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Tobacco and Lung Cancer: Epidemiology and Evidence

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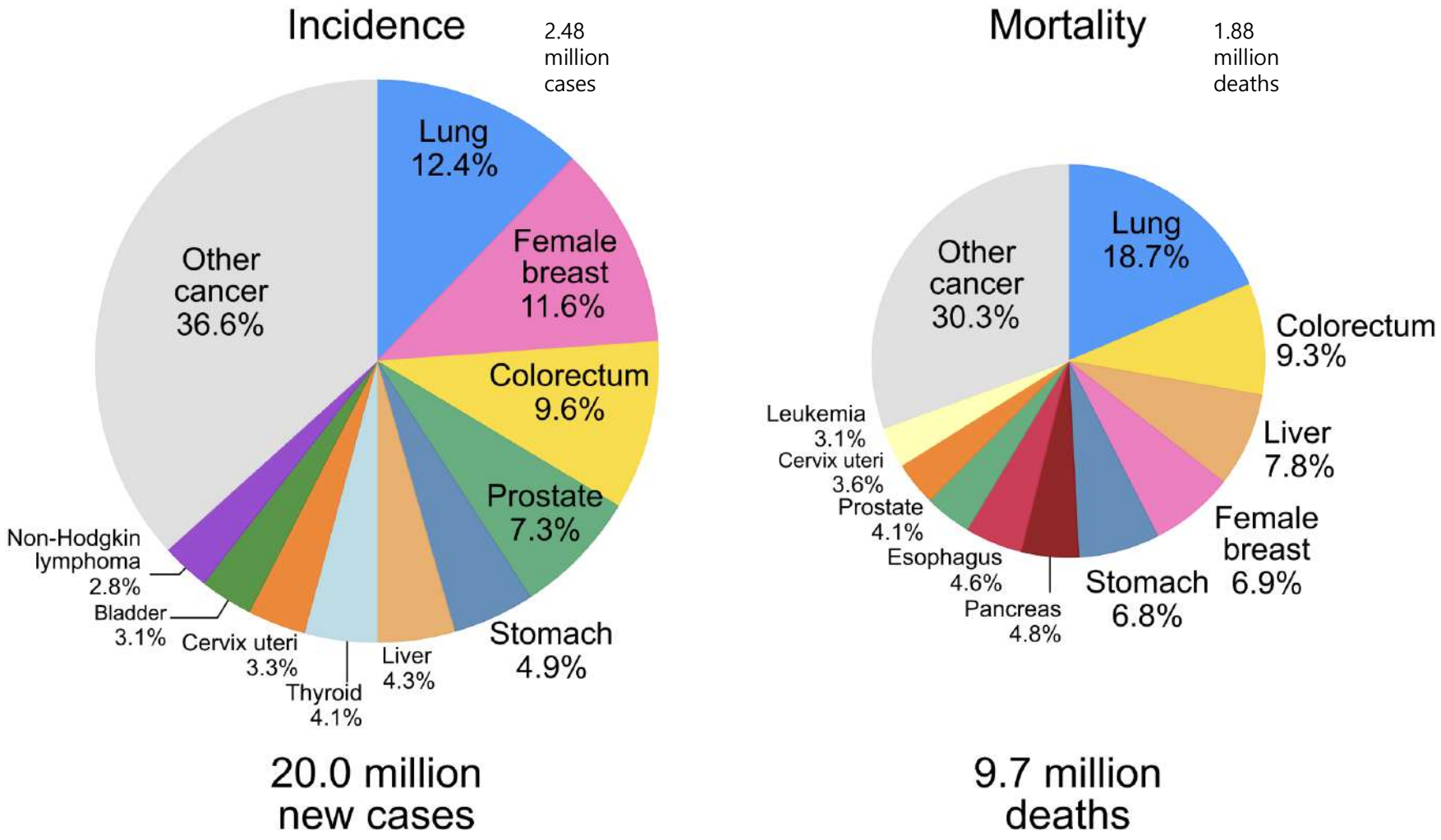
Lung cancer is the most common cancer, and the most common cause of cancer death, in the world



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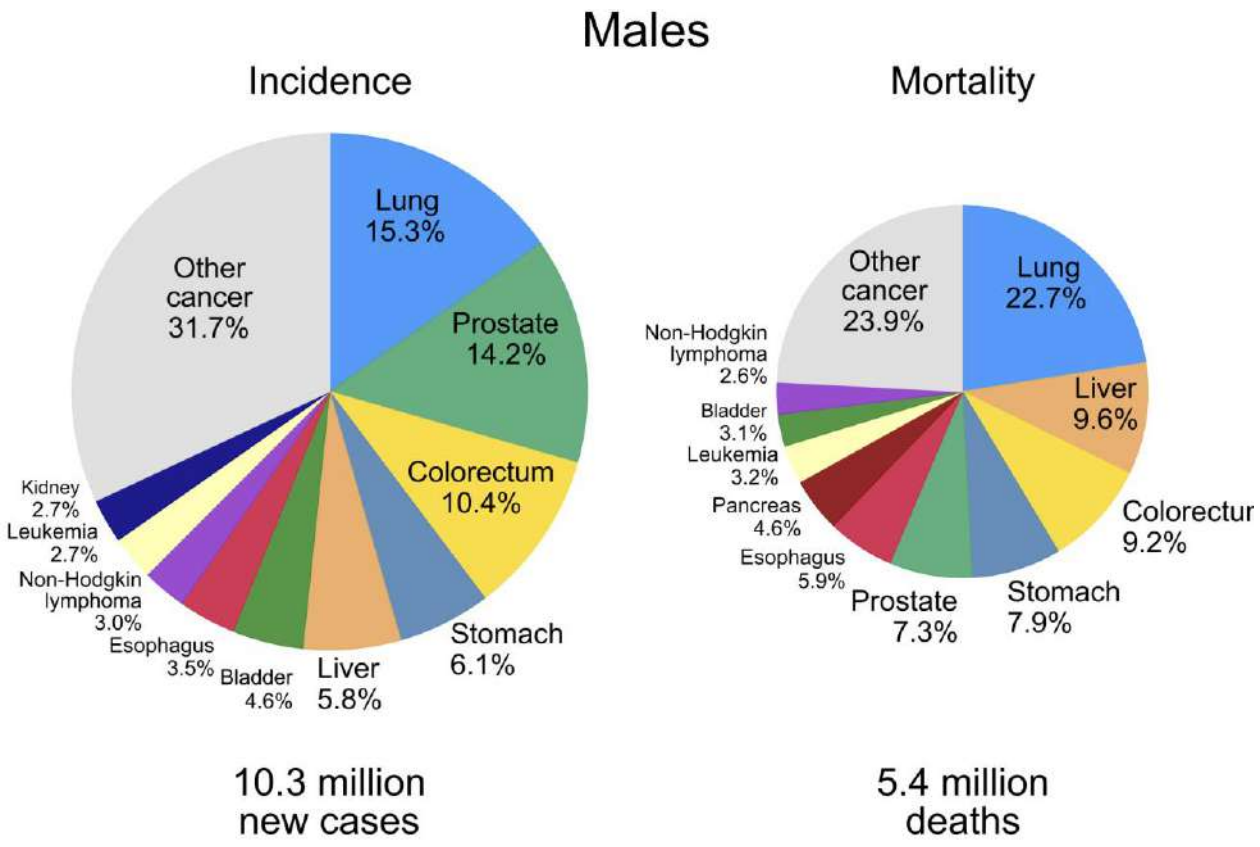
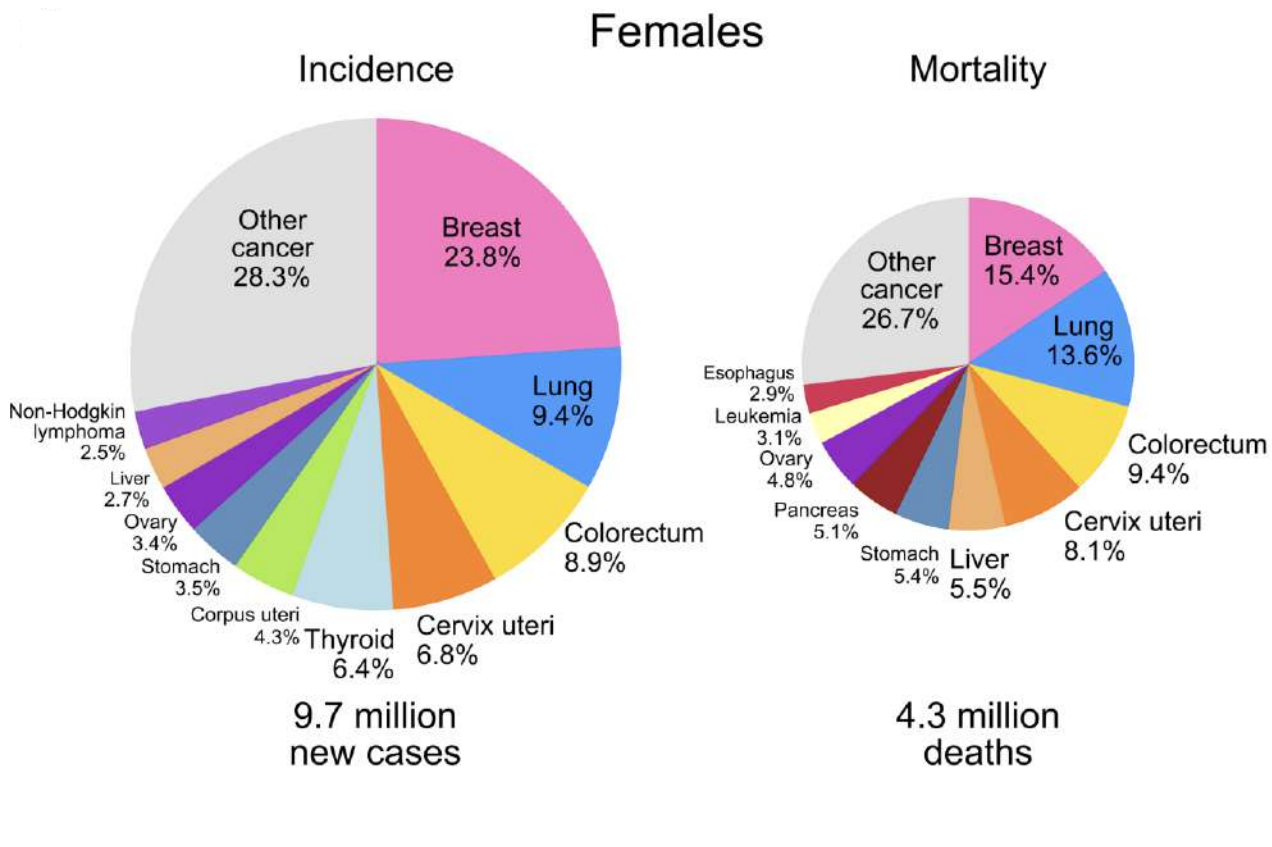
Lung cancer deaths are common in men and women





