解説　喫煙・受動喫煙の有害性

産業医科大学　産業生態科学研究所
健康開発科学研究室 教授 大和　浩

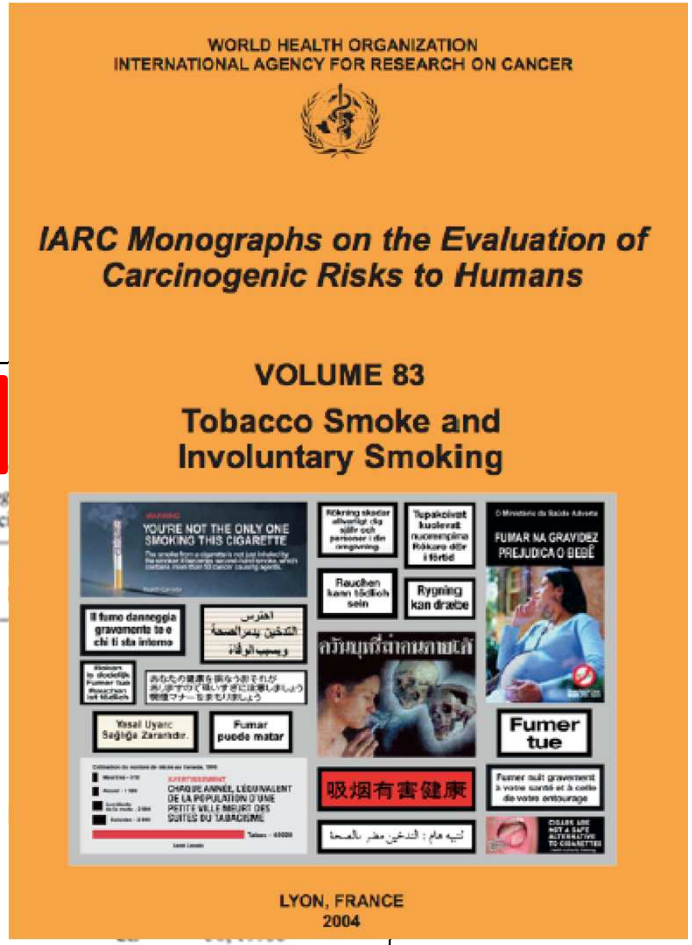
- 危険因子（喫煙・受動喫煙）の曝露で病気が増加
- 危険因子（喫煙・受動喫煙）の除去で病気が減少

かつ、それぞれ、量・反応(doze-response)関係が
明確に証明されているのは「タバコ煙」だけであることを
喫煙関連疾患（肺がん、心血管系疾患、糖尿病）で解説

国際がん研究機関(IARC)による発がん性分類
(IARC: International Agency for Research on Cancer)

分類	ヒトに対する発がん性	該当物質、環境、因子
Group 1	発がん性がある (Carcinogenic to humans)	アスベスト、ベンゼン、ベリリウム、カドミウム、ヒ素、アルコール飲料、喫煙、受動喫煙など
Group 2A	発がん性がおそらくある (Probably carcinogenic to humans)	アクリルアミド、アドリアマイシン、シスプラチン、紫外線、ディーゼル排ガス、コバルト、 交代制勤務など
Group 2B	発がん性があるかもしれない (Possibly carcinogenic to humans)	アセトアルデヒド、ブレオマイシン、 カーボンブラック、クロロホルム、コバルト、鉛、耐火セラミック繊維など
Group 3	発がん性を分類できない (Not classifiable as to its carcinogenicity to humans)	アクリル繊維、揮発性の麻酔薬、パラアミド繊維、塩素消毒した飲料水、石炭粉じん、水銀、 鉄鉱石、塩酸、過酸化水素、臭化メチルなど
Group 4	発がん性はおそらくない (Probably not carcinogenic to humans)	カプロラクタム(ナイロンの原料)

タバコ煙に含まれる
64種類の発がん性物質のリスト
（WHO 国際がん研究機関：IARC），2004年



Carcinogens in cigarette smoke (64 single agents identified)

IARC monograph vol 83,
Tobacco Smoke and Involuntary Smoking, 2004

Agent	Amount in mainstream cigarette smoke	*IARC Monographs* evaluation of carcinogenicity: In animals	In humans	IARC Group	Monograph volume, year
Polynuclear aromatic hydrocarbons					
Benz[*a*]anthracene	20–70 ng	Sufficient		2A	*32*, 1983a; *S7*, 1987
Benzo[*b*]fluoranthene	4–22 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo[*j*]fluoranthene	6–21 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo[*k*]fluoranthene	2–7 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo[*a*]pyrene	8–11.6 ng[a]	Sufficient		2A	*32*, 1983a; *S7*, 1987
Dibenz[*a*,*h*]anthracene	4 ng	Sufficient		2A	*32*, 1983a; *S7*, 1987
Dibenzo[*a*,*e*]pyrene	1.7–3.2 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Dibenzo[*a*,*h*]pyrene	present	Sufficient		2B	*32*, 1983a; *S7*, 1987
Indeno[1,2,3-*cd*]pyrene	4–20 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
5-Methylchrysene	ND–0.6 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Heterocyclic hydrocarbons					
Furan	20–40 µg[b]	Sufficient		2B	*63*, 1995b
Dibenz[*a*,*j*]acridine	ND–0.1 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Dibenz[*a*,*h*]acridine	ND–10 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Dibenzo[*c*,*g*]carbazole	ND–0.7 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzofuran	present	Sufficient		2B	*63*, 1995b
N-Nitrosamines					
N-Nitrosodiethylamine	ND–80 ng	Sufficient		2A	*17*, 1978; *S7*, 1987
N-Nitrosodimethylamine	ND–80 ng	Sufficient		2B	*17*, 1978; *S7*, 1987
N-Nitrosodiethylamine	ND–25 ng	Sufficient		2A	*17*, 1978; *S7*, 1987
N-Nitrosodimethylamine	1.1–110 ng[c]	Sufficient		2B	*17*, 1978; *S7*, 1987
N-Nitrosodiethanolamine	ND–36 ng[c]	Sufficient		2B	*17*, 1978; *77*, 2000
N′-Nitrosonornicotine	154–196 ng[c]	Sufficient		2B[d]	*37*, 1985b; *S7*, 1987

Agent	Amount in mainstream cigarette smoke	*IARC Monographs* evaluation of carcinogenicity: In animals	In humans	IARC Group	Monograph volume, year
Trp-P-1	0.3–0.5 ng	Sufficient			
Trp-P-2	0.8–1.1 ng	Sufficient			
Glu-P-1	0.37–0.89 ng	Sufficient			
Glu-P-2	0.25–0.88 ng	Sufficient			
PhIP	11–23 ng	Sufficient			
Aldehydes					
Formaldehyde	10.3–25 µg[a]	Sufficient			
Acetaldehyde	770–864 µg[a]	Sufficient			
Phenolic compounds					
Catechol	59–81 µg[a]	Sufficient			
Caffeic acid	< 3 µg	Sufficient			
Volatile hydrocarbons					
1,3-Butadiene	20–40 µg[b]	Sufficient	Limited	2A	*S7*, 1987; *71*, 1999
Isoprene	450–1000 µg	Sufficient		2B	*60*, 1994; *71*, 1999
Benzene	12–50 µg[b]	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[c]	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
Hydrazine	24–43 ng	Sufficient		2B	*S7*, 1987; *71*, 1999

アンモニア
カドミウム
ニトロサミン
ヒ素
ベンゾピレン
ホルムアルデヒド
ポロニウム-210　など

Carcinogens in cigarette smoke (64 single agents identified)

