.
35. Juel K. Increased mortality among Danish women: population based registry study. *BMJ* 2000; 321: 349-50.
36. Kesteloot H. Queen Margrethe II and mortality in Danish women. *Lancet* 2001; 357: 871-2.
37. Commission of median life expectancy - Ministry of Health. Life expectancy in Denmark. 2. report (Middellevetidsudvalget: Levetid i Danmark. 2 delrapport). Copenhagen: Ministry of Health, 1994; 1-115.
38. SEER. SEER Cancer Statistics Review, 1973-1996 (NCI 1999). <http://seer.ims.nci.nih.gov>. 1999.
39. Puska P, Tuomilehto J, Nissinen A, Vartainen E. The North Karelia project. 20 year results and experiences. Helsinki, Finland, The National Public Health Institute (KTL) 1995; 5-363.
40. Osler M. Smoking habits in Denmark (Danskernes rygevaner). Copenhagen: Danish Council on Smoking and Health, 1992; 1-55.
41. Onis Md, Villar J. La consommation de tabac chez la femme espagnole. *Wld Hlth Statist.Quart.* 1991; 44: 80-8.
42. La Vecchia C. Smoking in Italy, 1949-1983. *Prev Med* 1986; 15: 274-81.
43. La Vecchia C. Patterns of cigarette smoking and trends in lung cancer mortality in Italy. *J Epidemiol Commun Health* 1985; 39: 157-64.
44. Sasco AJ, Grizeau D, Danzon M. Is tobacco use finally decreasing in France? *Cancer Detect Prev* 1995; 19: 210-8.
45. Sasco AJ, Mélihan-Cheinin P, Ah-Song R, Hirsch A. Evaluation et comparaison de la législation de contrôle du tabagisme dans les Etats membres de l'Union Européenne. *Rev Epidém et Santé Publ* 1998; 46: 523-41.
46. Gong YL, Koplan JP, Feng W, Chen CC, Zheng P, Harris JR. Cigarette smoking in China. *JAMA* 1995; 274: 1232-4.
47. Rønneberg A, Lund KE, Hafstad A. Lifetime smoking habits among Norwegian men and women born between 1890 and 1974. *Int J Epidemiol* 1994; 23: 267-76.

48. Kjølner M, Rasmussen NK, Keiding L, Petersen HC, Nielsen GA. Health and morbidity in Denmark 1994 and development since 1987 (Sundhed og sygelighed i Danmark 1994 og udviklingen siden 1987). Copenhagen: Danish Institute for Clinical Epidemiology, 1995; 9-395.
49. Hansen NS. Sociology of smoking (Rygningens sociologi). Copenhagen: Danish Council of Smoking or Health, 1995; 4-71.
50. Sasco AJ, Grizeau D, Pobel D, Chatard O, Danzon M. Tabagisme et classe sociale en France de 1974 à 1991. Bull Cancer 1994; 81: 355-9.
51. Commission of median life expectancy - Ministry of Health. Social inequality in health. Differences in health, lifestyle, and use of health service. 2nd report from the Commission of median life expectancy (Social ulighed i sundhed. Forskelle i helbred, livstil og brug af sundhedsvæsenet. 2. delrapport fra Middellevetidsudvalget). Copenhagen: Ministry of health, 2000; 1-88.
52. Schenberg J, Sternhagen A, Mason T, et al. Occupation and lung cancer risk among New Jersey white males. J Natl Cancer Inst 1987; 79: 13-21.
53. Singleton J, Beaumont J. COMS II. California Occupational Mortality 1979-1981. Adjusted for smoking, alcohol, and socio-economic status. Sacramento: California Department of Health Services, 1989.
54. Hein HO, Suadicani P, Gyntelberg F. Ischemic heart disease incidence by social class and form of smoking: the Copenhagen Male Study -17 years' follow-up. J Intern Med 1992; 231: 477-83.
55. US Department of Health and Human Services. Cancer and chronic lung disease in the workplace. A report of the Surgeon General. Rockville, Maryland, USA: Office on Smoking and Health. Public Health Service. U.S. Department of Health and Human Services, 1985; 1-542.
56. World Health Organization. Health effects of interactions between tobacco use and exposure to other agents. Geneva: 1999; 1-149.
57. Parrot S, Godfrey C, Raw M. Costs of employee smoking in the workplace in Scotland. Tob Control 2000; 9: 187-92.

58. Markeds- og Medieinstituttet as. Report on smoking in working individuals 15 years of age or more (Rapport om røyking blant yrkesaktive personer 15 år+). 4889. Oslo, Norway, Norwegian Council on Smoking and Health (Statens Tobakksskaderåd) 1999: 1-29.
59. Ott WR. Mathematical models for predicting indoor air quality from smoking activity. *Environ Health Perspect* 1999; 107, Suppl 2: 375-81.
60. Benowitz NK. Biomarkers of environmental tobacco smoke exposure. *Environ Health Perspect* 1999; 107, Suppl 2: 349-55.
61. Cook DG, Whincup PH, Jarvis MJ, Strachan D, Papacosta O, Bryant A. Passive exposure to tobacco smoke in children aged 5-7 years: individual, family, and community factors. *BMJ* 1994; 308: 384-9.
62. Hecht SS, Carmella SG, Murphy SE, Akerkar S, Brunneman KD, Hoffmann D. A tobacco-specific lung carcinogen in the urine of men exposed to cigarette smoke. *N Eng J Med* 1993; 329: 1543-6.
63. Maclure M, Katz RB, Bryant MS, Skipper PL, Tannenbaum SR. Elevated blood levels of carcinogens in passive smokers. *Am J Pub Health* 1989; 79: 1381-4.
64. Bergman TA, Johnson DL, Boatright DT, Smallwood KG, Rando RJ. Occupational exposure of non-smoking nightclub musicians to environmental tobacco smoke. *Am Ind Hyg Assos* 1996; 57: 746-52.
65. Jarvis M, Foulds J, Feyerabend C. Exposure to passive smoking among bar staff. *Brit J Addict* 1992; 87: 111-3.
66. U.S. Environmental Protection Agency. Office on Research and Development. Respiratory effects of passive smoking: lung cancer and other diseases. Washington, District of Columbia, USA: U.S. Environmental Protection Agency, 1992; 1-200.
67. Siegel M. Involuntary tobacco smoking in the restaurant workplace. A review of employee exposure and health effects. *JAMA* 1995; 270: 3490-3.
68. Heloma A, Kähkönen E, Kaleva S, Reijula K. Smoking and exposure to tobacco smoke at medium-sized and large-scale workplaces. *Am J Ind Med* 2000; 37: 214-20.

69. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke. The third National Health and Nutrition Examination Survey, 1988 to 1991. *JAMA* 1996; 275: 1233-40.
70. Emmons KM, Abrams DB, Marshall R, et al. An evaluation of the relationship between self-report and biochemical measures of environmental tobacco smoke exposure. *Prev Med* 1994; 23: 35-9.
71. Becher H, Zatonski W, Joeckel KH. Passive smoking in Germany and Poland: Comparison of exposure levels, sources of exposure, validity and perception. *Epidemiology* 1992; 3: 509-14.
72. INRA. Les Européens et la santé publique. Eurobaromètre 43.0. DG V/F/2. 1995. La Commission Européenne.
73. Hammond SK, Sorensen G, Youngstrom R, Ockene JK. Occupational exposure to environmental tobacco smoke. *JAMA* 1995; 274: 956-60.
74. Hammond S, Katharine A. Exposure of U.S. workers to environmental tobacco smoke. *Environ Health Perspect* 1999; 107, Suppl 2: 329-40.
75. Hepworth S. Senior Honors thesis. Worcester, MA: Worcester Polytechnical Institute, 1987.
76. Wiebel, Friedrich J. Nichtraucherchutz am Arbeitsplatz. Bonn, Germany, Koalition gegen das Rauchen, 2001.
77. Willemsen MC, de Vries H, Genders R. Annoyance from environmental tobacco smoke and support for no-smoking policies at eight large Dutch workplaces. *Tob Control* 1996; 5: 132-8.
78. Danuser M, Weber A, Hartman AL, Krueger H. Effects of a bronchoprovocation challenge test with cigarette sidestream smoke on sensitive and healthy adults. *Chest* 1993; 103: 353-8.
79. Leuenberger P, Ackermann-Liebrich, Künzli N, Schindler C, Perruchoud AP, and SAPALDIA. SAPALDIA, past, present, and future (SAPALDIA: passé, present, et avenir). *Schweiz Med Wochenschr* 2000; 130: 2921-7.

80. Jindal SK, Gupta D, Singh A. Indices of morbidity in adult patients exposed to environmental tobacco smoke. *Chest* 1994; 106: 746-9.
81. McMurray RG, Hicks LL, Thompson DL. The effects of passive inhalation of cigarette smoke on exercise performance. *Eur J Appl Physiol* 1985; 54: 196-200.
82. Aranow W. Effect of passive smoking on angina pectoris. *N Eng J Med* 1978; 59: 21-4.
83. Leone A, Mori L, Bertanelli F, Fabiano P, Filippelli M. Indoor passive smoking: its effects on cardiac performance. *Int J Cardiol* 1991; 33: 247-52.
84. Howard G, Thun MJ. Why is environmental tobacco smoke more strongly associated with coronary heart disease than expected? A review of potential biases and experimental data. *Environ Health Perspect* 1999; 107, Suppl. 6: 853-8.
85. Burghuber OC, Punzengruber C, Sinzinger H, Haber P, Silberbauer K. Platelet sensitivity to prostacyclin in smokers and non-smokers. *Chest* 1986; 90: 34-8.
86. Iso H, Shimamoto T, Sato S, Koike K, Lida M, Komachi Y. Passive smoking and plasma fibrinogen concentrations. *Am J Epidemiol* 1996; 144: 1151-4.
87. Misra DP, Nguyen RHN. Environmental tobacco smoke and low birth weight: A hazard in the workplace? *Environ Health Perspect* 1999; 107, Suppl 6: 897-904.
88. Council on Tobacco and health. Women and smoking (Kvinder og rygning). Copenhagen: Council on Smoking or Health, 1999; 7-128.
89. Martinez FD, Wright AL, Taussig LM, and the Group Health Associates. The effect of paternal smoking on the birthweight of newborns whose mothers did not smoke. *Am J Pub Health* 1994; 84: 1489-91.
90. Dejin-Karlsson E, Hanson BS, Ostergren PE, Sjoberg NO, Marsal K. Does passive smoking in early pregnancy increase the risk of small-for-gestational-age infants? *Am J Pub Health* 1998; 88: 1523-7.
91. Chilmonczyk BA, Salmun LM, Magathlin KN, Neveux LM. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Eng J Med* 1993; 328: 1665-9.

92. Bakoula CG, Kafritsa YJ, Kavadias GD, Lazapoulou DD, et al. Objective passive smoking indicators and respiratory morbidity in young children. *Lancet* 1995; 346: 280-1.
93. Tobacco Free Initiative. International consultation on environmental tobacco smoke (ETS) and child health 11 - 14 January 1999, Geneva, Switzerland. WHO/NCD/TFI/99.10, 4-28. 1999. Geneva, Switzerland, World Health Organization, Division of noncommunicable diseases, Tobacco Free Initiative.
94. Murray AB, Morrison BJ. The decrease in severity in children of parents who smoke since parents have been exposing them to less smoke. *J Allergy Clin Immunol* 1993; 91: 102-10.
95. Wells AJ. Lung cancer from passive smoking at work. *Am J Pub Health* 1998; 88: 1025-9.
96. Wu AH, Henderson BE, Pike MC, Yu MSC. Smoking and other risk factors for lung cancer in women. *J Natl Cancer Inst* 1985; 74: 747-51.
97. Shimizu H, Morishita M, Mizuno K, et al. A case-control study of lung cancer in non-smoking women. *Tohoku J Exp Med* 1988; 154: 389-97.
98. Kalandidi A, Katsouyanni K, Voropoulou N, Basta G, Saracci R. Passive smoking and diet in the etiology of lung cancer. *Cancer Causes Control* 1990; 1: 15-21.
99. Reynolds P, von Behren J, Fontham ETH, et al. Occupational exposure to environmental tobacco smoke. *JAMA* 1996; 275: 441-2.
100. Kabat GC, Stellman SD, Wynder EL. Relations between exposure to environmental tobacco smoke and lung cancer in lifetime non-smokers. *Am J Epidemiol* 1995; 142: 141-8.
101. Nyberg F, Agrenius V, Svartengren K, Svensson C, Pershagen G. Environmental tobacco smoke and lung cancer in non-smokers: does time since exposure play a role? *Epidemiology* 1998; 9: 300-8.
102. Bofetta P, Agudo A, Ahrens A, et al. Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. *J Natl Cancer Inst* 1998; 90: 1440-50.
103. Jöckel K-H, Pohlmann H, Ahrens W, Krauss M. Environmental tobacco smoke and lung cancer. *Epidemiology* 1998; 9: 672-5.