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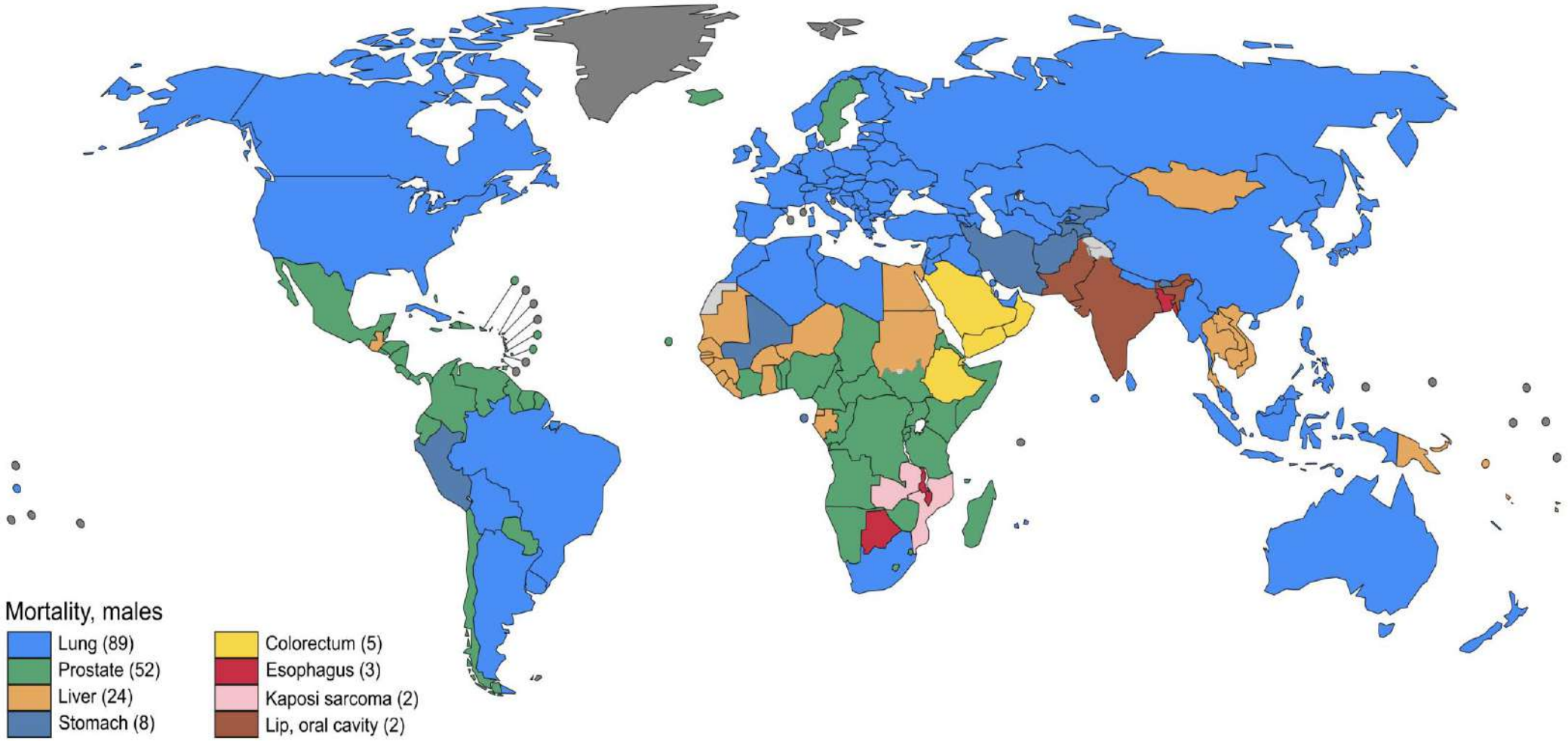
Leading cause of cancer deaths around the world : males



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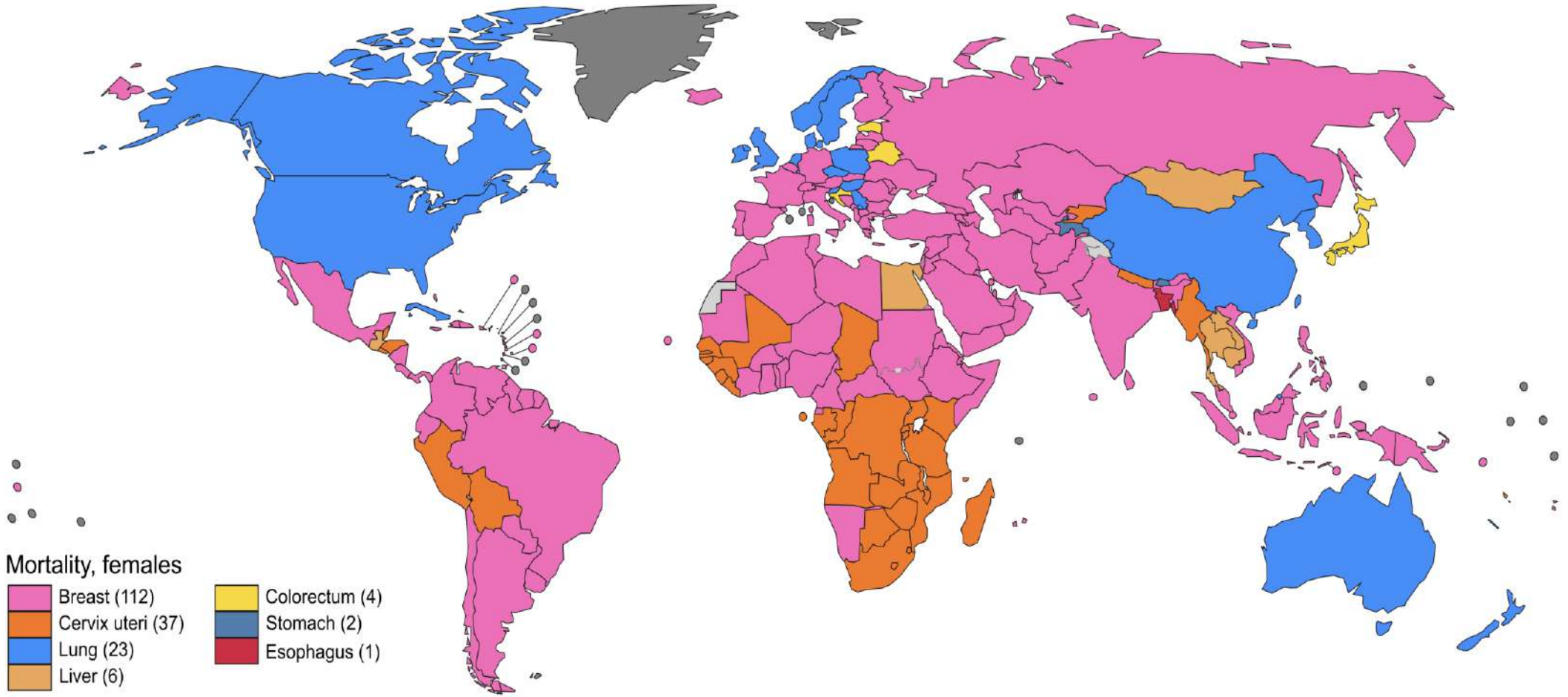
Leading cause of cancer deaths around the world : females



Bray et al. CA: Cancer J Clin 2024; 74: 229-263

Landmark Article
May 27, 1950
(*JAMA* 1950;143:329-336)

Tobacco Smoking as a Possible Etiologic Factor in Bronchiogenic Carcinoma

A Study of Six Hundred and Eighty-Four Proved Cases

Ernest L. Wynder and Evarts A. Graham, M.D.
St. Louis

General Increase.—There is rather general agreement that the incidence of bronchiogenic carcinoma has greatly increased in the last half-century. Statistical studies at the Charity Hospital of New Orleans (Ochsner and DeBakey),[1] the St. Louis City Hospital (Wheeler)[2] and the Veterans Administration Hospital of Hines, Ill. (Avery)[3] have revealed that at these hospitals cancer of the lung is now the most frequent visceral cancer in men.

Autopsy statistics throughout the world show a great increase in the incidence of bronchiogenic carcinoma in relation to cancer in general. Kenneway and Kenneway,[4] in a careful statistical study of death certificates in England and Wales from 1928 to 1945, have presented undoubted evidence of a great increase in deaths from cancer of the lung. In this country statistics compiled by the American Cancer Society show a similar trend during the past two decades.[5]

Tobacco as a Possible Cause of Increase.—The suggestion that smoking, and in particular cigaret smoking, may be important in the production of bronchiogenic carcinoma has been made by many writers on the subject even though well controlled and large scale clinical studies are lacking. Adler[6] in 1912 was one of the first to think that tobacco might play some role in this regard. Tylecote,[7] Hoffman,[8] McNally,[9] Lickint,[10] Arkin and Wagner,[11] Roffo[12] and Maier[13] were just a few of the workers who thought that there was some evidence that tobacco was an important factor in the increase of cancer of the lungs. Müller[14] in 1939, from a careful but limited clinical statistical study, offered good evidence that heavy smoking is an important etiologic factor. In 1941 Ochsner and DeBakey[15] called attention to the similarity of the curve of increased sales of cigarets in this country to the greater prevalence of primary cancer of the lung. They emphasized the possible etiologic relationship of cigaret smoking to this condition. In a recent paper Schrek[16] concluded that there is strong circumstantial evidence that cigaret smoking is an etiologic factor in cancer of the respiratory tract and finds that his data are in agreement with the results of a preliminary report presented by

From the Department of Surgery, Washington University School of Medicine and Barnes Hospital.

This study has been aided by a grant from the American Cancer Society. Other phases of it will be presented in subsequent publications.

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY SEPTEMBER 30 1950

SMOKING AND CARCINOMA OF THE LUNG

PRELIMINARY REPORT

BY

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AND

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Professor of Medical Statistics, London School of Hygiene and Tropical Medicine; Honorary Director of the Statistical Research Unit of the Medical Research Council

In England and Wales the phenomenal increase in the number of deaths attributed to cancer of the lung provides one of the most striking changes in the pattern of mortality recorded by the Registrar-General. For example, in the quarter of a century between 1922 and 1947 the annual number of deaths recorded increased from 612 to 9,287, or roughly fifteenfold. This remarkable increase is, of course, out of all proportion to the increase of population—both in total and, particularly, in its older age groups. Stocks (1947), using standardized death rates to allow for these population changes, shows the following trend: rate per 100,000 in 1901–20, males 1.1, females 0.7; rate per 100,000 in 1936–9, males 10.6, females 2.5. The rise seems to have been particularly rapid since the end of the first world war; between 1921–30 and 1940–4 the death rate of men at ages 45 and over increased sixfold and of women of the same ages approximately threefold. This increase is still continuing. It has occurred, too, in Switzerland, Denmark, the U.S.A., Canada, and Australia, and has been reported from Turkey and Japan.

Many writers have studied these changes, considering whether they denote a real increase in the incidence of the disease or are due merely to improved standards of diagnosis. Some believe that the latter factor can be regarded as wholly, or at least mainly, responsible—for example, Willis (1948), Clemmesen and Busk (1947), and Steiner (1944). On the other hand, Kennaway and Kennaway (1947) and Stocks (1947) have given good reasons for believing that the rise is at least partly real. The latter, for instance, has pointed out that "the increase of certified respiratory cancer mortality during the past 20 years has been as rapid in country districts as in the cities with the best diagnostic facilities, a fact which does not support the view that such increase merely reflects improved diagnosis of cases previously certified as bronchitis or other respiratory affections." He also draws attention to differences in mortality between some of the large cities of England and Wales, differences which it is difficult to explain in terms of diagnostic standards.

The large and continued increase in the recorded deaths even within the last five years, both in the national figures and in those from teaching hospitals, also makes it hard to believe that improved diagnosis is entirely responsible. In short, there is sufficient reason to reject that factor as the whole explanation, although no one would deny that it may well have been contributory. As a corollary, it is right and proper to seek for other causes.

Possible Causes of the Increase

Two main causes have from time to time been put forward: (1) a general atmospheric pollution from the exhaust fumes of cars, from the surface dust of tarred roads, and from gas-works, industrial plants, and coal fires; and (2) the smoking of tobacco. Some characteristics of the former have certainly become more prevalent in the last 50 years, and there is also no doubt that the smoking of cigarettes has greatly increased. Such associated changes in time can, however, be no more than suggestive, and until recently there has been singularly little more direct evidence. That evidence, based upon clinical experience and records, relates mainly to the use of tobacco. For instance, in Germany, Müller (1939) found that only 3 out of 86 male patients with cancer of the lung were non-smokers, while 56 were heavy smokers, and, in contrast, among 86 "healthy men of the same age groups" there were 14 non-smokers and only 31 heavy smokers. Similarly, in America, Schrek and his co-workers (1950) reported that 14.6% of 82 male patients with cancer of the lung were non-smokers, against 23.9% of 522 male patients admitted with cancer of sites other than the upper respiratory and digestive tracts. In this country, Thelwall Jones (1949—personal communication) found 8 non-smokers in 82 patients with proved carcinoma of the lung, compared with 11 in a corresponding group of patients with diseases other than cancer; this difference is slight, but it is more striking that there were 28 heavy smokers in the cancer group, against 14 in the comparative group.

Clearly none of these small-scale inquiries can be accepted as conclusive, but they all point in the same direction. Their evidence has now been borne out by the results of a large-scale inquiry undertaken in the U.S.A. by Wynder and Graham (1950).

Wynder and Graham found that of 605 men with epidermoid, undifferentiated, or histologically unclassified types of bronchial carcinoma only 1.3% were "non-smokers"—that is, had averaged less than one cigarette a day for the last 20 years—whereas 51.2% of them had smoked more than 20 cigarettes a day over the same

Risk of lung cancer was found to be 25 times higher in smokers than non-smokers

TABLE II.—*Comparison Between Lung-carcinoma Patients and Non-cancer Patients Selected as Controls, With Regard to Sex, Age, Social Class, and Place of Residence*

Age	No. of Lung-carcinoma Patients		No. of Non-cancer Control Patients	
	M	F	M	F
25– ..	2	1	2	1
30– ..	6	0	6	0
35– ..	18	3	18	3
40– ..	36	4	36	4
45– ..	87	10	87	10
50– ..	130	11	130	11
55– ..	145	9	145	9
60– ..	109	9	109	9
65– ..	88	9	89*	9
70–74 ..	28	4	27*	4
All ages	649	60	649	60

Social Class (Registrar-General's Categories. Men Only)	No. of Lung-carcinoma Patients	No. of Non-cancer Patients
I and II ..	77	87
III	388	396
IV and V ..	184	166
All classes ..	649	649
Place of residence		
County of London	330	377
Outer London	203	231
Other county borough ..	23	16
Urban district	95	54
Rural district ..	43	27
Abroad or in Services ..	15	4
Total (M + F) ..	709	709

* One control patient was selected, in error, from the wrong age group.