IARC monograph vol 83,
Tobacco Smoke and Involuntary Smoking, 2004

Agent	Amount in mainstream cigarette smoke	*IARC Monographs* evaluation of carcinogenicity			Monograph volume, year
		In animals	In humans	IARC Group	
Polynuclear aromatic hydrocarbons 多環芳香族炭化水素類					
Benz[*a*]anthracene	20–70 ng	Sufficient		2A	*32*, 1983a; *S7*, 1987
Benzo[*b*]fluoranthene	4–22 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo[*j*]fluoranthene	6–21 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo[*k*]fluoranthene	6–12 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo[*a*]pyrene	8.5–11.6 ng[a]	Sufficient		2A	*32*, 1983a; *S7*, 1987
Dibenz[*a,h*]anthracene	4 ng	Sufficient		2A	*32*, 1983a; *S7*, 1987
Dibenzo[*a,i*]pyrene	1.7–3.2 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Dibenzo[*a,e*]pyrene	Present	Sufficient		2B	*32*, 1983a; *S7*, 1987
Indeno[*1,2,3-cd*]pyrene	4–20 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
5-Methylchrysene	ND–0.6 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Heterocyclic hydrocarbons					
Furan	20–40 µg[b]	Sufficient		2B	*63*, 1995b
Dibenz(*a,h*)acridine	ND– 0.1 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Dibenz(*a,f*)acridine	ND–10 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Dibenzo(*c,g*)carbazole	ND– 0.7 ng	Sufficient		2B	*32*, 1983a; *S7*, 1987
Benzo(*b*)furan	present	Sufficient		2B	*63*, 1995b
***N*-Nitrosamines** ニトロサミン類					
N-Nitrosodimethylamine	0.1–180 ng[b]	Sufficient		2A	*17*, 1978; *S7*, 1987
N-Nitrosoethylmethylamine	ND–13 ng	Sufficient		2B	*17*, 1978; *S7*, 1987
N-Nitrosodiethylamine	ND–25 ng[b]	Sufficient		2A	*17*, 1978; *S7*, 1987
N-Nitrosopyrrolidine	1.5–110 ng[b]	Sufficient		2B	*17*, 1978; *S7*, 1987
N-Nitrosopiperidine	ND–9 ng	Sufficient		2B	*17*, 1978; *S7*, 1987
N-Nitrosodiethanolamine	ND–36 ng[b]	Sufficient		2B	*17*, 1978; *77*, 2000
N'-Nitrosonornicotine	154–196 ng[a]	Sufficient		2B[c]	*37*, 1985b; *S7*, 1987
4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone	110–133 ng[a]	Sufficient		2B[c]	*37*, 1985b; *S7*, 1987
Aromatic amines 芳香族アミン類					
2-Toluidine	30–200 ng[b]	Sufficient	Limited	2A	*S7*, 1987; *77*, 2000
2,6-Dimethylaniline	4–50 ng	Sufficient		2B	*57*, 1993
2-Naphthylamine	1–22 ng[b]	Sufficient	Sufficient	1	*4*, 1974; *S7*, 1987
4-Aminobiphenyl	2–5 ng[b]	Sufficient	Sufficient	1	*1*, 1972; *S7*, 1987
***N*-Heterocyclic amines**					
A-α-C	25–260 ng	Sufficient		2B	*40*, 1986b; *S7*, 1987
MeA-α-C	2–37 ng	Sufficient		2B	*40*, 1986b; *S7*, 1987
IQ	0.3 ng	Sufficient		2A	*S7*, 1987; *56*, 1993
Trp-P-1	0.3–0.5 ng	Sufficient		2B	*31*, 1983b; *S7*, 1987
Trp-P-2	0.8–1.1 ng	Sufficient		2B	*31*, 1983b; *S7*, 1987
Glu-P-1	0.37–0.89 ng	Sufficient		2B	*40*, 1986b; *S7*, 1987
Glu-P-2	0.25–0.88 ng	Sufficient		2B	*40*, 1986b; *S7*, 1987
PhIP	11–23 ng	Sufficient		2B	*56*, 1993b
Aldehydes					
Formaldehyde ホルムアルデヒド	0.3–25 µg[a]	Sufficient	Limited	2A	*S7*, 1987; *62*, 1995a
Acetaldehyde アセトアルデヒド	770–864 µg[a]	Sufficient		2B	*S7*, 1987; *71*, 1999
Phenolic compounds					
Catechol	59–81 µg[a]	Sufficient		2B	*S7*, 1987; *71*, 1999
Caffeic acid	< 3 µg	Sufficient		2B	*56*, 1993b
Volatile hydrocarbons					
1,3-Butadiene	20–40 µg[b]	Sufficient	Limited	2A	*S7*, 1987; *71*, 1999
Isoprene	450–1000 µg	Sufficient		2B	*60*, 1994; *71*, 1999
Benzene	12–50 µg[b]	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[a]	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
Hydrazine	24–43 ng	Sufficient		2B	*S7*, 1987; *71*, 1999
Urethane	20–38 ng[b]	Sufficient		2B	*7*, 1974; *S7*, 1987
Metals and metal compounds					
Arsenic ヒ素	40–120 ng[b]	Sufficient	Sufficient	1	*84*, 2004a
Beryllium ベリリウム	0.5 ng	Sufficient	Sufficient	1	*S7*, 1987; *58*, 1993a
Nickel ニッケル	ND–600 ng	Sufficient	Sufficient	1	*S7*, 1987; *49*, 1990
Chromium (hexavalent) クロム	4–70 ng	Sufficient	Sufficient	1	*S7*, 1987; *49*, 1990
Cadmium カドミウム	41–62 ng[b]	Sufficient	Sufficient	1	*S7*, 1987; *58*, 1993a
Cobalt コバルト	0.13–0.20 ng	Sufficient		2B	*52*, 1991
Lead (inorganic) 鉛	34–85 ng	Sufficient	Limited	2A	*23*, 1980; *S7*, 1987; *87*, 2004b
Radio-isotope					
Polonium-210	0.03–1.0 pCi	Sufficient		1	*78*, 2001

放射性物質：ポロニウム-210

米国公衆衛生総監、1986年報告「受動喫煙による健康影響」

THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING

a report of the Surgeon General

1986

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
Center for Health Promotion and Education
Office on Smoking and Health
Rockville, Maryland 20857

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, DC 20402

FOREWORD

The data reviewed in 17 previous U.S. Public Health Service reports on the health consequences of smoking have conclusively established cigarette smoking as the largest single preventable cause of premature death and disability in the United States.

The question whether tobacco smoke is harmful to smokers was answered more than 20 years ago. As a result, many scientists began to question whether the low levels of exposure to environmental tobacco smoke (ETS) received by nonsmokers could also be harmful.

The current Report, The Health Consequences of Involuntary Smoking, examines the evidence that even the lower exposure to smoke received by the nonsmoker carries with it a health risk. Use of the term "involuntary smoking" denotes that for many nonsmokers, exposure to ETS is the result of an unavoidable consequence of being in proximity to smokers. It is the first Report in the health consequences of smoking series to establish a health risk due to tobacco smoke exposure for individuals other than the smoker, and represents the work of more than 60 distinguished physicians and scientists, both in this country and abroad.

After careful examination of the available evidence, the following overall conclusions can be reached:

1. Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.
2. The children of parents who smoke, compared with the children of nonsmoking parents, have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.
3. Simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, exposure of nonsmokers to environmental tobacco smoke.

Exposure to environmental tobacco smoke occurs at home, at the worksite, in public, and in other places where smoking is permitted.

米国公衆衛生総監報告（1986年）の主要な結論

Table 1.2 **Major conclusions of the 1986 Surgeon General's report, *The Health Consequences of Involuntary Smoking***

1.	Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.
2.	The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.
3.	The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke.

Source: U.S. Department of Health and Human Services 1986, p. 7.

1. 受動喫煙は、健康な非喫煙者に肺がんなどの疾病をもたらす
2. 喫煙する両親をもった子どもは、喫煙しない両親をもった子どもよりも、呼吸器の感染症を起こす頻度が多く、呼吸器症状を増加させ、呼吸機能の発達が若干阻害される
3. 同じ空間を喫煙区域と禁煙区域に分けることは、受動喫煙の曝露濃度を多少減少させることができても、受動喫煙をなくすことはできない

The Health Consequences of Involuntary Exposure to Tobacco Smoke

A Report of the Surgeon General

Department of Health and Human Services

National Library of Medicine Cataloging in Publication

The health consequences of involuntary exposure to tobacco smoke : a report of the Surgeon General. – [Atlanta, Ga.] : U.S. Dept. of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, [2006]

Includes bibliographical references.

1. Tobacco Smoke Pollution -- adverse effects. I. United States. Public Health Service. Office of the Surgeon General. II. United States. Office on Smoking and Health.

O2NLM: WA 754 H4325 2006

Centers for Disease Control and Prevention
Coordinating Center for Health Promotion
National Center for Chronic Disease Prevention and Health Promotion
Office on Smoking and Health

This publication is available on the World Wide Web at
http://www.surgeongeneral.gov/library

Suggested Citation

U.S. Department of Health and Human Services. *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2006.

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402. ISBN 0-16-076152-2

Foreword

序文（2006年報告）

This twenty-ninth report of the Surgeon General documents the serious and deadly health effects of involuntary exposure to tobacco smoke. Secondhand smoke is a major cause of disease, including lung cancer and coronary heart disease, in healthy nonsmokers.

In 2005, it was estimated that exposure to secondhand smoke kills more than 3,000 adult nonsmokers from lung cancer, approximately 46,000 from coronary heart disease, and an estimated 430 newborns from sudden infant death syndrome. In addition, secondhand smoke causes other respiratory problems in nonsmokers such as coughing, phlegm, and reduced lung function. According to the CDC's National Health Interview Survey in 2000, more than 80 percent of the respondents aged 18 years or older believe that secondhand smoke is harmful and nonsmokers should be protected in their workplaces.

2005年、受動喫煙に曝露された非喫煙者が、肺がんで3,000人、
心血管疾患で46,000人死亡していること、
新生児430人が乳幼児突然死症候群で死亡している、と評価された。
それ以外に、受動喫煙は非喫煙者の咳、痰、肺機能障害の原因となる。

1. SUMMARY AND CONCLUSIONS

1.1. MAJOR CONCLUSIONS

Based on the weight of the available scientific evidence, the U.S. Environmental Protection Agency (EPA) has concluded that the widespread exposure to environmental tobacco smoke (ETS) in the United States presents a serious and substantial public health impact.

In adults:

- ETS is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers.

In children:

- ETS exposure is causally associated with an increased risk of lower respiratory tract infections (LRIs) such as bronchitis and pneumonia. This report estimates that 150,000 to 300,000 cases annually in infants and young children up to 18 months of age are attributable to ETS.

1. サマリーと結論（2006年）

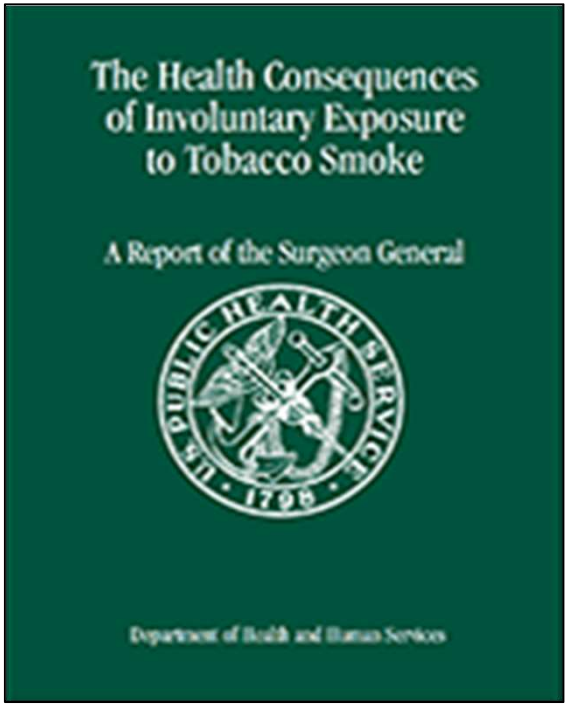
1.1 主たる結論

アメリカ環境保護庁(EPA)は受動喫煙により、アメリカ国民に深刻で重大な健康影響が発生していることを結論する。

成人：受動喫煙は肺に対する発がん性があり、米国で毎年3,000人の非喫煙者が受動喫煙により死亡。

小児：末梢気道疾患（気管支炎、肺炎）のリスクである。受動喫煙により、毎年15～30万人の乳幼児（18ヵ月以下）の末梢気道疾患の原因となっている。

A Report of Surgeon General
（米国公衆衛生総監報告）
“The health consequences on involuntary exposure to tobacco smoke”(2006)
「受動喫煙に起因する健康影響に関する結論」
“The debate is over. The science is clear”
「（有害性に関する）議論は終わった。科学的証拠は明白」でプレスカンファレンスは始まり、以下が述べられた