104. Reynolds P. Epidemiologic evidence for workplace ETS as a risk factor for lung cancer among non-smokers: specific risk estimates. *Environ Health Perspect* 1999; 107, Suppl 6: 865-72.
105. Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ* 1997; 315: 980-5.
106. Cardenas VM, Thun MJ, Austin H, et al. Environmental tobacco smoke and lung cancer mortality in the American Cancer society's cancer prevention study II. *Cancer Causes Control* 1997; 8: 57-64.
107. Wichmann, H. E., Brüske-Hohfeld, I., Möhner, M., Kreienbrock, L., Kreuzer, M., Jöckel, K. H., Ahrens, W., and Pohlabein, H. Abschlussericht. Zum Forschungsvorhaben: Lungenkrebsrisiko durch berufliche Exposition. GSF Forschungszentrum für Umwelt und Gesundheit. 1997.
108. Kreuzer M, Krauss M, Kreienbrock L, Jockel KH, Wichmann HE. Environmental tobacco smoke and lung cancer: a case-control study in Germany. *Am J Epidemiol.* 2000; 151: 241-50.
109. White JR, Froeb HF. Small-airways dysfunction in non-smokers chronically exposed to tobacco smoke. *N Eng J Med.* 1980; 302: 720-3.
110. He VS, Allen K, Prerost M, Hughes J, Whelton PK. Passive smoking and the risk of coronary heart disease - a metaanalysis of epidemiologic studies. *N Eng J Med* 1999; 340: 920-6.
111. Wells AJ. Heart disease from passive smoking in the workplace. *J Am Coll Card* 1998; 31: 1-9.
112. He Y, Lam TH, Li LS, et al. Passive smoking at work as a risk factor for coronary heart disease in Chinese women who have never smoked. *BMJ* 1994; 308: 380-4.
113. Kawachi I, Colditz GA, Speizer FE, et al. A prospective study of passive smoking and coronary heart disease. *Circulation* 1997; 95: 2374-9.
114. Butler, T. L. The relationship of passive smoking to various health outcomes among Seventh Day Adventists in California. 1988. University of California, Los Angeles (Thesis).

115. Svendsen KH, Kuller LH, Martin MJ, Ockene JK. Effects of passive smoking in the Multiple Risk Factor Intervention trial. *Am J Epidemiol* 1987; 126: 783-95.
116. Jackson, R. T. The Auckland heart study. 152-172. 1989. University of Auckland, New Zealand. (Thesis)
117. Muscat JE, Wynder EL. Exposure to environmental tobacco smoke and the risk of heart attack. *Int J Epidemiol* 1995; 715-9.
118. Dobson AJ, Alexander HM, Heller RF, Lloyd DM. Passive smoking and the risk of heart attack or coronary death. *Med J Aust* 1991; 154: 793-7.
119. Steenland K, Thun M, Lally C, Heath C, Jr. Environmental tobacco smoke and coronary heart disease in the American Cancer Society CPS-II cohort. *Circulation* 1996; 94: 622-8.
120. Steenland K. Risk assessment for heart disease and workplace ETS exposure among non-smokers. *Environ Health Perspect* 1999; 107, Suppl 6: 859-63.
121. Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischemic heart disease: an evaluation of evidence. *BMJ* 1997; 315: 973-80.
122. Tunstall-Pedoe H, Brown CA, Woodward M, Tavendale R. Passive smoking by self report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart health study. *J Epidemiol Commun Health* 1995; 49: 139-43.
123. Howard G, Wagenknecht LE. Environmental tobacco smoke and measures of subclinical vascular disease. *Environ Health Perspect* 1999; 107, Suppl 7: 837-40.
124. Roberts KA, Reza AA, Pinkerton KE, Rutledge JC. Effect of environmental tobacco smoke on LDL accumulation in the artery wall. *Circulation* 1996; 94: 2248-53.
125. Mitzoue T, Ueda R, Hino Y, Yoshimura T. Workplace exposure to environmental tobacco smoking and high density lipoprotein cholesterol among non-smokers. *Am J Epidemiol* 1999; 150: 1068-72.
126. Davis J, Shelton L, Watanabe I, Arnold J. Passive smoking affects endothelium and platelets. *Arch Int Med* 1989; 149: 386-9.
127. Feldman J, Shenker I, Etzel R, et al. Passive smoking alters lipids profiles in adolescents. *Pediatrics* 1991; 2: 259-64.