Smokers and Non-smokers

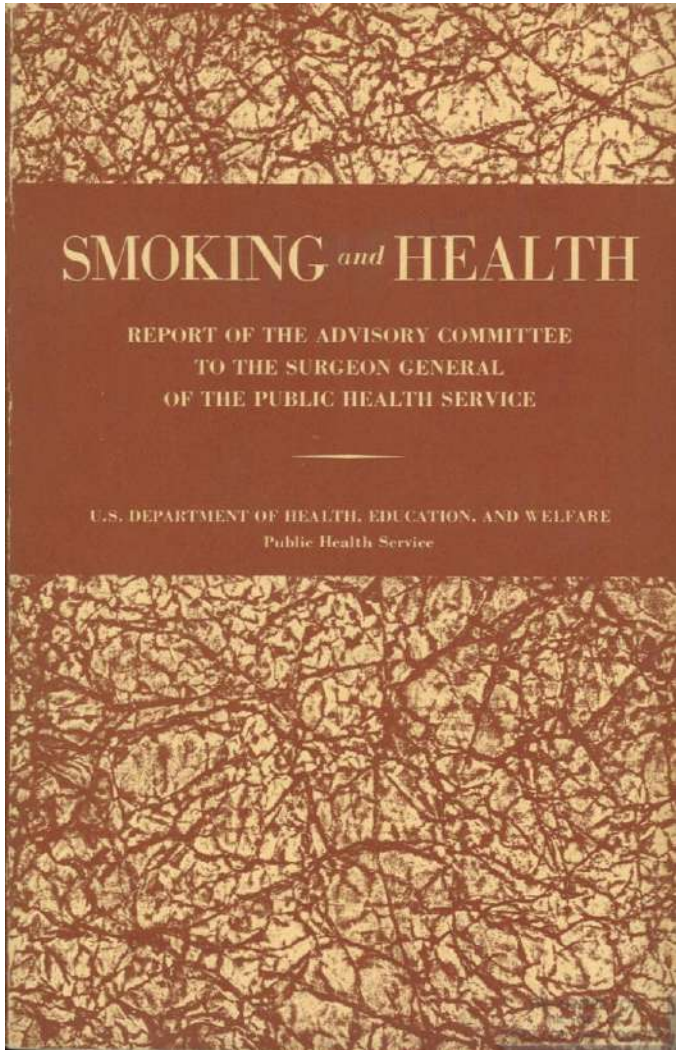
The simplest comparison that can be made to show whether there is any association at all between smoking and carcinoma of the lung is that between the proportion of lung-carcinoma patients who have been smokers and the proportion of smokers in the comparable group of subjects without carcinoma of the lung. Such a comparison is shown in Table IV.

TABLE IV.—*Proportion of Smokers and Non-smokers in Lung-carcinoma Patients and in Control Patients with Diseases Other Than Cancer*

Disease Group	No. of Non-smokers	No. of Smokers	Probability Test
Males: Lung-carcinoma patients (649)	2 (0·3%)	647	P (exact method) = 0·00000064
Control patients with diseases other than cancer (649) ..	27 (4·2%)	622	
Females: Lung-carcinoma patients (60)	19 (31·7%)	41	$\chi^2 = 5{\cdot}76$; n = 1 $0{\cdot}01 < P < 0{\cdot}02$
Control patients with diseases other than cancer (60) ..	32 (53·3%)	28	

Doll and Hill, BMJ 1950; 2: 739-748

1964 U.S. Surgeon General's Report



"All the News That's Fit to Print"

NEWS SUMMARY AND INDEX, PAGE 95

The New York

VOL. CXIII . No. 38,704. NEW YORK, SUNDAY, JANUARY 12, 1964.

CIGARETTES PERIL HEALTH, U. S. REPORT CONCLUDES; 'REMEDIAL ACTION' URGED

CANCER LINK CITED

Smoking Is Also Found 'Important' Cause of Chronic Bronchitis

Committee's summary of its findings, Pages 64 and 65.

By WALTER SULLIVAN
Special to The New York Times

WASHINGTON, Jan. 11—The long-awaited Federal report on the effects of smoking found today that the use of cigarettes contributed so substantially to the American death rate that "appropriate remedial action" was called for.

The committee that made the report gave no specific recommendations for action. But health officials said that possible steps might include educational campaigns, the requirement that cigarette packages carry warnings and control of advertising.

The report dealt a severe blow to the rear-guard action fought in recent years by the tobacco industry. It dismissed, one by one, the arguments raised to

Associated Press Wirephoto

DISCUSSES SMOKING REPORT: Dr. Luther Terry, the Surgeon General, at news conference held in Washington.

Johnson Chides the G.O.P. For Opposing His Budget

By WARREN WEAVER JR.
Special to The New York Times

SIX-PHASE INQUIRY ON ASSASSINATION CHARTED BY PANEL

Aides Chosen for Detailed Study of Kennedy Slaying and Security Agencies

By ANTHONY LEWIS
Special to The New York Times

WASHINGTON, Jan. 11—The staff of the commission investigating President Kennedy's assassination has divided its job into six broad areas of inquiry.

One covers every detail of Lee Oswald's activities on the day of the assassination, Nov. 22. Oswald was charged with the crime.

A second topic is the life and background of Oswald—an attempt to reconstruct his associations and ideas and psychology.

Oswald's career in the Marine Corps and his stay in the Soviet Union will be handled separately as a third.

His murder in the Dallas police station will be the fourth subject, including all the controversial questions of how it was allowed to happen.

Fifth will be the story of Jack Ruby, the nightclub operator who slipped into the police station and shot Oswald. This will be a particularly delicate subject because of possible conflict with Ruby's trial.

Study of Agencies

Finally, the staff will inquire exhaustively into the procedures used to protect President Kennedy. This will involve a scrutiny of the performances of the Secret Service, the Federal Bu-

U.S. AND BORDERS PLEDGE

FUNERAL PROCESSION I
bearing the body of a Panar

Mortality in relation to smoking: 50 years' observations on male British doctors

Richard Doll, Richard Peto, Jillian Boreham, Isabelle Sutherland

Abstract

Objective To compare the hazards of cigarette smoking in men who formed their habits at different periods, and the extent of the reduction in risk when cigarette smoking is stopped at different ages.

Design Prospective study that has continued from 1951 to 2001.

Setting United Kingdom.

Participants 34 439 male British doctors. Information about their smoking habits was obtained in 1951, and periodically thereafter; cause specific mortality was monitored for 50 years.

Main outcome measures Overall mortality by smoking habit, considering separately men born in different periods.

Results The excess mortality associated with smoking chiefly involved vascular, neoplastic, and respiratory diseases that can be caused by smoking. Men born in 1900-1930 who smoked only cigarettes and continued smoking died on average about 10 years younger than lifelong non-smokers. Cessation at age 60, 50, 40, or 30 years gained, respectively, about 3, 6, 9, or 10 years of life expectancy. The excess mortality associated with cigarette smoking was less for men born in the 19th century and was greatest for men born in the 1920s. The cigarette smoker versus non-smoker probabilities of dying in middle age (35-69) were 42% *v* 24% (a twofold death rate ratio) for those born in 1900-1909, but were 43% *v* 15% (a threefold death rate ratio) for those born in the 1920s. At older ages, the cigarette smoker versus non-smoker probabilities of surviving from age 70 to 90 were 10% *v* 12% at the death rates of the 1950s (that is, among men born around the 1870s) but were 7% *v* 33% (again a threefold death rate ratio) at the death rates of the 1990s (that is, among men born around the 1910s).

Conclusion A substantial progressive decrease in the mortality rates among non-smokers over the past half century (due to prevention and improved treatment of disease) has been wholly outweighed, among cigarette smokers, by a progressive increase in the smoker *v* non-smoker death rate ratio due to earlier and more intensive use of cigarettes. Among the men born around 1920, prolonged cigarette smoking from early adult life tripled age specific mortality rates, but cessation at age 50 halved the hazard, and cessation at age 30 avoided almost all of it.

Introduction

During the 19th century much tobacco was smoked in pipes or as cigars and little was smoked as cigarettes, but during the first few decades of the 20th century the consumption of manufactured cigarettes increased greatly.[1] This led eventually to a rapid increase in male lung cancer, particularly in the United Kingdom (where the disease became by the 1940s a major cause of death). Throughout the first half of the 20th century the hazards of smoking had remained largely unsuspected.[1] Around the middle of the century, however, several case-control studies of lung cancer were published in Western Europe[2-6] and North America,[7-10] leading to the conclusion in 1950 that smoking was "a cause, and an important cause" of the disease.[5]

1951 prospective study

This discovery stimulated much further research into the effects of smoking (not only on lung cancer but also on many other diseases), including a UK prospective study of smoking and death among British doctors that began in 1951 and has now continued for 50 years.[11-17] The decision that this study would be conducted among doctors was taken partly because it was thought that doctors might take the trouble to describe their own smoking habits accurately, but principally because their subsequent mortality would be relatively easy to follow, as they had to keep their names on the medical register if they were to continue to practise. Moreover, as most doctors would themselves have access to good medical care, the medical causes of any deaths among them should be reasonably accurately certified.

The 1951 study has now continued for much longer than originally anticipated, as the doctors did indeed prove easy to follow, and they provided further information about any changes in their smoking habits along the way (in 1957, 1966, 1971, 1978, and 1991). A final questionnaire was sent out in 2001.

By 1954 the early findings[11] had confirmed prospectively the excess of lung cancer among smokers that had been seen in the retrospective studies.[2-10] Findings on cause specific mortality in relation to smoking were published after four periods of follow up (after four years,[12] 10 years,[13] 20 years,[14 15] and 40 years[17]). The early results from this study,[12-14] together with those from several others that began soon after, showed that smoking was associated with mortality from many different diseases. Indeed, although smoking was a cause of the large majority of all UK lung cancer deaths, lung cancer accounted for less than half of the excess mortality among smokers.

As recently as the 1980s, however, the full eventual effects on overall mortality of smoking substantial numbers of cigarettes throughout adult life were still greatly underestimated, as no population that had done this had yet been followed to the end of its life span. The present report of the 50 year results chiefly emphasises the effects on overall mortality (subdivided by period of birth) of continuing to smoke cigarettes and of ceasing to do so at various ages.

50-year follow-up of smoking and non-smoking physicians in the U.K.

Cause of death	No of deaths 1951-2001	Age standardised mortality rate per 1000 men/year								Standardised tests for trend (χ^2 on 1 df)*	
		Lifelong non-smokers	Cigarette smokers (no other smoking habit previously reported)					Other smokers			
			Former	Current	Current (cigarettes/day) 1-14	15-24	≥25	Former	Current	N/X/C†	Amount†
Cancer of lung	1052	0.17	0.68	2.49	1.31	2.33	4.17	0.71	1.30	394	452
Cancers of mouth, pharynx, larynx, oesophagus	340	0.09	0.26	0.60	0.36	0.47	1.06	0.30	0.47	68	83
All other neoplasms	3893	3.34	3.72	4.69	4.21	4.67	5.38	3.66	4.22	32	36
Chronic obstructive pulmonary disease	640	0.11	0.64	1.56	1.04	1.41	2.61	0.45	0.64	212	258
Other respiratory disease	1701	1.27	1.70	2.39	1.76	2.65	3.11	1.69	1.67	44	70
Ischaemic heart disease	7628	6.19	7.61	10.01	9.10	10.07	11.11	7.24	7.39	138	133
Cerebrovascular disease	3307	2.75	3.18	4.32	3.76	4.35	5.23	3.24	3.28	48	65
Other vascular (including respiratory heart) disease	3052	2.28	2.83	4.15	3.37	4.40	5.33	2.99	3.08	77	94
Other medical conditions	2565	2.26	2.47	3.49	2.94	3.33	4.60	2.49	2.44	34	54
External causes	891	0.71	0.75	1.13	1.08	0.79	1.76	0.89	0.92	17	27
Cause unknown	277	0.17	0.28	0.52	0.39	0.57	0.59	0.25	0.31	16	24
All cause (No of deaths)	25 346	19.38 (2917)	24.15 (5354)	35.40 (4680)	29.34 (1450)	34.79 (1725)	45.34 (1505)	23.96 (5713)	25.70 (6682)	699	869

*Values of χ^2 on one degree of freedom for trend between three or four groups: values ≥15 correspond to P<0.0001.
†N/X/C compares three groups: lifelong non-smokers, former cigarette smokers, and current cigarette smokers. Amount compares four groups: never smoked regularly, and current cigarette smokers consuming 1-14, 15-24 or ≥25 cigarettes/day when last asked.