・受動喫煙は深刻な健康被害をもたらす
・受動喫煙は危険である
・受動喫煙に安全なレベル（閾値）は存在しない
・数百万人の非喫煙者（米国人）が受動喫煙に曝露されている
・すべての人が受動喫煙に曝露されない権利を有する
・無煙の環境を作ることが必要
・小児科医は子ども達を自宅での受動喫煙から守るために、家庭環境の無煙化を進めねばならない（気管支喘息、乳幼児突然死症候群の観点）

Major Conclusions

Surgeon General's Report 2006, p11

2006年報告の6つの主要な結論（後ほど解説）

This report returns to involuntary smoking, the topic of the 1986 Surgeon General's report. Since then, there have been many advances in the research on secondhand smoke, and substantial evidence has been reported over the ensuing 20 years. This report uses the revised language for causal conclusions that was implemented in the 2004 Surgeon General's report (USDHHS 2004). Each chapter provides a comprehensive review of the evidence, a quantitative synthesis of the evidence if appropriate, and a rigorous assessment of sources of bias that may affect interpretations of the findings. The reviews in this report reaffirm and strengthen the findings of the 1986 report. With regard to the involuntary exposure of nonsmokers to tobacco smoke, the scientific evidence now supports the following major conclusions:

1. Secondhand smoke causes premature death and disease in children and in adults who do not smoke.

2. Children exposed to secondhand smoke are at an increased risk for sudden infant death syndrome (SIDS), acute respiratory infections, ear problems, and more severe asthma. Smoking by parents causes respiratory symptoms and slows lung growth in their children.

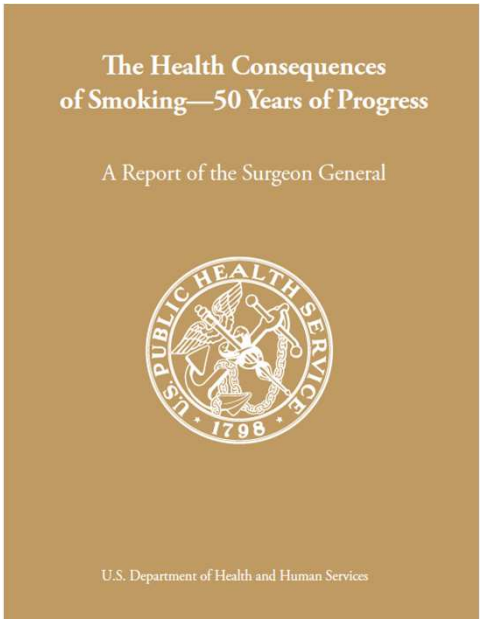
3. Exposure of adults to secondhand smoke has immediate adverse effects on the cardiovascular system and causes coronary heart disease and lung cancer.

4. The scientific evidence indicates that there is no risk-free level of exposure to secondhand smoke.

5. Many millions of Americans, both children and adults, are still exposed to secondhand smoke in their homes and workplaces despite substantial progress in tobacco control.

6. Eliminating smoking in indoor spaces fully protects nonsmokers from exposure to secondhand smoke. Separating smokers from nonsmokers, cleaning the air, and ventilating buildings cannot eliminate exposures of nonsmokers to secondhand smoke.

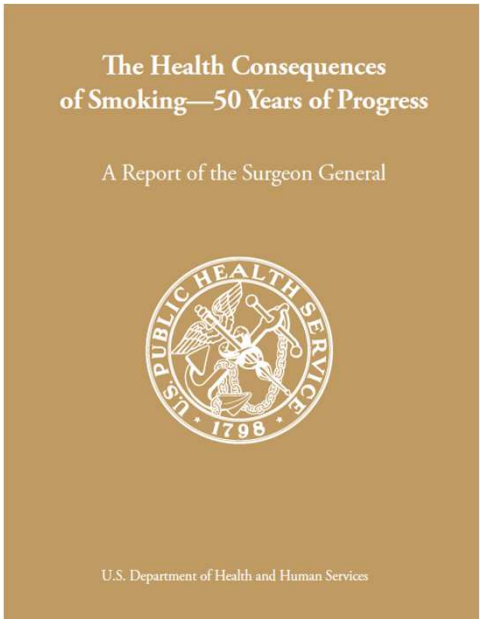
A Report of Surgeon General（米国公衆衛生総監報告）
“The health consequences of smoking
– 50 Years of Progress”(2014年報告)
「喫煙による健康影響：50年間の進歩」



1964年のSurgeon General’s report 後、
成人喫煙率は43%(1965年)→18%(現在)まで低下したが、
いまだに、米国では毎年4000万人が新たな喫煙者＝脅威に変わりない

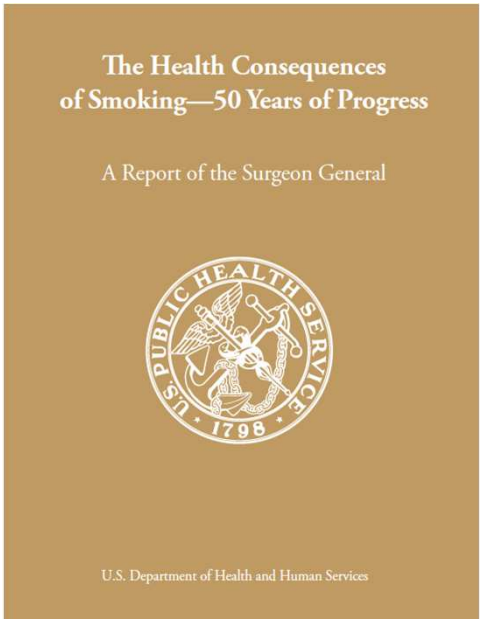
本報告の10の結論は以下の通りである。
1） 1世紀にわたるタバコの流行は、本来、予防できたはずの公衆衛生上の悲劇をもたらした。1964年の米国公衆衛生総監報告以降も2000万人の早世をもたらした。
2） タバコ産業がもたらしたタバコ疫病は、現在も続いている。そのために、タバコ産業は、喫煙の被害を計画的に過小評価させることで国民をミスリードする、という積極的な戦略をおこなってきた。

A Report of Surgeon General（米国公衆衛生総監報告）
“The health consequences of smoking
– 50 Years of Progress”(2014年報告)
「喫煙による健康影響：50年間の進歩」



3） 1964年のSurgeon General’s report 後、
喫煙はほぼすべてのがんの原因であること、
健康状態を悪化させること、胎児に悪影響があることが判明した。
最初の本報告から50年後の現在でも、これまで無関係と思われていた疾病、
つまり、糖尿病や関節リウマチ、大腸癌などが喫煙と関連することが明らか
となりつつある。
4） 受動喫煙が（非喫煙者の）発がん、呼吸器疾患、心疾患の原因となること、
および、乳幼児や小児の健康に悪影響を及ぼすことが明らかとなった。
5）過去50年で女性の喫煙者が急増した結果、現在、喫煙による
女性の被害（肺がん、COPD、心疾患）は、男性と同程度に増加した。
6） 喫煙は、多くの疾患の原因になるだけでなく、全身の炎症、免疫機能の障害
などの悪影響が発生することが分かった。

A Report of Surgeon General（米国公衆衛生総監報告）
“The health consequences of smoking
– 50 Years of Progress”(2014年報告)
「喫煙による健康影響：50年間の進歩」

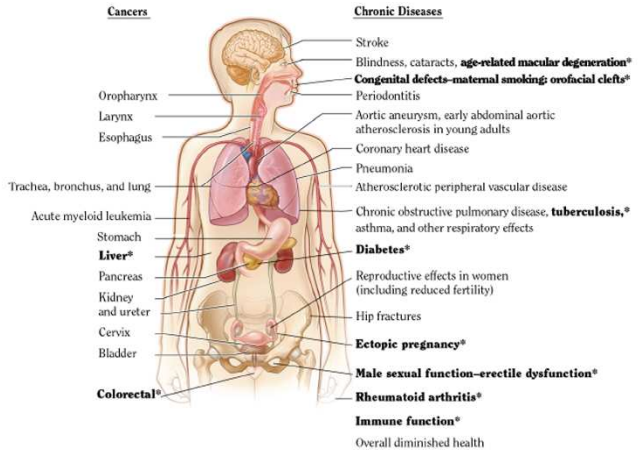


7) 1964年以降、喫煙率は減少したが、人種や教育レベル、社会経済的要因（貧困）により、喫煙率の不均衡（高い集団）が米国全体に残っている。
8) 1964年の本報告以来、包括的な喫煙対策が取られたことでタバコの使用が効果的に減少したことが証明されてきた。さらに強力な喫煙対策を継続することで、より大きな効果が期待される。
9) タバコ、その他のタバコ製品の使用が、早世と疾病として米国社会にもたらした負担は莫大なものである。タバコ・タバコ製品を消滅させることで、社会的な負担は急速に減少する。
10)喫煙と健康（への悪影響）に関する50年間の本報告は、タバコの消費量を減らし、喫煙関連疾患と早世を予防するための公衆衛生活動に重大で科学的な根拠を提供してきた。