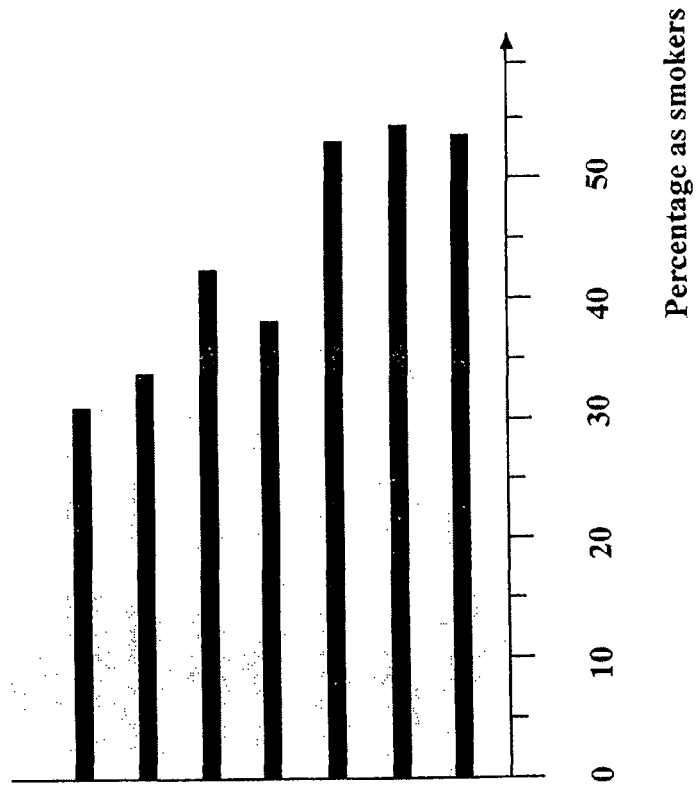
128. Moskowitz WB, Mosteller M, Schieken RM, et al. Lipoprotein and oxygen transport alterations in passive smoking pre adolescent children: the MCV Twin Study. *Circulation* 1990; 81: 586-92.
129. Coultas DB. Health effects of passive smoking. 8. Passive smoking and risk of adult asthma and COPD: An update. *Thorax* 1998; 53: 381-7.
130. Xu X, Li B. Exposure-response relationship between passive smoking and adult pulmonary function. *Am J Resp Crit Care Med* 1995; 151: 41-6.
131. Brunekreef B, Fischer P, Remijn B, van der Lende R, Schouten J, Quanjer P. Indoor air pollution and its effect on pulmonary function in adult non-smoking women. III. Passive smoking and pulmonary function. *Int J Epidemiol* 1985; 14: 227-30.
132. Leuenberger P, Schwartz J, Ackermann-Librich U, et al. Passive smoking exposure in adults and chronic respiratory symptoms (SAPALDIA study). *Am J Resp Crit Care Med* 1994; 150: 1222-8.
133. Nuorti JP, Butler JC, Fairly MM, Harrison LH, et al. Cigarette smoking and invasive pneumococcal pneumonia. *N Eng J Med* 2000; 342: 681-9.
134. Glantz S, Parmley WW. Passive smoking and heart disease: epidemiology, physiology, and biochemistry. *Circulation* 1991; 31: 1-12.
135. Repace JL, Jinot J, Bayard S, Emmons K, Hammond SK. Air nicotine and saliva cotinine as indicators of workplace passive smoking exposure and risk. *Risk Anal* 1998; 18: 71-84.
136. Glantz SA, Parmley WW. Passive smoking and heart disease. *JAMA* 1995; 273: 1047-53.
137. Dybing E, Sanner T. Passive smoking and risk of heart disease (Passiv røyking og risiko for hjerteinfarkt). *Nor J Epidemiol* 1995; 5: 135-40.
138. Sanner T, Dybing E. Health injury from passive smoking (Helseskader ved passiv røyking). *Tidskr Norsk Lægeforen* 1996; 116: 617-20.
139. Public reviews by the Swedish state (Statens offentliga utredningar (SOU). Health on equal terms. National goals for public health. Final review by the National Committee for Public Health (Hälsa på lika villkor - nationella mål for folkhälsan. Slutbetänkande av Nationella folkhälsokommittén). Stockholm: Ministry of Social Affairs (Socialdepartementet), 2000; 3-251.

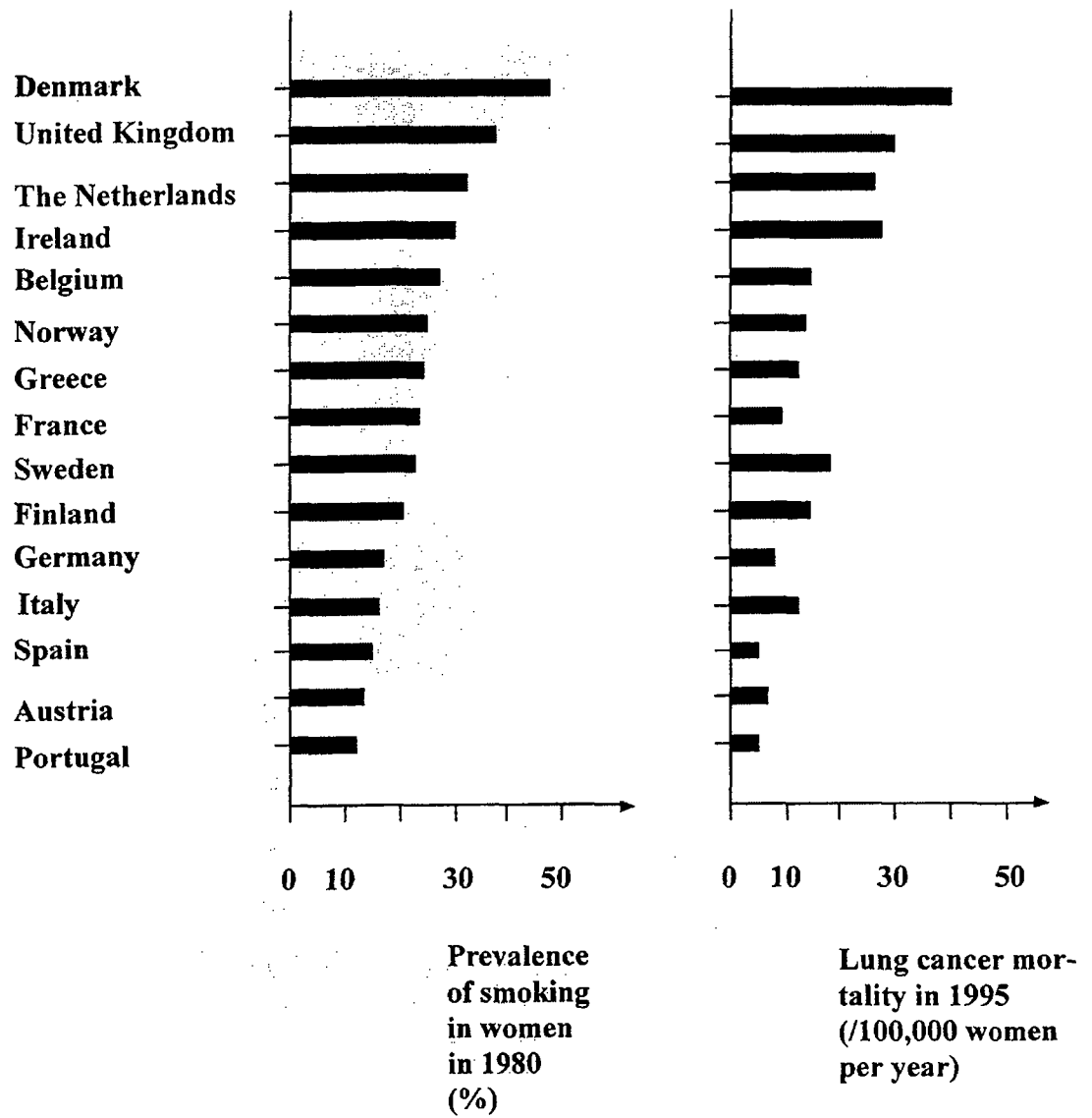
140. Puska P, Korhonen HJ, Uutela A, Helakorpi S, Piha T. Anti-smoking policy in Finland. In: Puska P, Elovainio L, Vertio H, eds. Smokefree Europe. A forum for networks, Helsinki: Finnish Center for Health Promotion, 1997: 26-42.
141. World Health Organization. Health for all year 2000. Copenhagen: World Health Organization, 1985.