Effect of cigarette smoking on life-expectancy

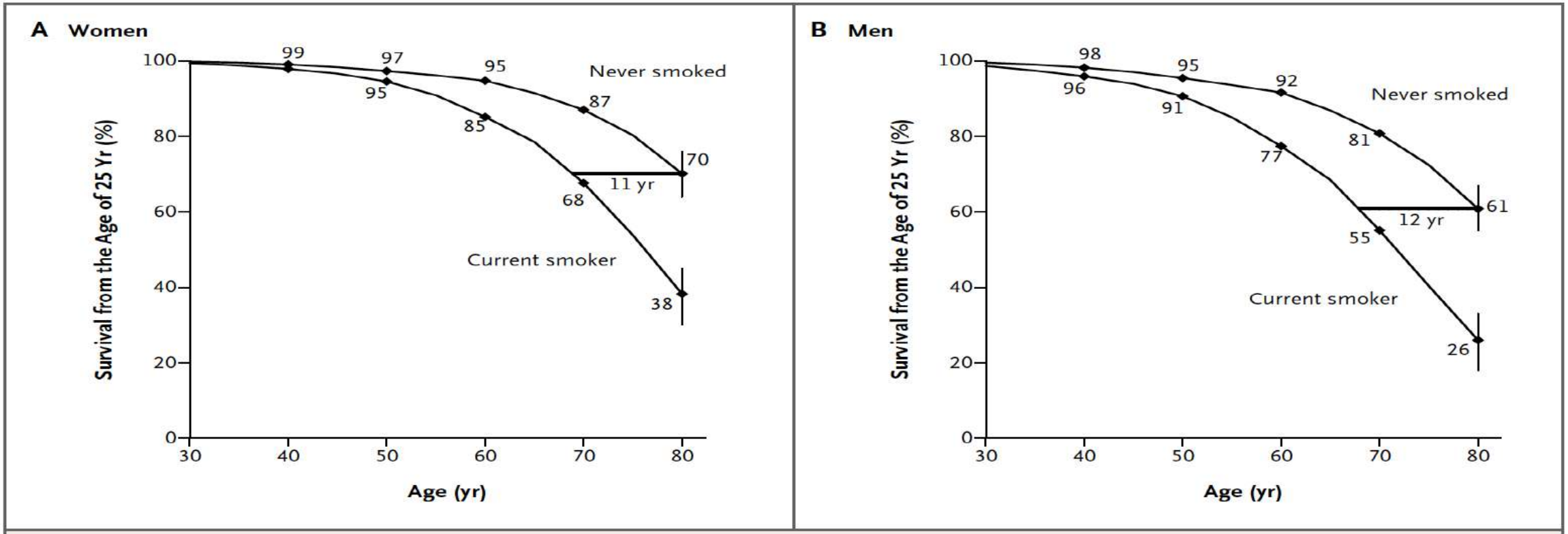


Figure 2. Survival Probabilities for Current Smokers and for Those Who Never Smoked among Men and Women 25 to 80 Years of Age.
The vertical lines at 80 years of age represent the 99% confidence intervals for cumulative survival probabilities, as derived from the standard errors estimated with the use of the jackknife procedure. Survival probabilities have been scaled from the National Health Interview Survey to the U.S. rates of death from all causes at these ages for 2004,[13,16] with adjustment for differences in age, educational level, alcohol consumption, and adiposity (body-mass index).

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ARTICLES

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Health effects associated with smoking: a Burden of Proof study

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As a leading behavioral risk factor for numerous health outcomes, smoking is a major ongoing public health challenge. Although evidence on the health effects of smoking has been widely reported, few attempts have evaluated the dose–response relationship between smoking and a diverse range of health outcomes systematically and comprehensively. In the present study, we re-estimated the dose–response relationships between current smoking and 36 health outcomes by conducting systematic reviews up to 31 May 2022, employing a meta-analytic method that incorporates between-study heterogeneity into estimates of uncertainty. Among the 36 selected outcomes, 8 had strong-to-very-strong evidence of an association with smoking, 21 had weak-to-moderate evidence of association and 7 had no evidence of association. By overcoming many of the limitations of traditional meta-analyses, our approach provides comprehensive, up-to-date and easy-to-use estimates of the evidence on the health effects of smoking. These estimates provide important information for tobacco control advocates, policy makers, researchers, physicians, smokers and the public.

Among both the public and the health experts, smoking is recognized as a major behavioral risk factor with a leading attributable health burden worldwide. The health risks of smoking were clearly outlined in a canonical study of disease rates (including lung cancer) and smoking habits in British doctors in 1950 and have been further elaborated in detail over the following seven decades[1,2]. In 2005, evidence of the health consequences of smoking galvanized the adoption of the first World Health Organization (WHO) treaty, the Framework Convention on Tobacco Control, in an attempt to drive reductions in global tobacco use and second-hand smoke exposure[3]. However, as of 2020, an estimated 1.18 billion individuals globally were current smokers and 7 million deaths and 177 million disability-adjusted life-years were attributed to smoking, reflecting a persistent public health challenge[4]. Quantifying the relationship between smoking and various important health outcomes—in particular, highlighting any significant dose–response relationships—is crucial to understanding the attributable health risk experienced by these individuals and informing responsive public policy.

Existing literature on the relationship between smoking and specific health outcomes is prolific, including meta-analyses, cohort studies and case–control studies analyzing the risk of outcomes such as lung cancer[5–7], chronic obstructive pulmonary disease (COPD)[8–10] and ischemic heart disease[11–14] due to smoking. There are few if any attempts, however, to systematically and comprehensively evaluate the landscape of evidence on smoking risk across a diverse range of health outcomes, with most current research focusing on risk or attributable burden of smoking for a specific condition[7,15], thereby missing the opportunity to provide a comprehensive picture of the health risk experienced by smokers. Furthermore, although evidence surrounding specific health outcomes, such as lung cancer, has generated widespread consensus, findings about the attributable risk of other outcomes are much more heterogeneous and inconclusive[16–18]. These studies also vary in their risk definitions, with many comparing dichotomous exposure measures of ever smokers versus nonsmokers[19,20]. Others examine the distinct risks of current smokers and former smokers compared with never smokers[21–23]. Among the studies that do analyze dose–response relationships, there is large variation in the units and dose categories used in reporting their findings (for example, the use of pack-years or cigarettes per day)[24,25], which complicates the comparability and consolidation of evidence. This, in turn, can obscure data that could inform personal health choices, public health practices and policy measures. Guidance on the health risks of smoking, such as the *Surgeon General's Reports* on smoking[26,27], is often based on experts' evaluation of heterogenous evidence, which, although extremely useful and well suited to carefully consider nuances in the evidence, is fundamentally subjective.

The present study, as part of the Global Burden of Diseases, Risk Factors, and Injuries Study (GBD) 2020, re-estimated the continuous dose–response relationships (the mean risk functions and associated uncertainty estimates) between current smoking and 36 health outcomes (Supplementary Table 1) by identifying input studies using a systematic review approach and employing a meta-analytic method[28]. The 36 health outcomes that were selected based on existing evidence of a relationship included 16 cancers (lung cancer, esophageal cancer, stomach cancer, leukemia, liver cancer, laryngeal cancer, breast cancer, cervical cancer, colorectal

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The association between smoking and respiratory tract cancers is strong and unequivocal

Table 2 | Strength of the evidence for the relationship between current smoking and the 36 health outcomes analyzed

Risk-outcome pair	Risk unit	Mean risk at different exposure levels				85th percentile risk level	Mean risk at 85th percentile risk level	ROSs	Average BPRF	Average increased risk (%)	Star rating	Pub. bias	No. of studies
		5	10	20	40								
Laryngeal cancer	Pack-years	2.30 (1.88, 2.84)	3.77 (2.73, 5.30)	7.25 (4.48, 12.05)	14.62 (7.62, 29.11)	50.50	17.73 (8.82, 37.07)	1.56	4.75	374.95	5	0	5
Aortic aneurism (ref. age: 55–59 years)	Cigarettes per day	2.52 (1.79, 3.60)	3.78 (2.31, 6.33)	5.39 (2.89, 10.36)	6.22 (3.17, 12.64)	30.00	6.08 (3.13, 12.27)	0.92	2.50	149.73	5	0	14
Peripheral artery disease (ref. age: 60–64 years)	Cigarettes per day	2.52 (1.67, 3.90)	3.80 (2.10, 7.14)	5.69 (2.62, 12.94)	7.82 (3.13, 20.68)	31.25	7.16 (2.98, 18.16)	0.86	2.37	136.53	5	0	6
Lung cancer	Pack-years	1.58 (1.19, 2.15)	2.48 (1.40, 4.53)	5.11 (1.84, 14.99)	11.62 (2.49, 58.73)	50.88	13.42 (2.63, 74.59)	0.73	2.07	106.66	5	1	78
Other pharynx cancer	Pack-years	1.65 (1.30, 2.13)	2.20 (1.51, 3.30)	3.02 (1.77, 5.30)	3.89 (2.02, 7.78)	63.75	4.72 (2.24, 10.45)	0.65	1.92	92.26	5	0	8

Dai et al. Nature Medicine 2022; 28: 2045-2055

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We found a very strong and significant harmful relationship between pack-years of current smoking and the RR of lung cancer (Fig. 1b). The mean RR of lung cancer at 20 pack-years of smoking was 5.11 (95% uncertainty interval (UI) inclusive of between-study heterogeneity = 1.84–14.99). At 50.88 pack-years (85th percentile of exposure), the mean RR of lung cancer was 13.42 (2.63–74.59). See Table 2 for mean RRs at other exposure levels. The BPRF, which represents the most conservative interpretation of the evidence (Fig. 1a), suggests that smoking in the 15th–85th percentiles of exposure increases the risk of lung cancer by an average of 107%, yielding an ROS of 0.73.

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How Tobacco Smoke Causes Disease

The Biology and Behavioral Basis for Smoking-Attributable Disease

A Report of the Surgeon General

U.S. Department of Health and Human Services

Carcinogens in mainstream tobacco smoke

Table 5.1 IARC evaluations of carcinogens in mainstream cigarette smoke

Carcinogen[a]	Quantity (per cigarette)	IARC evaluations of evidence of carcinogenicity in humans: In animals	In humans	IARC group[b]	*IARC Monograph*[c] (volume, year)
Polycyclic aromatic hydrocarbons					
Benz[*a*]anthracene	20–70 ng	Sufficient		2A	*32*, 1983; *S7*, 1987
Benzo[*b*]fluoranthene	4–22 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Benzo[*j*]fluoranthene	6–21 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Benzo[*k*]fluoranthene	6–12 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Benzo[*a*]pyrene	8.5–17.6 ng	Sufficient	Limited	1	*32*, 1983; *S7*, 1987; *92*, in press
Dibenz[*a,h*]anthracene	4 ng	Sufficient		2A	*32*, 1983; *S7*, 1987
Dibenzo[*a,i*]pyrene	1.7–3.2 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Dibenzo[*a,e*]pyrene	Present	Sufficient		2B	*32*, 1983; *S7*, 1987
Indeno[*1,2,3-cd*]pyrene	4–20 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
5-methylchrysene	ND–0.6 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Heterocyclic compounds					
Furan	20–40 µg	Sufficient		2B	*63*, 1995a
Dibenz[*a,h*]acridine	ND–0.1 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Dibenz[*a,j*]acridine	ND–10 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Dibenzo[*c,g*]carbazole	ND–0.7 ng	Sufficient		2B	*32*, 1983; *S7*, 1987
Benzo[*b*]furan	Present	Sufficient		2B	*63*, 1995a
***N*-nitrosamines**					
N-nitrosodimethylamine	0.1–180 ng	Sufficient		2A	*17*, 1978; *S7*, 1987
N-nitrosoethylmethylamine	ND–13 ng	Sufficient		2B	*17*, 1978; *S7*, 1987
N-nitrosodiethylamine	ND–25 ng	Sufficient		2A	*17*, 1978; *S7*, 1987
N-nitrosopyrrolidine	1.5–110 ng	Sufficient		2B	*17*, 1978; *S7*, 1987
N-nitrosopiperidine	ND–9 ng	Sufficient		2B	*17*, 1978; *S7*, 1987
N-nitrosodiethanolamine	ND–36 ng	Sufficient		2B	*17*, 1978; *77*, 2000
N'-nitrosonornicotine	154–196 ng	Sufficient	Limited	1	*37*, 1985; *S7*, 1987; *89*, in press
4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone	110–133 ng	Sufficient	Limited	1	*37*, 1985; *S7*, 1987; *89*, in press
Aromatic amines					
2-toluidine	30–200 ng	Sufficient	Limited	2A	*S7*, 1987; *77*, 2000
2,6-dimethylaniline	4–50 ng	Sufficient		2B	*57*, 1993
2-naphthylamine	1–22 ng	Sufficient	Sufficient	1	*4*, 1974; *S7*, 1987
4-aminobiphenyl	2–5 ng	Sufficient	Sufficient	1	*1*, 1972; *S7*, 1987
Heterocyclic aromatic amines					
2-amino-9*H*-pyrido[2,3-*b*]indole	25–260 ng	Sufficient		2B	*40*, 1986; *S7*, 1987
2-amino-3-methyl-9*H*-pyrido[2,3-*b*]indole	2–37 ng	Sufficient		2B	*40*, 1986; *S7*, 1987
2-amino-3-methylimidazo[4,5-*f*]quinoline	0.3 ng	Sufficient		2A	*S7*, 1987; *56*, 1993
3-amino-1,4-dimethyl-5*H*-pyrido[4,3-*b*]indole	0.3–0.5 ng	Sufficient		2B	*31*, 1983; *S7*, 1987
3-amino-1-methyl-5*H*-pyrido[4,3-*b*]indole	0.8–1.1 ng	Sufficient		2B	*31*, 1983; *S7*, 1987
2-amino-6-methylpyrido[1,2-*a*:3′,2′-*d*]imidazole	0.37–0.89 ng	Sufficient		2B	*40*, 1986; *S7*, 1987
2-aminodipyrido[1,2-*a*:3′,2′-*d*]imidazole	0.25–0.88 ng	Sufficient		2B	*40*, 1986; *S7*, 1987
2-amino-1-methyl-6-phenylimidazo[4,5-*b*]pyridine	11–23 ng	Sufficient		2B	*56*, 1993