能動喫煙（左）と受動喫煙（右）により発生する疾患

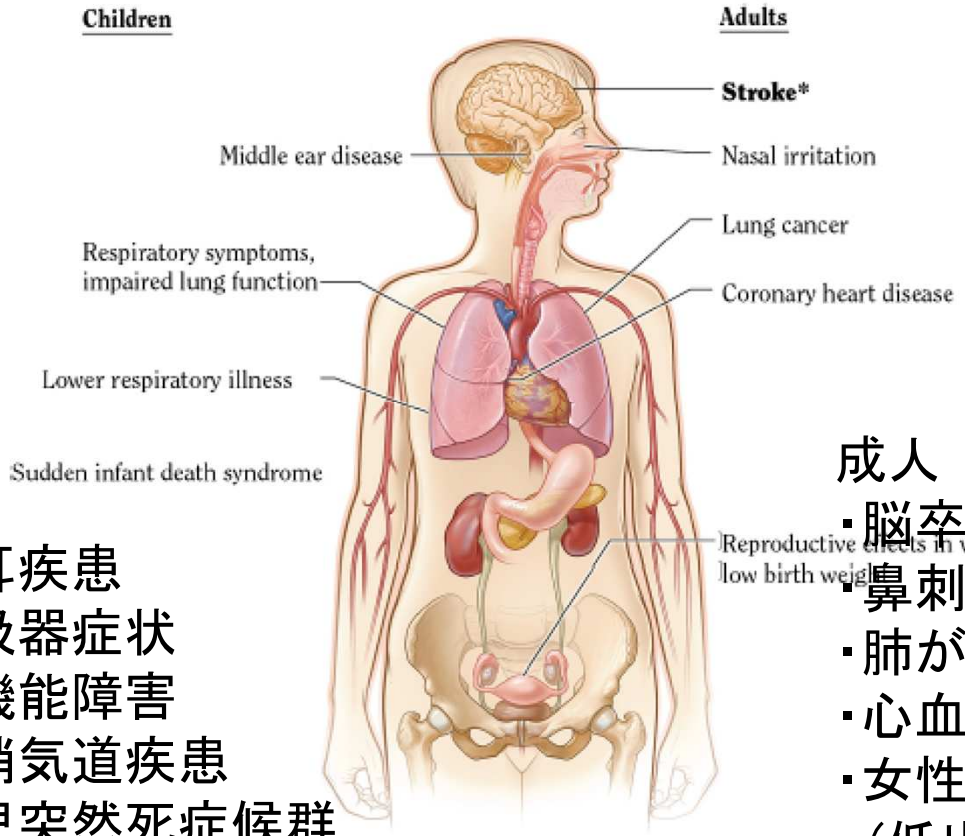
2014報告より

Figure 1.1A The health consequences causally linked to smoking



Source: USDHHS 2004, 2006, 2012.
Note: Each condition presented in bold text and followed by an asterisk (*) is a new disease that has been causally linked to smoking in this report.

Figure 1.1B The health consequences causally linked to exposure to secondhand smoke



小児
- 中耳疾患
- 呼吸器症状
- 肺機能障害
- 末梢気道疾患
- 乳児突然死症候群

成人
- 脳卒中
- 鼻刺激症状
- 肺がん
- 心血管疾患
- 女性の生殖機能（低出生体重児）

Source: USDHHS 2004, 2006.
Note: Each condition presented in bold text and followed by an asterisk (*) is a new disease that has been causally linked to exposure to secondhand smoke in this report.

Surgeon General's Report（2006年報告，p11）
Major Conclusions、6つの結論解説

With regard to the involuntary exposure of nonsmokers to tobacco smoke, the scientific evidence now supports the following major conclusions:

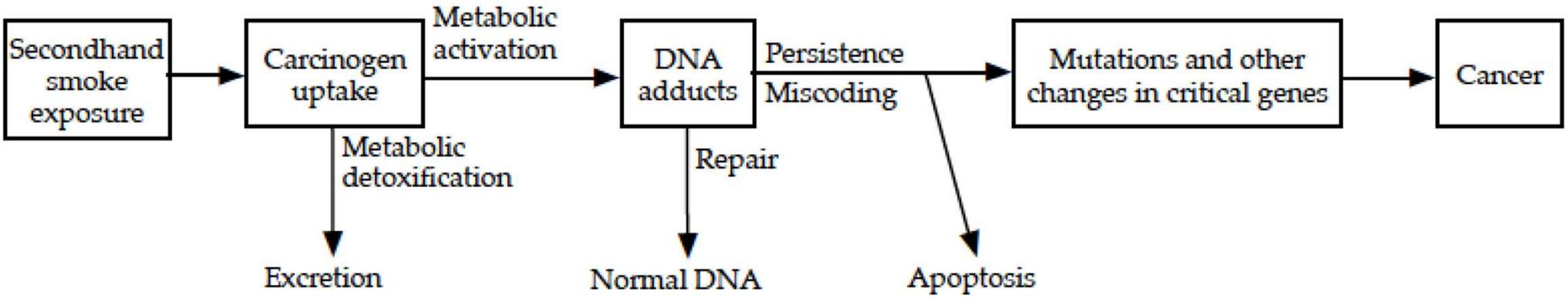
1. Secondhand smoke causes premature death and disease in children and in adults who do not smoke.

1. 受動喫煙はタバコを吸わない成人、小児の早世と疾病の原因となる

A Report of the Surgeon General　(2006年報告，43頁）
The Health Consequences of Involuntary Exposure to Tobacco Smoke

発がん性のメカニズムに関する結論

Figure 2.2　Scheme showing the steps linking secondhand smoke exposure and cancer via tobacco smoke carcinogens



受動喫煙→発がん性物質摂取→DNA付加体による損傷→突然変異→発がん

個別の論文：職場の受動喫煙で非喫煙者の肺がんリスクが**1.24**倍に増加

25研究の
メタ分析

Leslie S, et al. Am J Pub Health 97:545–551, 2007.

Surgeon General's Report

受動喫煙による肺がんリスク：1.15～1.43倍（メタアナリシスによる定量）

(2006年報告，436頁)

Table 7.4 Quantitative estimate of lung cancer risk with differing sources of exposure to secondhand smoke

Study	Data source	Exposure vs. referent	Relative risk	95% confidence interval
過去の論文（配偶者から曝露）	**Previous meta-analyses**			
Hackshaw et al. 1997	37 studies	Smoking vs. nonsmoking spouse	1.24	1.13–1.36
Zhong et al. 2000	40 studies (including 37 from Hackshaw et al. 1997)	Smoking vs. nonsmoking husband	1.20	1.12–1.29
2006年報告（配偶者から曝露）	**Spousal smoking (52 studies)**			
Meta-analysis conducted for this 2006 Surgeon General's report	Case-control (44 studies)	Smoking vs. nonsmoking spouse	1.21	1.13–1.30
	Cohort (8 studies)	Smoking vs. nonsmoking spouse	1.29	1.125–1.49
	Men	Smoking vs. nonsmoking wife	1.37	1.05–1.79
米国公衆衛生総監2006年報告書のためのメタアナリシス	Women	Smoking vs. nonsmoking husband	1.22	1.13–1.31
	United States and Canada	Smoking vs. nonsmoking spouse	1.15	1.04–1.26
	Europe	Smoking vs. nonsmoking spouse	1.16	1.03–1.30
	Asia	Smoking vs. nonsmoking spouse	1.43	1.24–1.66

Table 7.4の続き

職場の受動喫煙(25研究) **Workplace exposure (25 studies)** 肺がん1.13～1.32倍

Meta-analysis conducted for this 2006 Surgeon General's report	Nonsmokers (25 studies)	Workplace secondhand smoke vs. none	1.22	1.13–1.33
	Nonsmoking men (11 studies)	Workplace secondhand smoke vs. none	1.12	0.86–1.50
	Nonsmoking women (25 studies)	Workplace secondhand smoke vs. none	1.22	1.10–1.35
	Nonsmokers in the United States and Canada (8 studies)	Workplace secondhand smoke vs. none	1.24	1.03–1.49
	Nonsmokers in Europe (7 studies)	Workplace secondhand smoke vs. none	1.13	0.96–1.34
	Nonsmokers in Asia (10 studies)	Workplace secondhand smoke vs. none	1.32	1.13–1.55

米国公衆衛生総監
2006年報告書
のための
メタアナリシス

小児期の受動喫煙(24研究) **Childhood exposure (24 studies)** 肺がん0.81～1.59倍

Meta-analysis conducted for this 2006 Surgeon General's report	Men and women	Maternal smoking	1.15	0.86–1.52
	Men and women	Paternal smoking	1.10	0.89–1.36
	Men and women	Smoking by either parent	1.11	0.94–1.31
	Women	Maternal smoking	1.28	0.93–1.78
	Women	Paternal smoking	1.17	0.91–1.50
	United States and Canada (8 studies)	Smoking by either parent	0.93	0.81–1.07
	Europe (6 studies)	Smoking by either parent	0.81	0.71–0.92
	Asia (10 studies)	Smoking by either parent	1.59	1.18–2.15

2006年報告書
のための
メタアナリシス

(2006年報告，436頁)

(2006年報告，445頁）

Surgeon General's Report

Conclusions

結論：肺がん

1. 受動喫煙が非喫煙者の肺がんのリスクになることについて、十分な証拠が得られた。
   職場、家庭、地域と関係なく結論づけられた。
2. 喫煙者とともに生活することにより、（非喫煙者の）肺がんリスクは20～30%上昇する。

Lung Cancer

1. The evidence is sufficient to infer a causal relationship between secondhand smoke exposure and lung cancer among lifetime nonsmokers. This conclusion extends to all secondhand smoke exposure, regardless of location.

2. The pooled evidence indicates a 20 to 30 percent increase in the risk of lung cancer from secondhand smoke exposure associated with living with a smoker.

受動喫煙による肺がん：
日本人のエビデンス（大規模コホート）、家庭内曝露

Int. J. Cancer: **122**, 653–657 (2008)

Passive smoking and lung cancer in Japanese non-smoking women: A prospective study

Norie Kurahashi[1]*, Manami Inoue[1], Ying Liu[1], Motoki Iwasaki[1], Shizuka Sasazuki[1], Tomotaka Sobue[2] and Shoichiro Tsugane[1] for the JPHC Study Group

TABLE II – ASSOCIATION BETWEEN LUNG CANCER INCIDENCE AND PASSIVE SMOKING FROM THE HUSBAND IN LIFELONG NON-SMOKING WOMEN (n = 28,414)

Type of exposure	All lung cancer			Adenocarcinoma		
	Case (N)	Person-years	Multivariate HR (95% CI)	Case (N)	Person-years	Multivariate HR (95% CI)
From husband						
Never	25	97,466	1	15	97,392	1
Former	28	94,427	1.12 (0.63–1.98)	21	94,358	1.50 (0.73–3.09)
Current	56	185,919	1.34 (0.81–2.21)	46	185,855	2.03 (1.07–3.86)
Number of cigarettes per day						
<20	14	52,441	1.02 (0.51–2.04)	13	52,438	1.73 (0.77–3.88)
≥20	41	131,107	1.47 (0.87–2.49)	33	131,055	2.20 (1.13–4.28)
p for trend			0.14			0.02
Pack years of exposure						
<30	17	76,125	1.05 (0.55–2.02)	16	76,122	1.86 (0.86–4.01)
≥30	36	104,330	1.46 (0.85–2.50)	28	104,279	2.06 (1.04–4.10)
p for trend			0.17			0.03

Adjusted for age, study area, alcohol consumption, family history of lung cancer and menopausal status.