Bibliography Chapter 2

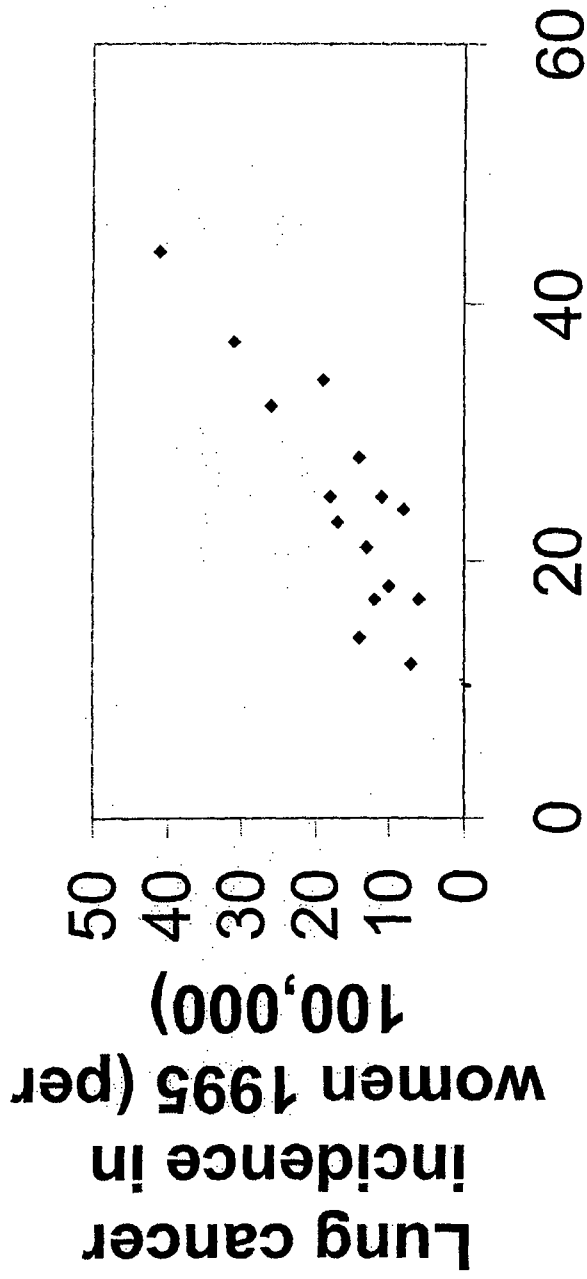
1. Sasco AJ, Dubois G, Ah-Song R, Whidden M (eds). Passive smoking: the health impact. A European report. Europe against cancer programme, 1999, 64 p.
 2. Sasco AJ, d'Harcourt D. EuroLego project. Comparative study of anti-smoking legislation in countries of the European Union. Revised final report. IARC, Lyon, 2001, 377 p.
 3. Dautzenberg B (dir.). La lutte contre le tabagisme en entreprise: le guide. Editions Margaux-Orange, Paris, 2000.
 4. ASH. Smoking in the workplace. UK Edition. 6th September 1999, London.
- Mauffret M, Rousseau-Giral AM, Zaidman C. La loi relative à la lutte contre le tabagisme et l'alcoolisme. Rapport d'évaluation. Instance présidée par Guy Berger. Comité interministériel de l'évaluation des politiques publiques. Premier Ministre. Commissariat Général du Plan, octobre 1999, 180 p.
 - Protection contre le tabagisme et ventilation dans les bars et restaurants. Etude sur le respect du décret n° 92-478 du 29 mai 1992. DDASS des Hauts-de-Seine. Service Santé Environnement, Septembre 1999.
 - Putzu S, Luise R, Masullo V. Italia: Fumo no. Informazioni generali sull'attuale situazione normativa e giurisprudenziale italiana sul 'problema fumo'. CODACONS, Roma, 2000.
 - Sasco AJ, Mélihan-Cheinin P, Ah-Song R, Hirsch A. Evaluation et comparaison de la législation de contrôle du tabagisme dans les Etats membres de l'Union européenne. Rev. Epidémiol. Santé Publ., 1998, 46, 523-531.

Higher manager
Medium manager
Lower manager
Skilled worker
Unskilled worker
Early retirement
Unemployed