Table 5.1 Continued

Carcinogen[a]	Quantity (per cigarette)	IARC evaluations of evidence of carcinogenicity in humans: In animals	In humans	IARC group[b]	*IARC Monograph*[c] (volume, year)
Aldehydes					
Formaldehyde	10.3–25 µg	Sufficient	Sufficient	1	*S7*, 1987; *62*, 1995b
Acetaldehyde	770–864 µg	Sufficient		2B	*S7*, 1987; *71*, 1999
Phenolic compounds					
Catechol	59–81 µg	Sufficient		2B	*S7*, 1987; *71*, 1999
Caffeic acid	<3 µg	Sufficient		2B	*56*, 1993
Volatile hydrocarbons					
1,3-butadiene	20–40 µg	Sufficient	Limited	2A	*S7*, 1987; *71*, 1999
Isoprene	450–1,000 µg	Sufficient		2B	*60*, 1994; *71*, 1999
Benzene	12–50 µg	Sufficient	Sufficient	1	*29*, 1982; *S7*, 1987
Nitrohydrocarbons					
Nitromethane	0.5–0.6 µg	Sufficient		2B	*77*, 2000
2-nitropropane	0.7–1.2 ng	Sufficient		2B	*S7*, 1987; *71*, 1999
Nitrobenzene	25 µg	Sufficient		2B	*65*, 1996
Miscellaneous organic compounds					
Acetamide	38–56 µg	Sufficient		2B	*S7*, 1987; *71*, 1999
Acrylamide	Present	Sufficient		2A	*S7*, 1987; *60*, 1994
Acrylonitrile	3–15 µg	Sufficient		2B	*S7*, 1987; *71*, 1999
Vinyl chloride	11–15 ng	Sufficient	Sufficient	1	*19*, 1979; *S7*, 1987
1,1-dimethylhydrazine	Present	Sufficient		2B	*4*, 1974; *71*, 1999
Ethylene oxide	7 µg	Sufficient	Limited	1	*60*, 1994; *S7*, 1987
Propylene oxide	0–100 ng	Sufficient		2B	*60*, 1994; *S7*, 1987
Urethane	20–38 ng	Sufficient		2B	*7*, 1974; *S7*, 1987
Metals and inorganic compounds					
Arsenic	40–120 ng	Sufficient	Sufficient	1	*84*, 2004
Beryllium	0.5 ng	Sufficient	Sufficient	1	*S7*, 1987; *58*, 1993
Nickel	ND–600 ng	Sufficient	Sufficient	1	*S7*, 1987; *49*, 1990
Chromium (hexavalent)	4–70 ng	Sufficient	Sufficient	1	*S7*, 1987; *49*, 1990
Cadmium	41–62 ng	Sufficient	Sufficient	1	*S7*, 1987; *58*, 1993
Cobalt	0.13–0.20 ng	Sufficient		2B	*52*, 1991
Lead (inorganic)	34–85 ng	Sufficient	Limited	2A	*23*, 1980; *S7*, 1987; *87*, in press
Hydrazine	24–43 ng	Sufficient		2B	*S7*, 1987; *71*, 1999
Radioisotope polonium-210	0.03–1.0 picocurie	Sufficient		1	*78*, 2001

Source: Adapted from Hoffmann et al. 2001 and International Agency for Research on Cancer 2004 with permission from American Chemical Society, © 2001 and International Agency for Research on Cancer, © 2004.

Note: **IARC** = International Agency for Research on Cancer; **ND** = not detected; **ng** = nanograms; ***S7*** = Supplement 7; **µg** = micrograms.

[a]Virtually all these compounds are known carcinogens in experimental animals, and IARC found sufficient evidence for carcinogenicity in animals for all the compounds.

[b]Using data on cancer in humans and, in some cases, other data, IARC established classifications for compounds as group 1 (carcinogenic to humans), group 2A (probably carcinogenic to humans), and group 2B (possibly carcinogenic to humans).

[c]If more than two IARC evaluations were performed, only the two most recent monographs are listed.

Percentage contribution of risk factors to all–age DALYs of trachea/bronchus/lung cancer in 2021 (Global Burden of Disease)

Location	Risk factors															
	Ambient particulate matter pollution, % (95% UI)	Diet low in fruits, % (95% UI)	High fasting plasma glucose, % (95% UI)	Household air pollution from solid fuels, % (95% UI)	Occupational exposure to arsenic, % (95% UI)	Occupational exposure to asbestos, % (95% UI)	Occupational exposure to beryllium, % (95% UI)	Occupational exposure to cadmium, % (95% UI)	Occupational exposure to chromium, % (95% UI)	Occupational exposure to diesel engine exhaust, % (95% UI)	Occupational exposure to nickel, % (95% UI)	Occupational exposure to polycyclic aromatic hydrocarbons, % (95% UI)	Occupational exposure to silica, % (95% UI)	Residential radon, % (95% UI)	Secondhand smoke, % (95% UI)	Smoking, % (95% UI)
Global	15.0 (9.4–21.0)	3.5 (1.8–4.9)	2.3 (−0.5 to 5.2)	4.2 (1.6–9.7)	0.6 (0.1–1.1)	7.2 (5.0–9.4)	0.0 (0.0–0.0)	0.1 (0.0–0.1)	0.1 (0.1–0.1)	1.4 (1.2–1.5)	0.6 (0.1–1.5)	0.4 (0.3–0.4)	3.3 (1.5–5.1)	4.1 (−1.9 to 10.4)	5.1 (0.6–9.5)	59.5 (55.4–63.3)
Global Female	14.1 (8.5–20.0)	3.4 (1.8–4.9)	2.3 (−0.4 to 5)	4.7 (1.8–10.9)	0.7 (0.2–1.2)	2.9 (1.8–4)	0.0 (0.0–0.0)	0.1 (0.0–0.1)	0.1 (0.1–0.1)	1.2 (1.0–1.5)	0.6 (0.1–1.4)	0.4 (0.3–0.5)	2.6 (1.2–4.0)	4.1 (−1.9 to 10.5)	6.6 (0.8–12.4)	31.7 (28.1–35.0)
Global Male	15.4 (9.9–21.5)	3.5 (1.8–5.0)	2.4 (−0.5 to 5.2)	4.0 (1.6–9.5)	0.6 (0.1–1.1)	9.3 (6.2–12.8)	0.0 (0.0–0.0)	0.0 (0.0–0.1)	0.1 (0.1–0.1)	1.4 (1.2–1.7)	0.6 (0.1–1.5)	0.4 (0.3–0.4)	3.7 (1.6–5.6)	4.1 (−1.9 to 10.4)	4.3 (0.5–8.1)	72.9 (69.2–76.3)
5 SDI quintiles regions																
High SDI	8.2 (4.8–11.8)	2.7 (1.4–3.9)	2.9 (−0.6 to	0.0 (0.0–0.4)	0.5 (−0.1 to 1.1)	16.2 (11.6–20.4)	0.0 (0.0–0.0)	0.0 (0.0–0.1)	0.0 (0.0–0.2)	0.5 (0.4–0.6)	0.4 (−0.1 to 1.5)	0.1 (0.1–0.2)	2.7 (0.7–4.4)	4.2 (−2.0 to 11.0)	3.2 (0.4–6.0)	61.3 (56.8–65.4)

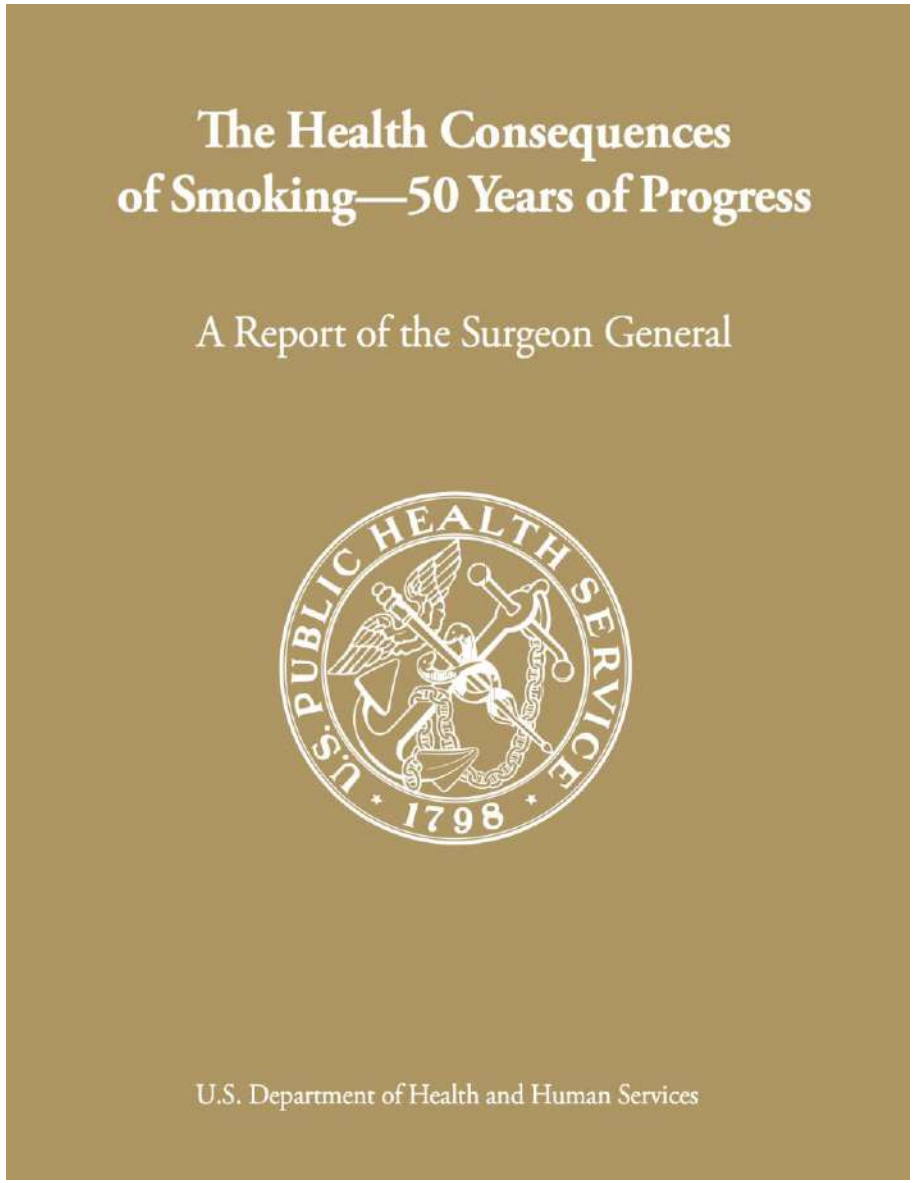
Kuang et al. eClinicalMedicine 2024;75: 102804

Risk factors for primary lung cancer among never-smoking women in South Korea

- Retrospective cohort study from a general health examination and questionnaire through the NHIS
- Factors associated with development of lung cancer in never-smoking women:
  - older age,
  - lower body mass index (BMI)
  - less exercise
  - frequent alcohol drinking
  - meat-based diet
  - rural residence
  - previous history of cancer