全肺がん：夫が非喫煙の妻の発症を1.0、夫が元喫煙は1.12倍、現喫煙は1.34倍
夫の喫煙本数が20本以下で1.02倍、20本以上は1.47倍
夫からの曝露が30箱・年以下で1.05倍、30箱・年以上は1.46倍

受動喫煙による肺がん：
日本人のエビデンス（大規模コホート）、家庭内曝露

Int. J. Cancer: **122**, 653–657 (2008)

Passive smoking and lung cancer in Japanese non-smoking women: A prospective study

Norie Kurahashi[1]*, Manami Inoue[1], Ying Liu[1], Motoki Iwasaki[1], Shizuka Sasazuki[1], Tomotaka Sobue[2] and Shoichiro Tsugane[1] for the JPHC Study Group

TABLE II – ASSOCIATION BETWEEN LUNG CANCER INCIDENCE AND PASSIVE SMOKING FROM THE HUSBAND IN LIFELONG NON-SMOKING WOMEN (n = 28,414)

Type of exposure	All lung cancer			Adenocarcinoma		
	Case (N)	Person-years	Multivariate HR (95% CI)	Case (N)	Person-years	Multivariate HR (95% CI)
From husband						
Never	25	97,466	1	15	97,392	1
Former	28	94,427	1.12 (0.63–1.98)	21	94,358	1.50 (0.73–3.09)
Current	56	185,919	1.34 (0.81–2.21)	46	185,855	2.03 (1.07–3.86)
Number of cigarettes per day						
<20	14	52,441	1.02 (0.51–2.04)	13	52,438	1.73 (0.77–3.88)
≥20	41	131,107	1.47 (0.87–2.49)	33	131,055	2.20 (1.13–4.28)
p for trend			0.14			0.02
Pack years of exposure						
<30	17	76,125	1.05 (0.55–2.02)	16	76,122	1.86 (0.86–4.01)
≥30	36	104,330	1.46 (0.85–2.50)	28	104,279	2.06 (1.04–4.10)
p for trend			0.17			0.03

Adjusted for age, study area, alcohol consumption, family history of lung cancer and menopausal status.

肺腺がん：夫が非喫煙の妻の発症を1.0、夫が元喫煙は1.50倍、現喫煙は2.03倍
夫の喫煙が20本以下は1.73倍、20本以上は2.20倍で、量反応関係あり（p=0.02）
夫からの曝露が30箱・年以下は1.86倍、30箱・年は2.06倍、量反応関係あり（p=0.03）

受動喫煙による肺がん：
日本人のエビデンス（大規模コホート）、家庭・職場曝露

Int. J. Cancer: **122**, 653–657 (2008)

Passive smoking and lung cancer in Japanese non-smoking women: A prospective study

Norie Kurahashi[1*], Manami Inoue[1], Ying Liu[1], Motoki Iwasaki[1], Shizuka Sasazuki[1], Tomotaka Sobue[2] and Shoichiro Tsugane[1] for the JPHC Study Group

TABLE III – ASSOCIATION BETWEEN LUNG CANCER INCIDENCE AND PASSIVE SMOKING AT THE WORKPLACE AND FROM TWO SOURCES IN LIFELONG NON-SMOKING WOMEN (n = 28,414)

Type of exposure	All lung cancer			Adenocarcinoma		
	Case (N)	Person-years	Multivariate HR (95%CI)	Case (N)	Person-years	Multivariate HR (95% CI)
At workplace						
<1 time/week	77	279,421	1	60	279,299	1
≥1 times/week	30	94,652	1.32 (0.85–2.04)	20	94,568	1.16 (0.69–1.97)
From two sources						
Source of exposure						
Almost never[1]	17	80,428	1	12	80,395	1
Workplace only[2]	8	16,236	2.74 (1.11–6.76)	3	16,195	1.21 (0.26–5.55)
Husband only[3]	60	198,994	1.49 (0.84–2.62)	48	198,904	1.79 (0.90–3.55)
Workplace + Husband	22	78,417	1.61 (0.83–3.11)	17	78,373	1.93 (0.88–4.23)

[1]Women exposed at the workplace less than one time per week.–[2]Women exposed at the workplace one or more times per week.–[3]Women exposed from husbands who are former or current smokers.
Adjusted for age, study area, alcohol consumption, family history of lung cancer and menopausal status.

全肺がん：職場での曝露が週1時間以下を1.0、週1時間以上の曝露で1.32倍
職場でも家庭でも曝露なしを1.0、職場のみで曝露は2.74倍
家庭（夫）からのみ曝露で1.49倍、職場と家庭の両方の曝露で1.61倍

受動喫煙による肺がん：
日本人のエビデンス（大規模コホート）、家庭・職場曝露

Int. J. Cancer: **122**, 653–657 (2008)

Passive smoking and lung cancer in Japanese non-smoking women: A prospective study

Norie Kurahashi[1*], Manami Inoue[1], Ying Liu[1], Motoki Iwasaki[1], Shizuka Sasazuki[1], Tomotaka Sobue[2] and Shoichiro Tsugane[1] for the JPHC Study Group

TABLE III – ASSOCIATION BETWEEN LUNG CANCER INCIDENCE AND PASSIVE SMOKING AT THE WORKPLACE AND FROM TWO SOURCES IN LIFELONG NON-SMOKING WOMEN (n = 28,414)

Type of exposure	All lung cancer			Adenocarcinoma		
	Case (N)	Person-years	Multivariate HR (95%CI)	Case (N)	Person-years	Multivariate HR (95% CI)
At workplace						
<1 time/week	77	279,421	1	60	279,299	1
≥1 times/week	30	94,652	1.32 (0.85–2.04)	20	94,568	1.16 (0.69–1.97)
From two sources						
Source of exposure						
Almost never[1]	17	80,428	1	12	80,395	1
Workplace only[2]	8	16,236	2.74 (1.11–6.76)	3	16,195	1.21 (0.26–5.55)
Husband only[3]	60	198,994	1.49 (0.84–2.62)	48	198,904	1.79 (0.90–3.55)
Workplace + Husband	22	78,417	1.61 (0.83–3.11)	17	78,373	1.93 (0.88–4.23)

[1]Women exposed at the workplace less than one time per week.–[2]Women exposed at the workplace one or more times per week.–[3]Women exposed from husbands who are former or current smokers.
Adjusted for age, study area, alcohol consumption, family history of lung cancer and menopausal status.

肺腺がん：職場での曝露が週1時間以下を1.0、週1時間以上の曝露で1.16倍
職場でも家庭でも曝露なしを1.0、職場のみで曝露は1.21倍、
家庭（夫）からのみ曝露で1.79倍、職場と家庭の両方の曝露で1.93倍

受動喫煙により乳がんのリスクも上昇、1.15倍　(米国公衆衛生総監2006年報告，471頁)

Table 7.10　Pooled risk estimates and 95% confidence intervals (CI) for breast cancer meta-analysis

	All women		Premenopausal		Postmenopausal	
Exposure	**n***	**Relative risk (95% CI)**	**n**	**Relative risk (95% CI)**	**n**	**Relative risk (95% CI)**
Adulthood						
All sources	18	1.15 (1.02–1.29) [0.000]†	10	1.45 (1.04–2.01) [0.000]	9	0.90 (0.81–1.01) [0.691]
Spouse	9	1.17 (0.96–1.44) [0.002]	4	1.40 (0.92–2.12) [0.1]	3	0.86 (0.67–1.12) [0.645]
Home	8	1.01 (0.85–1.19) [0.006]	4	1.28 (0.94–1.74) [0.355]	3	0.92 (0.76–1.11) [0.591]
Work	6	1.06 (0.84–1.35) [0.008]	4	1.21 (0.70–2.09) [0.000]	3	0.83 (0.53–1.29) [0.086]
Childhood (parent)	9	1.01 (0.90–1.12) [0.101]	4	1.14 (0.90–1.45) [0.342]	3	1.04 (0.86–1.26) [0.242]
Both childhood and adulthood	4	1.39 (0.88–2.18) [0.021]	3	1.63 (0.68–3.91) [0.016]	2	1.02 (0.74–1.42) [0.160]
Ever exposed (in studies measuring lifetime exposure)	10	1.40 (1.12–1.76) [0.000]	6	1.85 (1.19–2.87) [0.001]	5	1.04 (0.84–1.30) [0.048]
"Best" of each study‡	21	1.20 (1.08–1.35) [0.000]	11	1.64 (1.25–2.14) [0.001]	10	1.00 (0.88–1.12) [0.321]
Cohort studies	7	1.02 (0.92–1.13) [0.162]				
Case-control studies	14	1.40 (1.17–1.67) [0.000]				

*n = Number of studies included in each analysis.
†[in brackets] = p value for test of heterogeneity (null hypothesis is no heterogeneity).
‡"Best" of each study includes the most comprehensive measure of association from each study: ever being exposed in any setting was preferred over all sources during adulthood, which was preferred over spousal exposure.

Conclusions

受動喫煙による発がんについての結論
1. 50種類以上の発がん性物質が特定（2004年IARCは64種類、現在は70種類）
2. 受動喫煙による発がん性は動物実験で明白
3. 喫煙により尿中のタバコ由来発がん性物質の代謝産物が有意に上昇、
これらの代謝物質は肺がんの発生を高めることと関連
4. 受動喫煙による発がんのメカニズムは、そのレベルは低いものの、能動喫煙と類似

Evidence of Carcinogenic Effects from Secondhand Smoke Exposure

1. More than 50 carcinogens have been identified in sidestream and secondhand smoke.

2. The evidence is sufficient to infer a causal relationship between exposure to secondhand smoke and its condensates and tumors in laboratory animals.