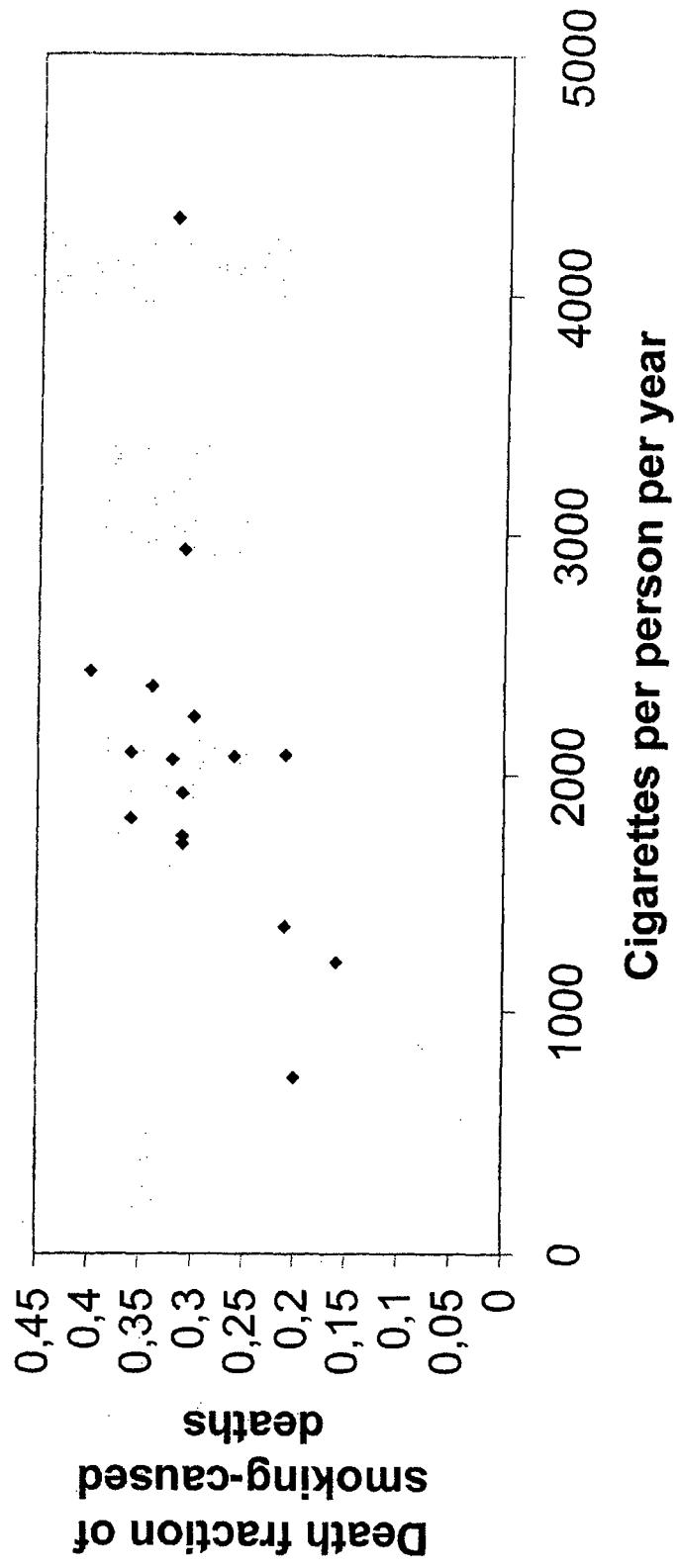


Smoking and lung cancer

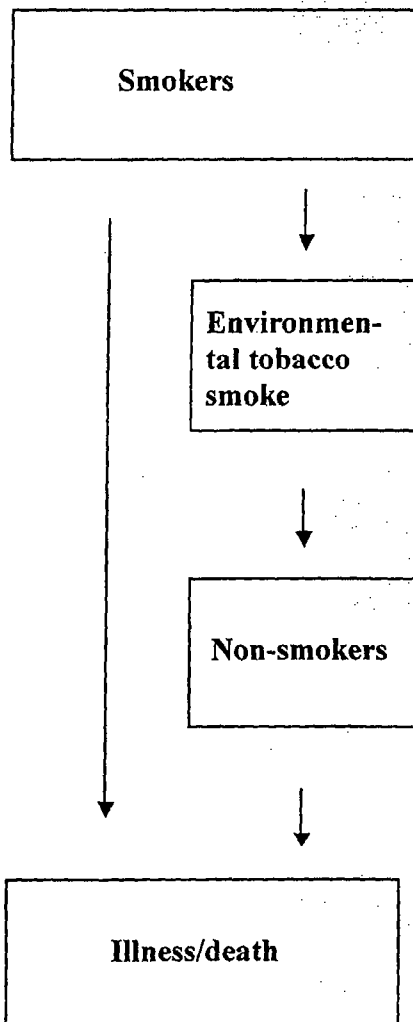


Prevalence of smoking in women (%)

Consumption of cigarettes and fraction of smoking-caused deaths in men at age 35-69 years (1995)



Links from smoking to passive smoking and to tobacco-caused disease and death



Measurements

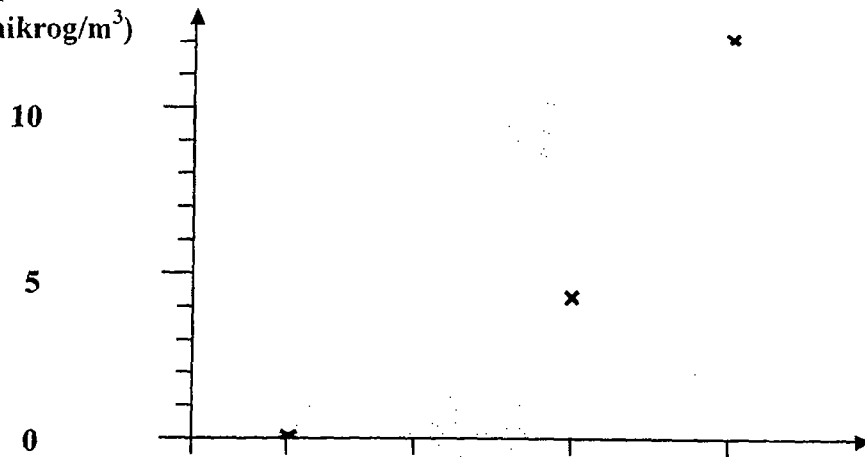
Number of smoked cigarettes per hour/day at site of exposure

Nicotine concentration in air at exposure

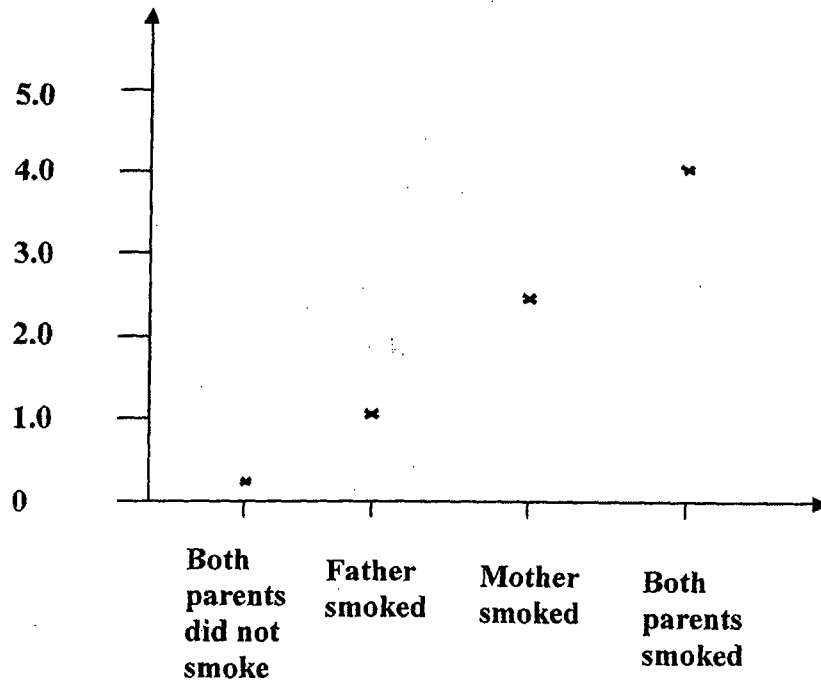
Cotinine concentration in saliva, serum, and urine in non-smokers