Ko et. al. Korean J Intern Med 2020;35:692-702

The 1964 Surgeon General's Report, revisited



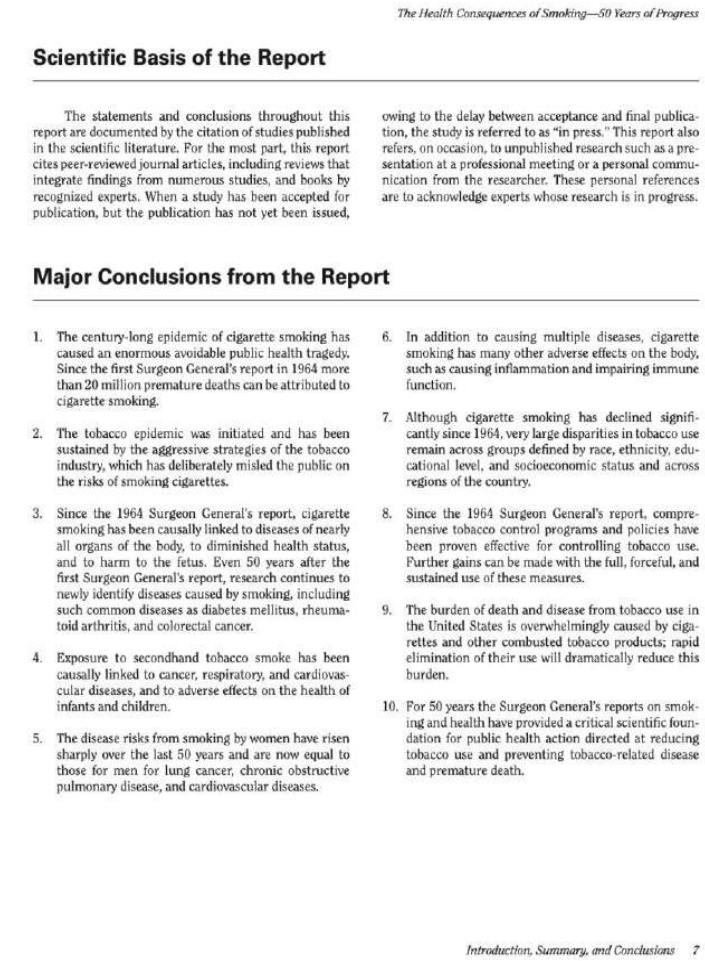
Scientific Basis of the Report

The statements and conclusions throughout this report are documented by the citation of studies published in the scientific literature. For the most part, this report cites peer-reviewed journal articles, including reviews that integrate findings from numerous studies, and books by recognized experts. When a study has been accepted for publication, but the publication has not yet been issued, owing to the delay between acceptance and final publication, the study is referred to as "in press." This report also refers, on occasion, to unpublished research such as a presentation at a professional meeting or a personal communication from the researcher. These personal references are to acknowledge experts whose research is in progress.

Major Conclusions from the Report

1. The century-long epidemic of cigarette smoking has caused an enormous avoidable public health tragedy. Since the first Surgeon General's report in 1964 more than 20 million premature deaths can be attributed to cigarette smoking.
2. The tobacco epidemic was initiated and has been sustained by the aggressive strategies of the tobacco industry, which has deliberately misled the public on the risks of smoking cigarettes.
3. Since the 1964 Surgeon General's report, cigarette smoking has been causally linked to diseases of nearly all organs of the body, to diminished health status, and to harm to the fetus. Even 50 years after the first Surgeon General's report, research continues to newly identify diseases caused by smoking, including such common diseases as diabetes mellitus, rheumatoid arthritis, and colorectal cancer.
4. Exposure to secondhand tobacco smoke has been causally linked to cancer, respiratory, and cardiovascular diseases, and to adverse effects on the health of infants and children.
5. The disease risks from smoking by women have risen sharply over the last 50 years and are now equal to those for men for lung cancer, chronic obstructive pulmonary disease, and cardiovascular diseases.
6. In addition to causing multiple diseases, cigarette smoking has many other adverse effects on the body, such as causing inflammation and impairing immune function.
7. Although cigarette smoking has declined significantly since 1964, very large disparities in tobacco use remain across groups defined by race, ethnicity, educational level, and socioeconomic status and across regions of the country.
8. Since the 1964 Surgeon General's report, comprehensive tobacco control programs and policies have been proven effective for controlling tobacco use. Further gains can be made with the full, forceful, and sustained use of these measures.
9. The burden of death and disease from tobacco use in the United States is overwhelmingly caused by cigarettes and other combusted tobacco products; rapid elimination of their use will dramatically reduce this burden.
10. For 50 years the Surgeon General's reports on smoking and health have provided a critical scientific foundation for public health action directed at reducing tobacco use and preventing tobacco-related disease and premature death.

The health consequences of smoking



The Health Consequences of Smoking—50 Years of Progress

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Introduction, Summary, and Conclusions 7

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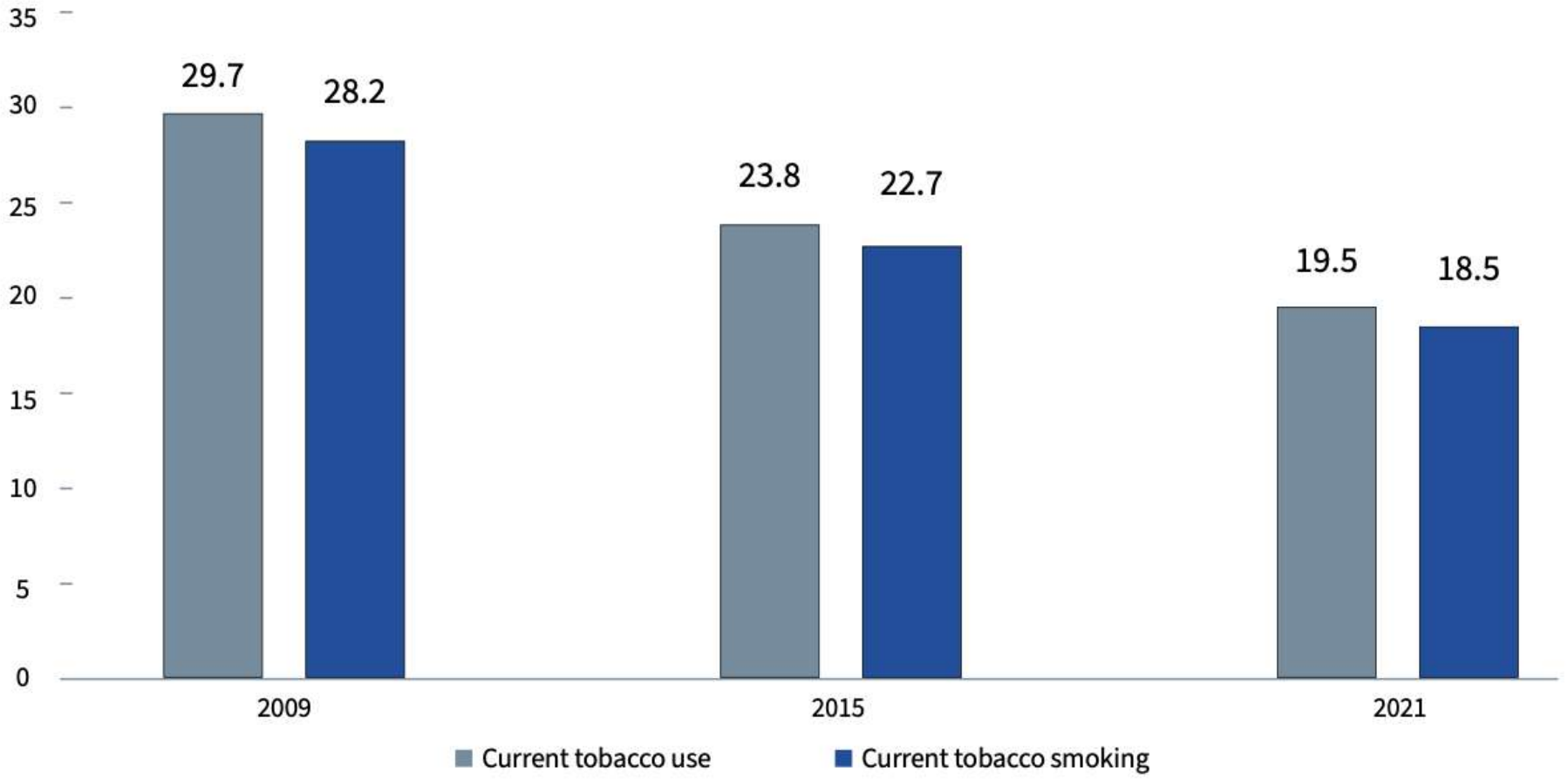
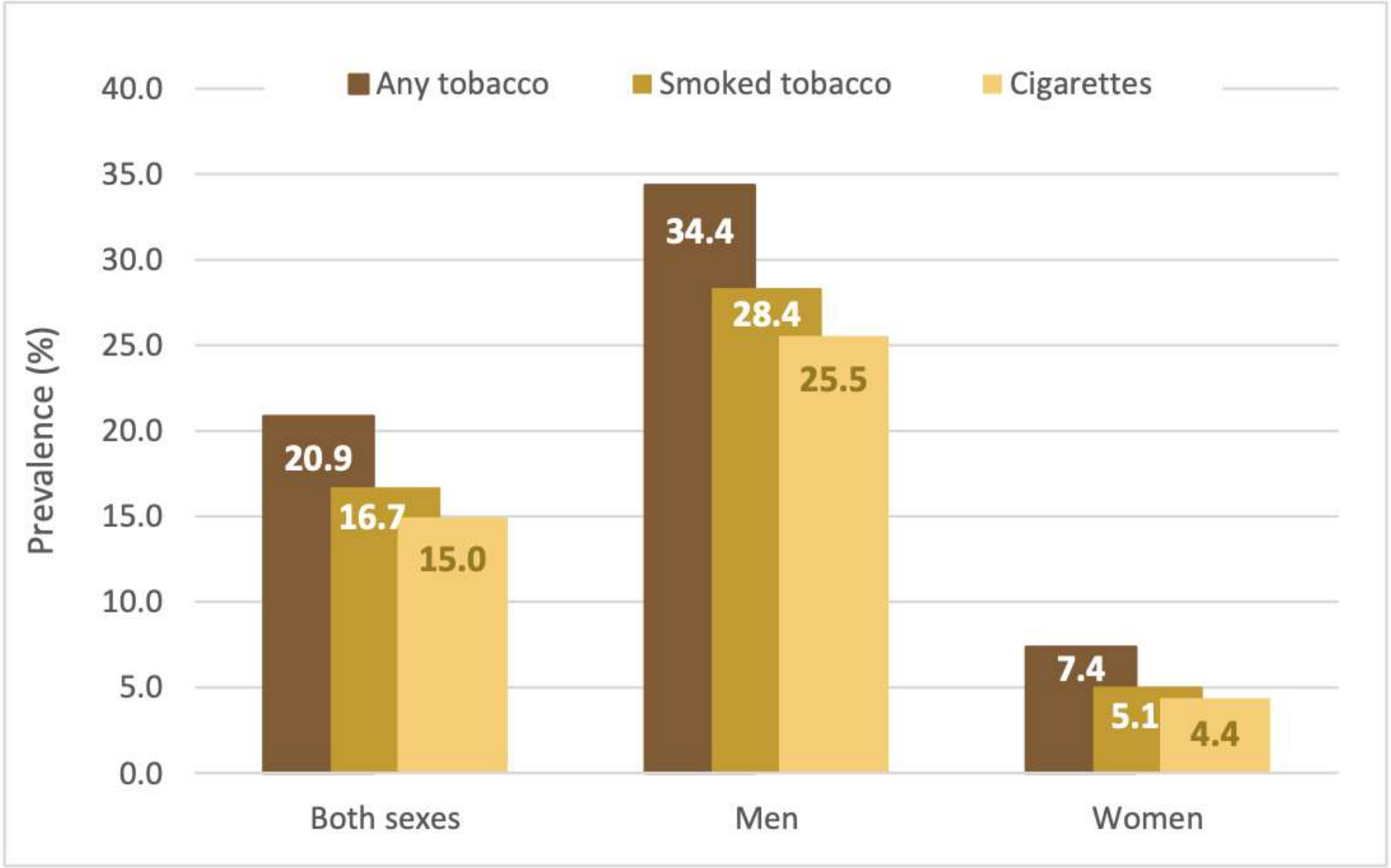
Global smoking prevalence



WHO Global Tobacco Report 2023

Smoking prevalence by gender, 2022



WHO global report on trends in prevalence of tobacco use 2000–2030

Burden of tobacco use in the Republic of Korea

Adult Smoking Prevalence in South Korea

15+ years old; 2022

Men	Women
32.7%	5.4%

Adult smoking prevalence in South Korea is 19%.