3. The evidence is sufficient to infer that exposure of nonsmokers to secondhand smoke causes a significant increase in urinary levels of metabolites of the tobacco-specific lung carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). The presence of these metabolites links exposure to secondhand smoke with an increased risk for lung cancer.

4. The mechanisms by which secondhand smoke causes lung cancer are probably similar to those observed in smokers. The overall risk of secondhand smoke exposure, compared with active smoking, is diminished by a substantially lower carcinogenic dose.

2006, p65

Surgeon General's Report

Sudden Infant Death Syndrome

2006年報告書, p180-194

Conclusion

1. The evidence is sufficient to infer a causal relationship between exposure to secondhand smoke and sudden infant death syndrome.

乳児突然死症候群に関する結論1：
受動喫煙と乳児突然死症候群(SIDS)には十分な因果関係あり

Case-control　study13報告のうち9番目の報告

Blair et al. 1996	Case-control (195 cases, 780 controls, 4 per case matched for age) United Kingdom (Southwest, Yorkshire, and Trent) 1993–1995	• Smoking status of mother, father, and others in household • Number of smokers in household • Number of cigarettes smoked daily in household	Postpartum exposure from • Mother • Father • Other household members

Surgeon General's SIDS

Sudden In

2006年報告書,
主要な結論1
乳児突然死症候群の原因
p180–194

Case–control study13報告のうち9番目の報告

両親（特に、母）、自宅内喫煙者の数、自宅内喫煙本数、曝露時間が長い程、SIDSのリスク上昇

Parental smoking status
Only father smoked: OR = 3.41 (95% CI, 1.98–5.88)
Only mother smoked: OR = 7.01 (95% CI, 3.91–12.56)
Both parents smoked: OR = 8.41 (95% CI, 5.08–13.92)
Adjusted for maternal smoking during pregnancy

Multivariate analysis
Postnatal paternal smoking, additive to maternal smoking
OR = 2.50 (95% CI, 1.48–4.22)
Adjusted for mother's age, mothers without partners, parity, multiple births, short gestation, socioeconomic status, sleeping position, maternal alcohol consump parental use of illegal drugs, parental bed sharing, breastfeeding, and birth weight

Postnatal paternal smoking, additional adjustment for maternal smoking during pregnancy
Nonsignificant (p = 0.1601)

Number of smokers at home
1 smoker: OR = 2.44 (95% CI, 1.36–4.37)
2 smokers: OR = 5.15 (95% CI, 3.24–8.21)
>2 smokers: OR = 10.43 (95% CI, 3.34–32.54)

Cigarettes/day smoked at home
1–19 cigarettes/day: OR = 2.47 (95% CI, 1.29–4.73)
20–39 cigarettes/day: OR = 3.96 (95% CI, 2.40–6.55)
>39 cigarettes/day: OR = 7.57 (95% CI, 4.00–14.32)

Infant's daily exposure to tobacco smoke (hours)
1–2: OR = 1.99 (95% CI, 1.14–3.46)
3–5 : OR = 3.84 (95% CI, 1.97–7.48)
6–8: OR = 6.78 (95% CI, 3.17–14.49)
>8: OR = 8.29 (95% CI, 4.28–16.05)

Exposure data were self-reported (questionnaire); multivariate analysis found nonsignificant effect for other smoking members of household; unclear if postnatal dose-response analyses adjusted for maternal prenatal smoking or other confounding factors; dose-response analyses were limited to households where smoking was allowed in the same room as the infant; exposure to secondhand smoke in the home has an independent effect on the risk of SIDS

父のみ	3.41倍
母のみ	7.01倍
両親	8.41倍

Blair et al. 1996

自宅での喫煙者数	
1名	2.44倍
2名	5.15倍
2名以上	10.43倍

自宅での喫煙本数	
1~19本	2.47倍
20～39本	3.96倍
39本以上	10.43倍

受動喫煙曝露時間	
1~2時間	1.99倍
3~5時間	3.84倍
6~8時間	6.78倍
8時間以上	8.29倍

Mechanisms of Respiratory Tract Injury and Disease Caused by Secondhand Smoke Exposure

2006年報告書、46～52頁

受動喫煙による呼吸器系疾患の障害と疾患のメカニズム
受動喫煙と下記の疾患に関する研究から下記の結論を得た。

- 気管支喘息
- 呼吸器感染症
- 慢性閉塞性肺疾患（COPD）
- 乳児突然死症候群（SIDS）

Conclusions

1. The evidence indicates multiple mechanisms by which secondhand smoke exposure causes injury to the respiratory tract.

2. The evidence indicates mechanisms by which secondhand smoke exposure could increase the risk for sudden infant death syndrome.

呼吸器系疾患の結論

1. 科学的証拠により、受動喫煙は呼吸器を紹介することは明らか
2. 乳児突然死症候群のリスクも上昇させることから、そのメカニズムも示された。

個別論文のメタアナリシス：職場と家庭の受動喫煙で心血管疾患が1.25倍に増加

Study (Year)	Exposure	No Exposure
	no. of events/no. at risk	
Cohort		
Hirayama[7,8] (1984)	376/69,645	118/21,895
Garland et al.[9] (1985)	17/492	2/203
Svendsen et al.[10] (1987)	5/286	8/959
Butler[11] (1988)	4/430	60/6077
Butler[11] (1988)	50/2802	95/3630
Sandler et al.[12] (1989)	673/10,799	685/8236
Hole et al.[13] (1989)	54/1538	30/917
Humble et al.[14] (1990)	49/296	27/217
Steenland et al.[15] (1996)	571/67,369	2574/164,831
Kawachi et al.[16] (1997)	135/25,959	17/6087
	Case Patients	**Controls**
	no. with exposure/no. without exposure	
Case–control		
Lee et al.[17] (1986)	70/48	269/182
He et al.[18] (1989)	25/9	30/38
Jackson[19] (1989)	18/21	87/148
Dobson et al.[20] (1991)	65/278	133/692
La Vecchia et al.[21] (1993)	24/66	37/157
He et al.[22] (1994)	48/11	76/50
Muscat and Wynder[23] (1995)	63/51	70/88
Ciruzzi et al.[24] (1998)	131/205	117/329
Overall		

All

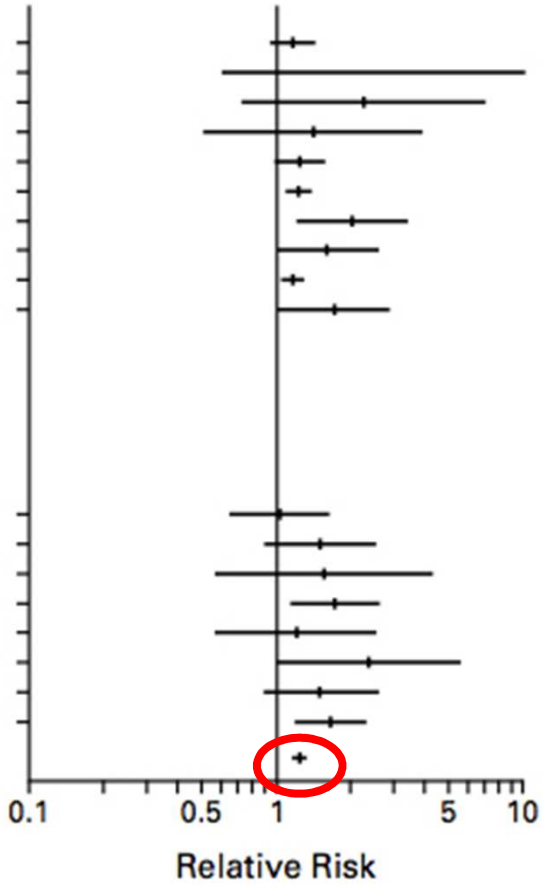


Figure 1. Relative Risks of Coronary Heart Disease Associated with Passive Smoking among Nonsmokers in 18 Epidemiologic Studies. The horizontal bars represent the 95 percent confidence intervals. The relative risk in the study by Garland et al.[9] was 14.9.

He J, et al.: Passive smoking and the risk of coronary heart disease– A meta-analysis of epidemiologic studies. N Engl J Med. 340: 920–926, 1999.

個別論文のメタアナリシス

夫の喫煙本数が多いほど妻の冠動脈疾患が増加

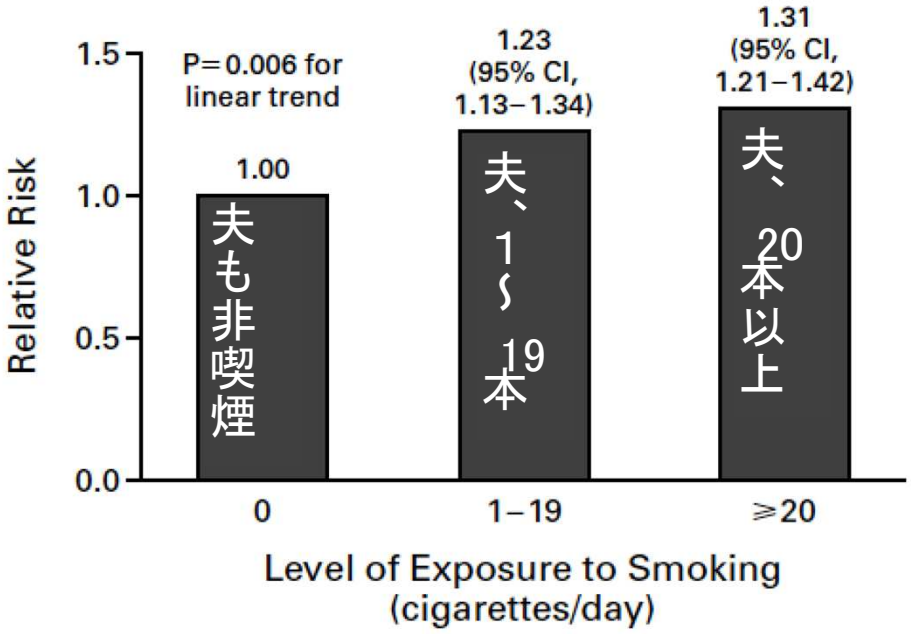


Figure 2. Pooled Relative Risks of Coronary Heart Disease Associated with Various Levels of Exposure to Spouse's Smoking among Nonsmokers.