Relative risk of acute myocardial infarction, lung cancer, and chronic bronchitis

Nicotine concentration in air (mikrog/m³)



Saliva cotinine concentration (mikrog/L)



Links from smoking to passive smoking and to tobacco-caused disease and death

Society

Smokers in workplaces

Environmental tobacco smoke

Non-smokers in workplaces

Illness/death

Interventions to break the links

Education, mass media campaigns and legislation

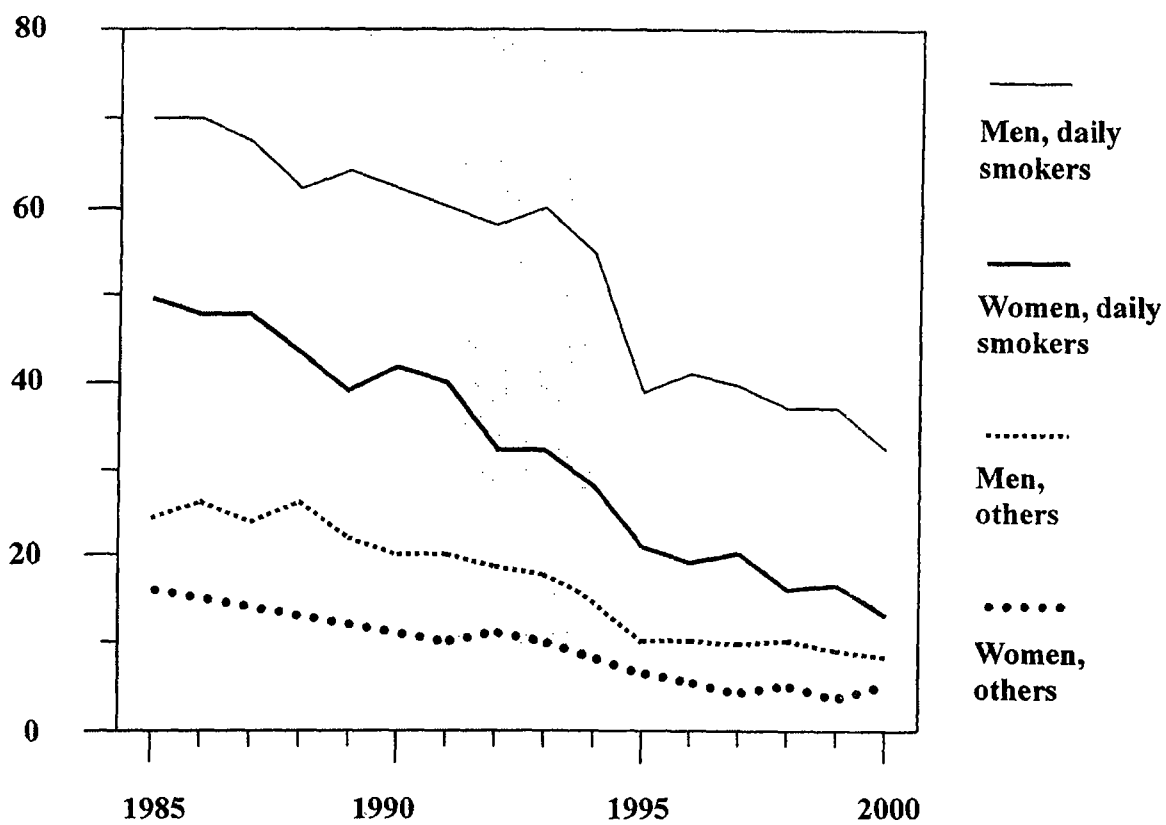
Smoking cessation counseling at workplaces

Restrictions of smoking in workplaces

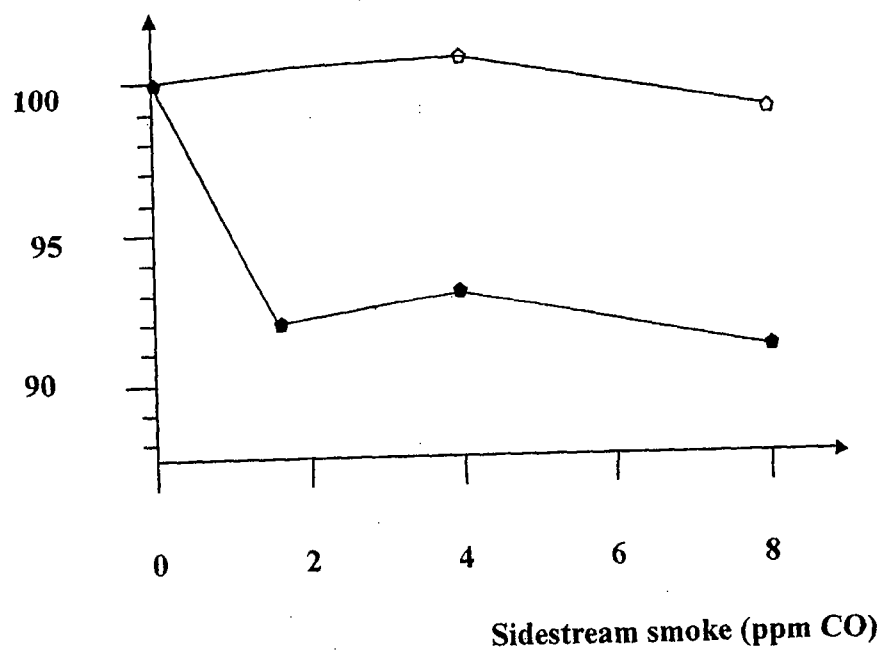
Support for non-smokers right to breath smokefree air as social norm