Youth Smoking Prevalence in South Korea

10-14 years old; 2022

Men	Women
8.7%	5.0%

Youth smoking prevalence in South Korea is 6.9%.

Deaths Caused by Tobacco in South Korea

% deaths attributable to tobacco use in 2021

Men	Women
19.9%	5.8%

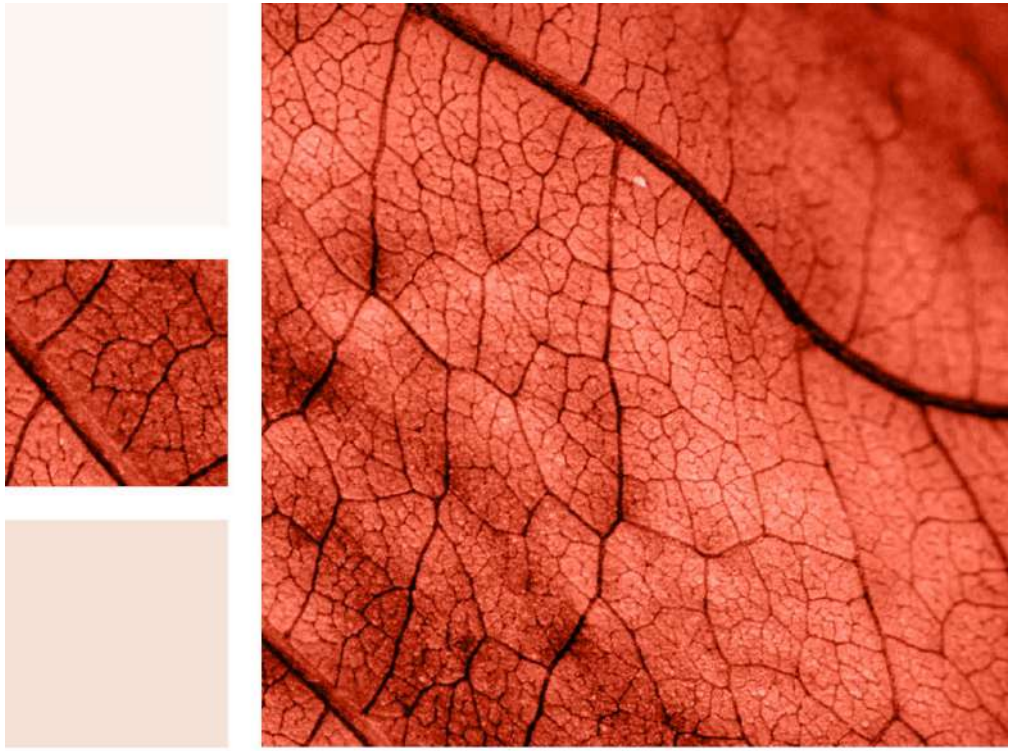
13.4% of all deaths in South Korea are caused by tobacco use.

Source: Tobacco Atlas (https://tobaccoatlas.org/factsheets/republic-of-korea/)

WHO FRAMEWORK CONVENTION ON TOBACCO CONTROL

FCTC

WHO FRAMEWORK CONVENTION
ON TOBACCO CONTROL

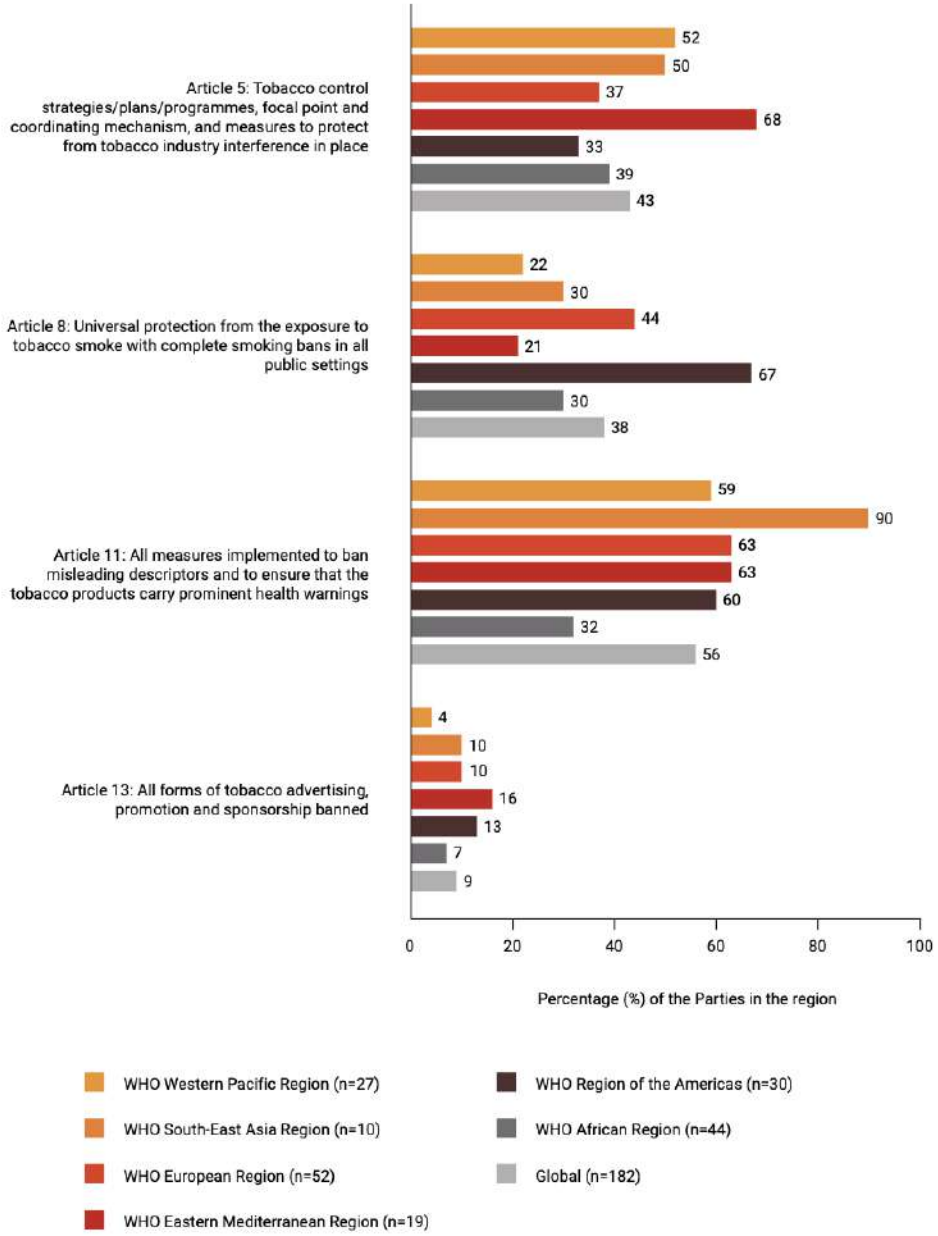


2023

2023 Global Progress Report on Implementation of the WHO Framework Convention on Tobacco Control

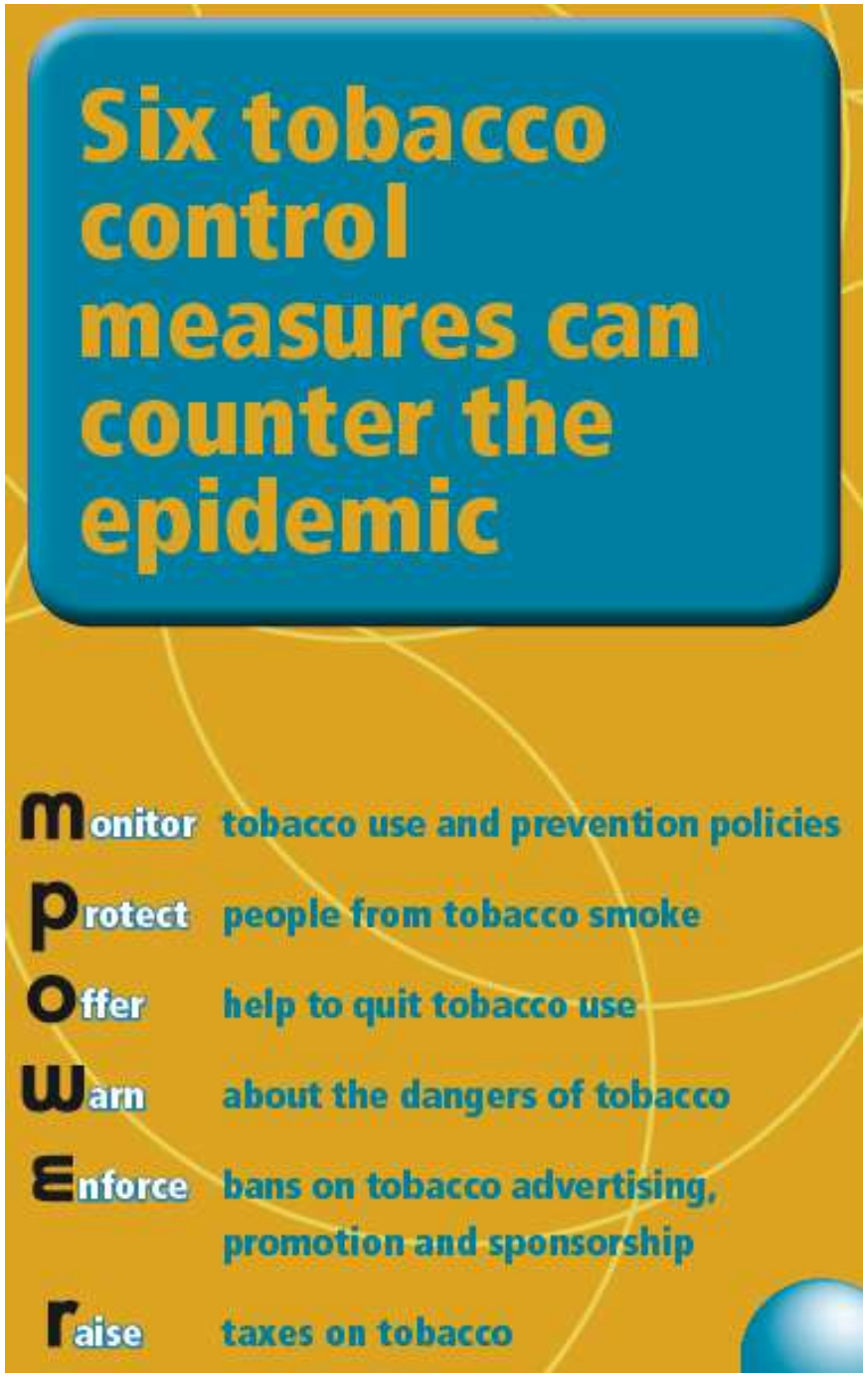
FCTC
WHO FRAMEWORK CONVENTION ON TOBACCO CONTROL

Fig. 1. Percentage of Parties that have reported implementing all the key measures under articles 5, 8, 11 and 13 in 2023, globally and by WHO region. The list of the included indicators is provided in the respective article chapters.

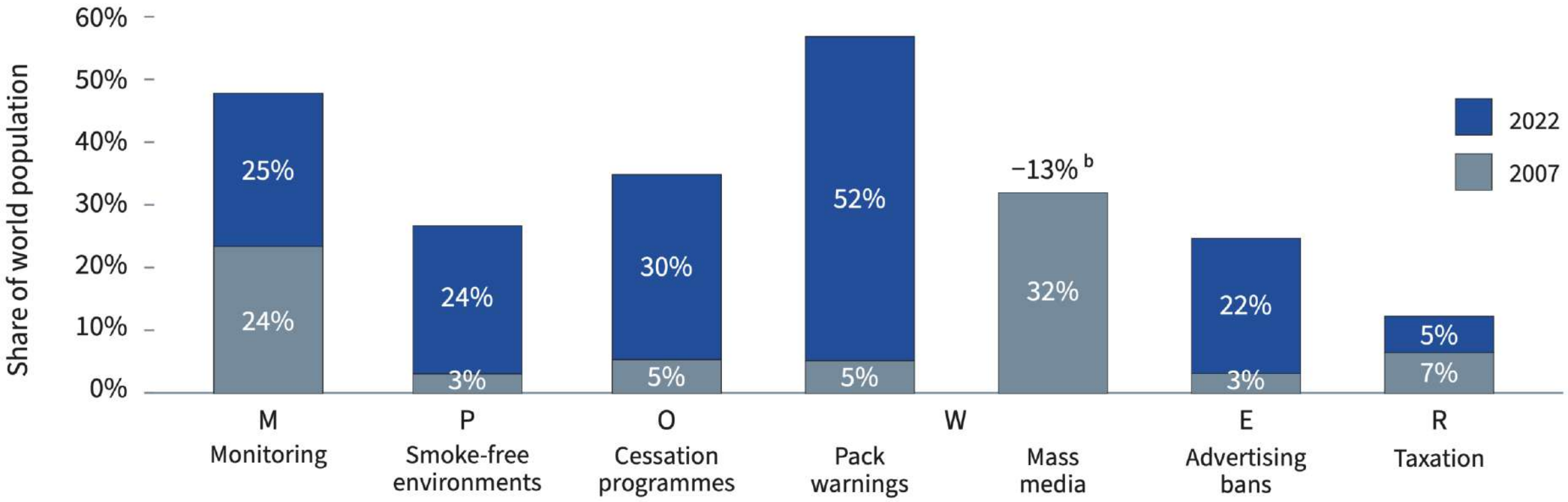


mpower

World Health Organization



Percentage of the world's population covered by MPOWER strategic components



[a] 2010 for W mass media, 2008 for R taxation. [b] The share of the world's population covered by mass media campaigns decreased since 2010.