Data were obtained from Hirayama,[7,8] Svendsen et al.,[10] Sandler et al.,[12] Hole et al.,[13] Steenland et al.,[15] He et al.,[18,22] and La Vecchia et al.[21] CI denotes confidence interval.

喫煙する夫との同居が長いほど妻の冠動脈疾患が増加

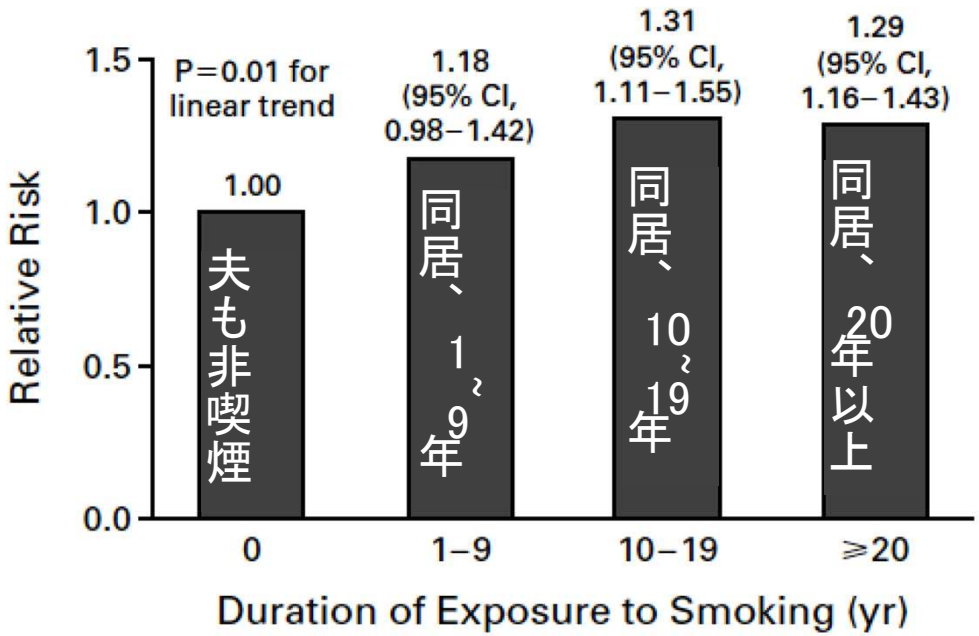


Figure 3. Pooled Relative Risks of Coronary Heart Disease Associated with Various Durations of Exposure to Spouse's Smoking among Nonsmokers.

Data were obtained from Butler,[11] Steenland et al.,[15] Kawachi et al.,[16] He et al.,[18,22] Muscat and Wynder,[23] and Ciruzzi et al.[24] CI denotes confidence interval.

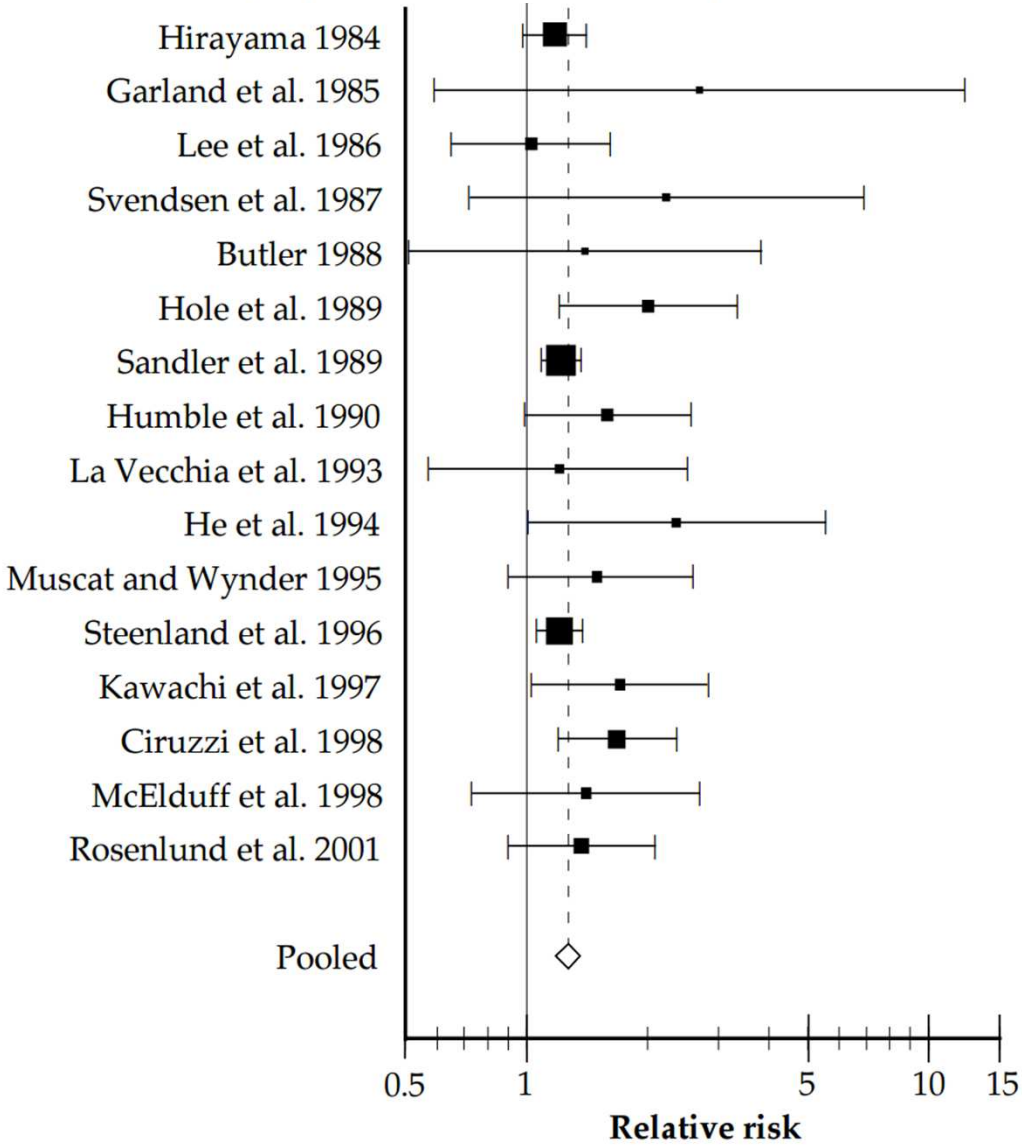
He J, et al.: Passive smoking and the risk of coronary heart disease– A meta-analysis of epidemiologic studies. N Engl J Med. 340: 920–926, 1999.

Surgeon General's Report 2006.　(2006年報告，524頁）

受動喫煙による冠動脈疾患リスク：1.27 倍（95%CI, 1.19−1.36）

Figure 8.1　Relative risks of coronary heart disease associated with secondhand smoke exposure among nonsmokers*

Note: The horizontal bars represent the 95% confidence intervals (CIs), and the size of the box for each study reflects each study's weight in the pooled estimate, with a larger box indicating a larger weight.
*Pooled estimate = 1.27 (95% CI, 1.19–1.36), the dashed line.



米国公衆衛生総監
報告書2006のための
メタアナリシス

Surgeon General's Report 2006.　米国公衆衛生総監2006報告書のためのメタアナリシス　(2006年報告，526頁)

受動喫煙による冠動脈疾患リスク：量・反応関係あり

Table 8.4　Studies included in the dose-response meta-analysis and pooled results

	Low to moderate exposure		Moderate to high exposure	
Study	**Cigarettes/day**	**Relative risk (95% confidence interval)**	**Cigarettes/day**	**Relative risk (95% confidence interval)**
Svendsen et al. 1987	1–19	0.90 (0.02–6.70)	>19	3.21 (0.71–11.98)
Hole et al. 1989	1–15	2.09 (0.60–7.23)	>15	4.12 (1.21–14.05)
Hirayama et al. 1990	1–19	1.08 (0.9–1.3)	>19	1.3 (1.06–1.6)
La Vecchia et al. 1993	1–14	1.13 (0.45–2.82)	>14	1.3 (0.5–3.4)
He et al. 1994	6–20	1.61 (0.49–5.34)	>20	3.56 (0.81–15.58)
Steenland et al. 1996	1–19	1.31 (1.06–1.62)	>19	1.14 (0.97–1.34)
Ciruzzi et al. 1998	1–20	1.24 (0.61–2.52)	>20	4.03 (0.99–16.32)
Rosenlund et al. 2001	1–19	1.02 (0.73–1.42)	>19	1.58 (0.97–2.56)
Pooled results	Fixed effects: Random effects:	1.16 (1.03–1.32) 1.16 (1.03–1.32)		1.26 (1.12–1.42) 1.44 (1.13–1.82)

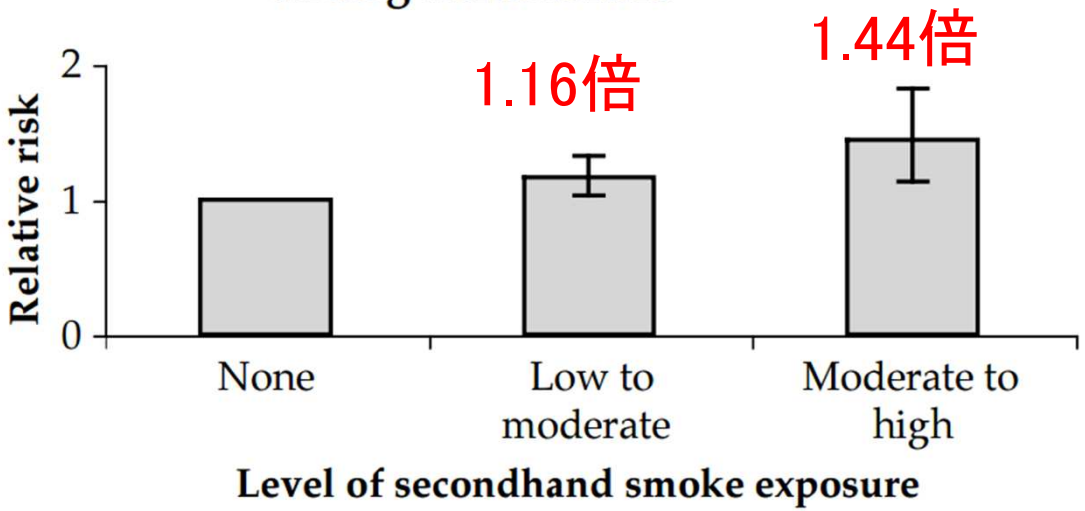
軽～中等度曝露　　中～高度曝露

Surgeon General's Report 2006.