Litigation for nonsmokers with illness from environmental tobacco smoke

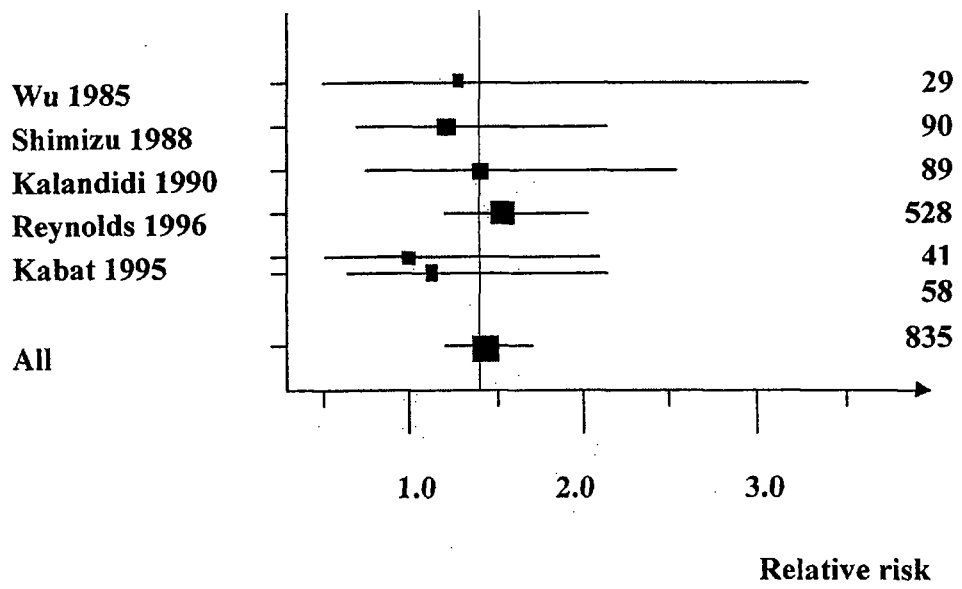
**Workers with daily exposure
to tobacco smoke at work
(%)**

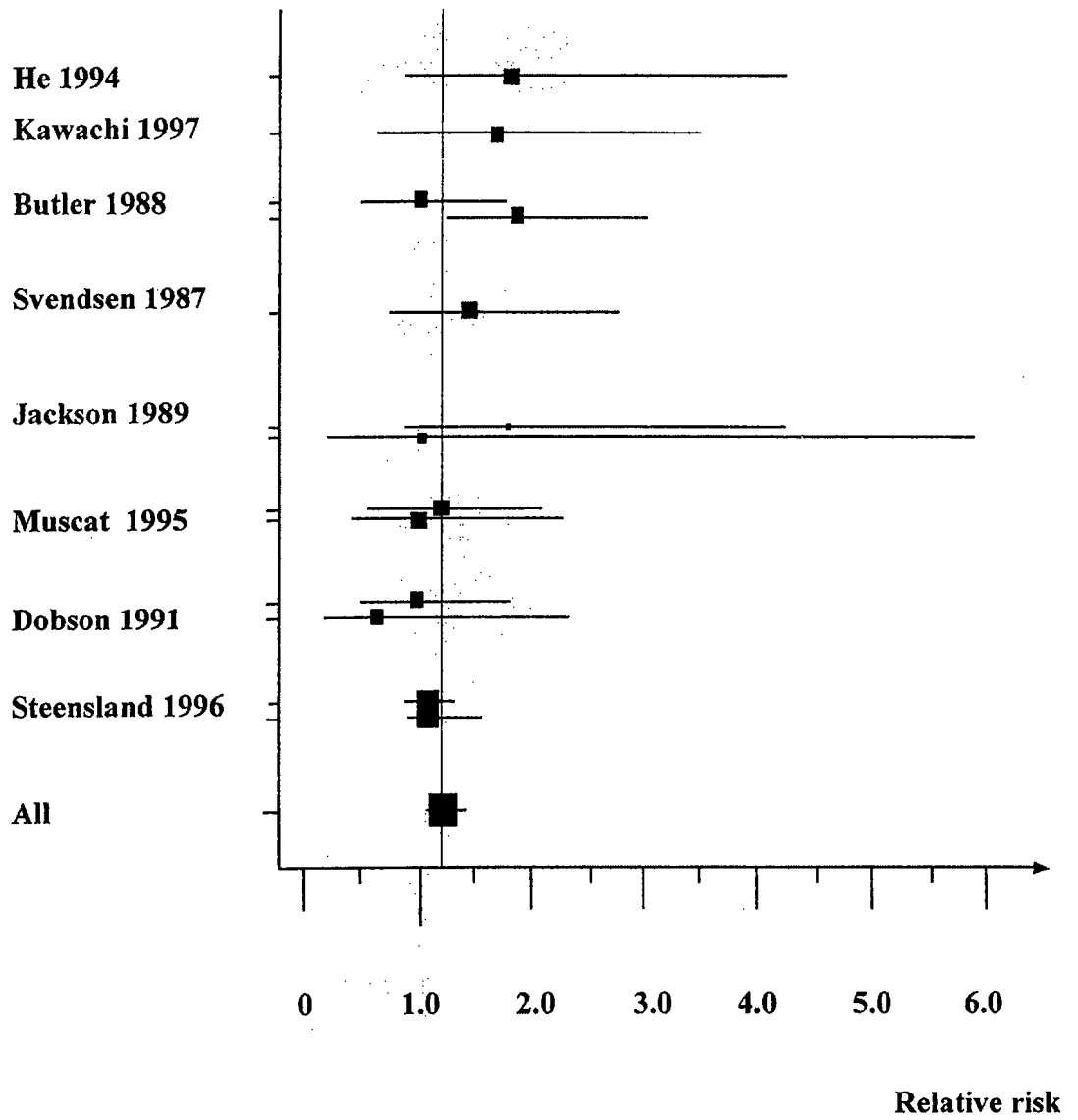


Forced expiratory volume₁
(FEV₁, %)



Number of cases





Risk of tobacco-caused disease