WHO Global Tobacco Report 2023

Using the legal system to fight Big Tobacco : The US experience

- 1998 Master Settlement Agreement
- 2006 Racketeer Influenced and Corrupt Organization (RICO) prosecution

The Master Settlement Agreement (MSA)

- In 1998, 46 U.S. state attorneys general reached an agreement with 4 major tobacco manufacturers to recover costs associated with treatment of tobacco-related illness
- Agreement was reached with Phillip Morris, R.J. Reynolds, Brown& Williamson, and Lorillard
- 40 additional companies later signed on

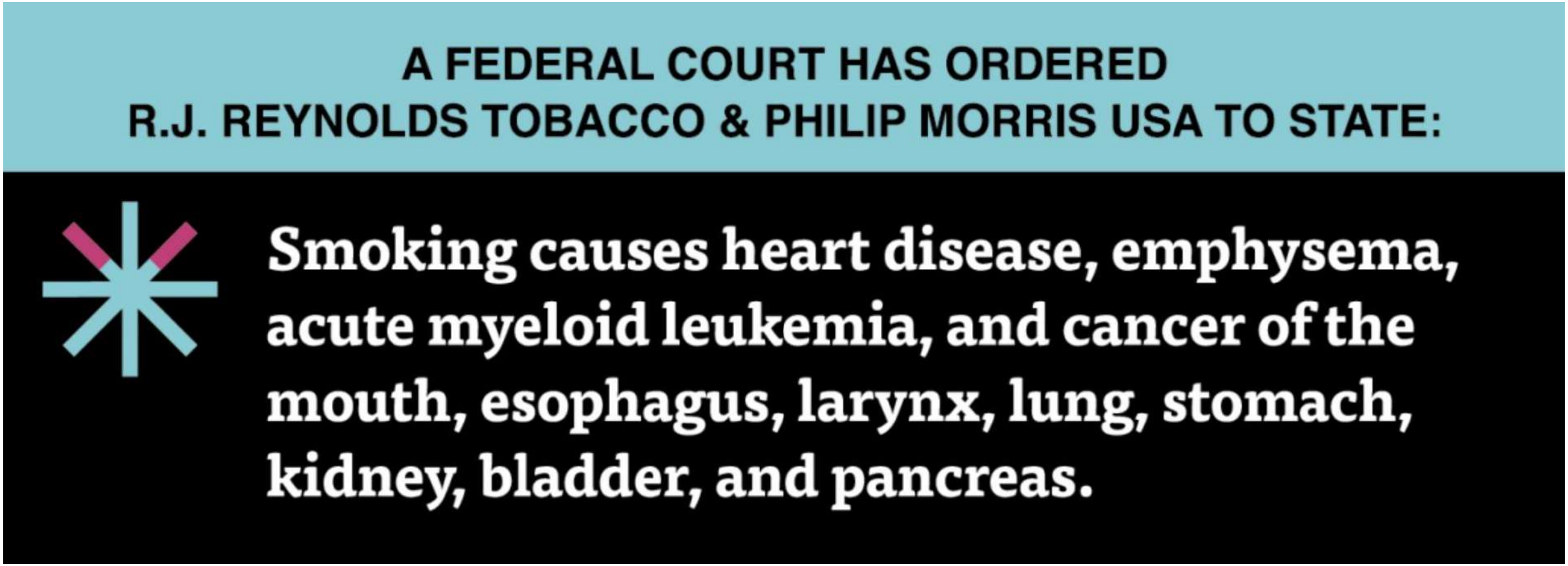
- Terms of the agreement:
  - Companies agreed to pay $206 billion USD ($400 billion in today's terms)
- New limits were created for the advertising, marketing and promotion of cigarettes.
- Tobacco advertising that targets people younger than age 18 was prohibited.
- Cartoons in cigarette advertising were eliminated.
- Outdoor, billboard and public transit advertising of cigarettes was eliminated.
- Cigarette brand names could no longer be used on merchandise.
- Many millions of tobacco company internal documents were made available to the public.

U.S. Federal RICO prosecution

- In 1999, the U..S Department of Justice sued the major cigarette manufacturers, charging RICO violations
- In 2006, U.S. Federal Judge Gladys Kessler found that U.S. tobacco companies had violated the Racketeer Influenced and Corrupt Organization (RICO) law
- "The evidence in this case clearly establishes that Defendants have not ceased engaging in unlawful activity…. Their continuing misconduct misleads consumers in order to maximize Defendants' revenues by recruiting new smokers (the majority of whom are under the age of 18), preventing current smokers from quitting, and thereby sustaining the industry."

- Remedies:
  - Prohibit the tobacco companies from committing acts of racketeering in the future or making false, misleading or deceptive statements concerning cigarettes and their health risks.
  - Ban terms including "low tar," "light," "ultra light," "mild" and "natural" that that have been used to mislead consumers about the health risks of smoking.
  - Extend and expand existing requirements that the tobacco companies make public their internal documents produced in litigation.
  - Require the tobacco companies to report marketing data annually to the government.

Required signs placed in 220,000 stores in the U.S.



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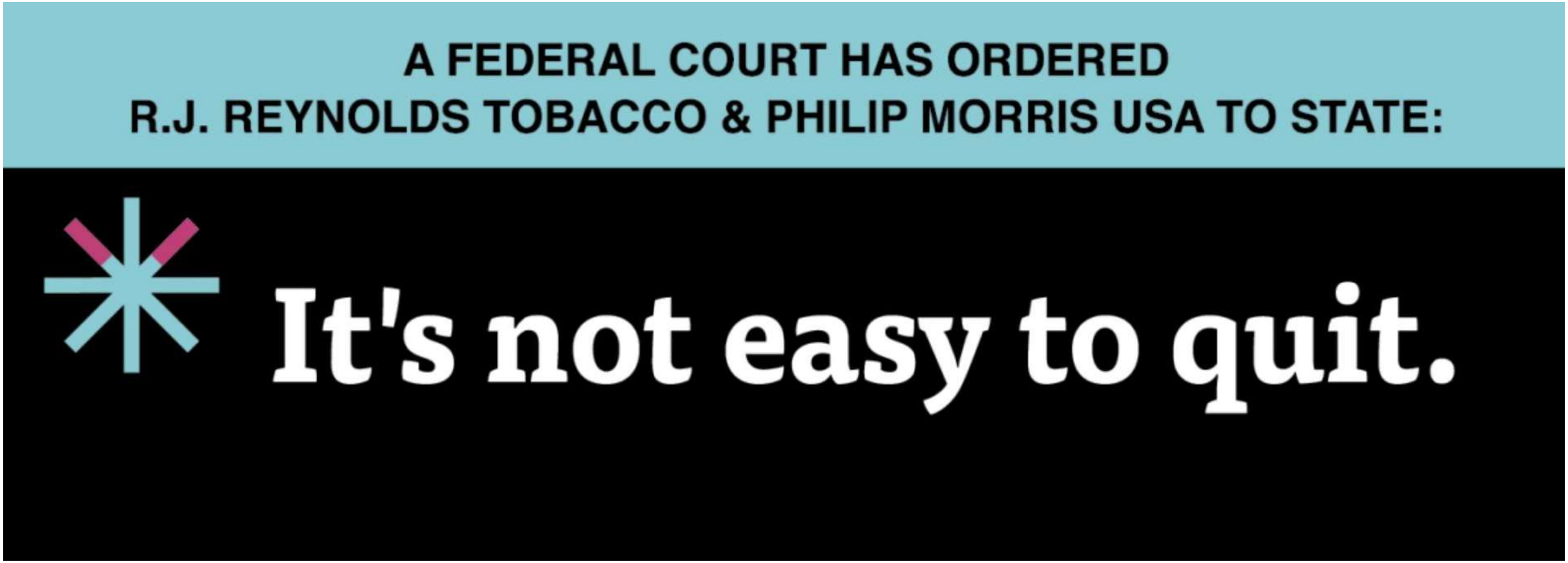

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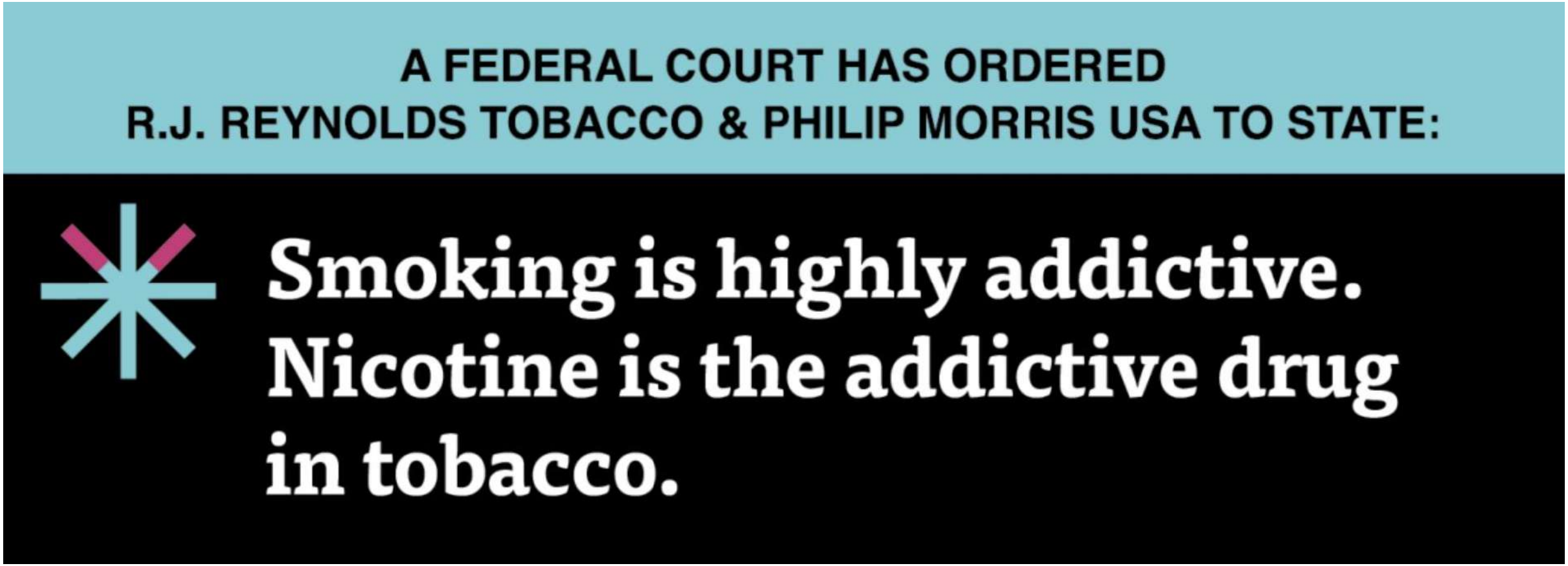


A FEDERAL COURT HAS ORDERED
R.J. REYNOLDS TOBACCO & PHILIP MORRIS USA TO STATE:
"Low tar" and "light" cigarette smokers inhale essentially the same amount of tar and nicotine as they would from regular cigarettes.
NOW AVAILABLE
$13.48 Plus Tax
$13.43 Plus Tax
$12.02 Plus Tax
$11.52 Plus Tax
$12.47
Reese's
KING SIZE
HERSHEY'S
Reese's STICKS

Conclusions

- The link between cigarette smoking and adverse outcomes, particularly cancers of the larynx, trachea, bronchus, and lung is strong and unequivocal
- Lifelong smokers give up over 10 years of life
- The Framework Convention on Tobacco Control was the first public health treaty ever negotiated
- In addition to public education, smoking bans, taxations, limitations on advertising and sponsorship, aggressive legal measures by governments may be helpful in combatting the tobacco industry