(2006年報告，526頁）

（前頁のグラフ化）受動喫煙による冠動脈疾患リスク：量・反応関係あり

Figure 8.3 **Pooled relative risks of coronary heart disease associated with various levels of exposure to secondhand smoke among nonsmokers**



Note: None, low to moderate (1–14 or 1–19 cigarettes per day), and moderate to high (≥15 or ≥20 cigarettes per day).

左：受動喫煙なし、
中：軽～中度曝露（1~14本、1～19本／日）
右：中～高度曝露（　15本、　20本以上／日）

米国公衆衛生総監報告書2006のためのメタアナリシス

Major Conclusions

4. The scientific evidence indicates that there is no risk-free level of exposure to secondhand smoke.

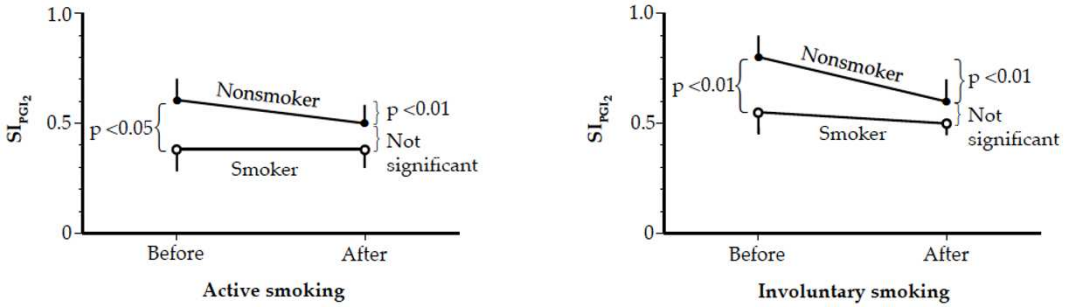
米国公衆衛生総監報告(2006)の主要な結論
4. 受動喫煙に安全なレベル(閾値)は存在しない

循環器系疾患への影響

2006年報告書, p53

・喫煙者・非喫煙者へのタバコ煙曝露前後の血小板凝集能

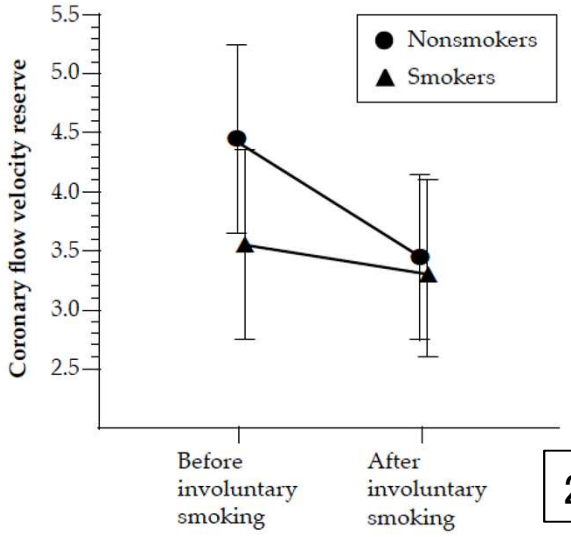
Figure 2.4 Effect of active and involuntary smoking on platelet aggregation in smokers and nonsmokers



Note: The sensitivity index, SI_{PGI_2}, is defined as the inverse of the concentration of prostaglandin I_2, which is necessary to inhibit adenosine disphosphate-induced platelet aggregation by 50 percent. Lower values of SI_{PGI_2} indicate greater platelet aggregation.
Source: Burghuber et al. 1986. Adapted with permission.

・タバコ煙曝露による冠動脈血流速度の低下

Figure 2.6 Coronary flow velocity changes before and after secondhand smoke exposure



2006, p57

2006年報告書, p64

Conclusions 循環器系疾患の結論

1. The evidence is sufficient to infer that exposure to secondhand smoke has a prothrombotic effect.

2. The evidence is sufficient to infer that exposure to secondhand smoke causes endothelial cell dysfunctions.

3. The evidence is sufficient to infer that exposure to secondhand smoke causes atherosclerosis in animal models.

1. 科学的証拠により、受動喫煙は血栓形成促進効果を亢進させることは明らか
2. 受動喫煙により血管内皮の機能が障害されることが明らか
3. 動物実験でも動脈硬化を促進させることが明らか

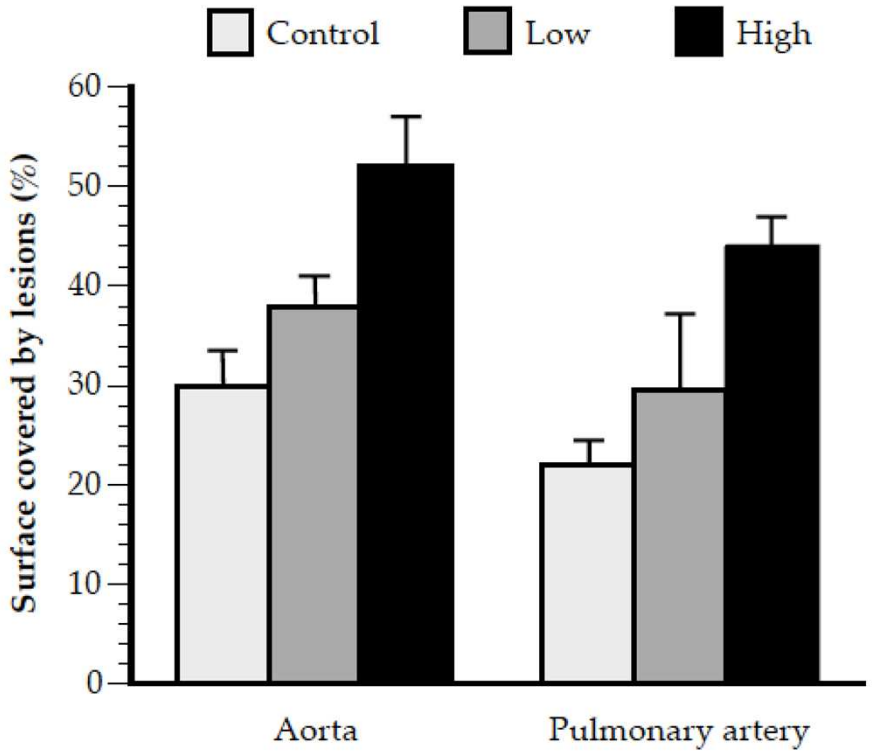
Major Conclusions

4. The scientific evidence indicates that there is no risk-free level of exposure to secondhand smoke.

米国公衆衛生総監報告(2006)の主要な結論
4. 受動喫煙に安全なレベル(閾値)は存在しない

動物実験でも受動喫煙曝露による悪影響

Figure 2.7 Secondhand smoke exposure and lipid deposits in rabbits



Note: Exposure to secondhand smoke increased lipid deposits in arteries of rabbits in a dose-dependent manner. Bars are for controls (clear air), and low doses and high doses of secondhand smoke exposures. Error bars represent standard error of the mean.
Source: Zhu et al. 1993b. Reprinted with permission.

2006年報告書，主要な結論4、p65

呼吸器系疾患（と循環器疾患）のメカニズムから
「受動喫煙の曝露に安全閾は存在しない」と考えられる

functioning of the heart, blood, and vascular systems in ways that increase the risk of a cardiac event. Furthermore, many of these acute and chronic changes in blood and vascular function appear to be as large as those seen in active smokers. The immediate effects in some measures of blood and vascular functioning among nonsmokers from even brief exposures (i.e., 30 minutes or less) to secondhand smoke are comparable in magnitude to the effects observed in active smokers. Thus, the evidence reviewed in this chapter supports the biologic plausibility of adverse cardiovascular health outcomes that are associated with exposure to secondhand smoke, which are reviewed in Chapter 8.

As the portal of entry for secondhand smoke, the respiratory system is the initial site of deposition for the particulate and gaseous compounds found in secondhand smoke. This chapter identifies the multiple mechanisms by which secondhand smoke exposure can induce both acute and chronic adverse health effects within the respiratory tract that affect infants, children, and adults. The evidence for underlying mechanisms of respiratory injury from exposure to secondhand smoke suggests that a safe level of exposure may not exist, thus implying that any exposure carries some risk. For infants, children, and adults with asthma or with more sensitive respiratory systems, even very brief exposures to secondhand smoke can trigger intense bronchopulmonary responses that could be life threatening in the most susceptible individuals.

Animal and human studies indicate that prenatal and postnatal exposure to nicotine and other toxicants in tobacco smoke may affect the neuroregulation of breathing, apneic spells, and sudden infant death. Experimental data on the neurotoxicity of prenatal and neonatal exposure to nicotine and secondhand smoke in animal models can be related to several potential causal mechanisms for SIDS, including adverse effects on brain cell development, synaptic development and function, and neurobehavioral activity. Finally, studies have documented that exposure to tobacco smoke from active smoking has a broad effect on immune function and host defenses against infectious agents. Evidence indicates that exposure to secondhand smoke appears to also impair immune function in both children and adult nonsmokers, which increases susceptibility to infection.

Major Conclusions

2. Children exposed to secondhand smoke are at an increased risk for sudden infant death syndrome (SIDS), acute respiratory infections, ear problems, and more severe asthma. Smoking by parents causes respiratory symptoms and slows lung growth in their children.

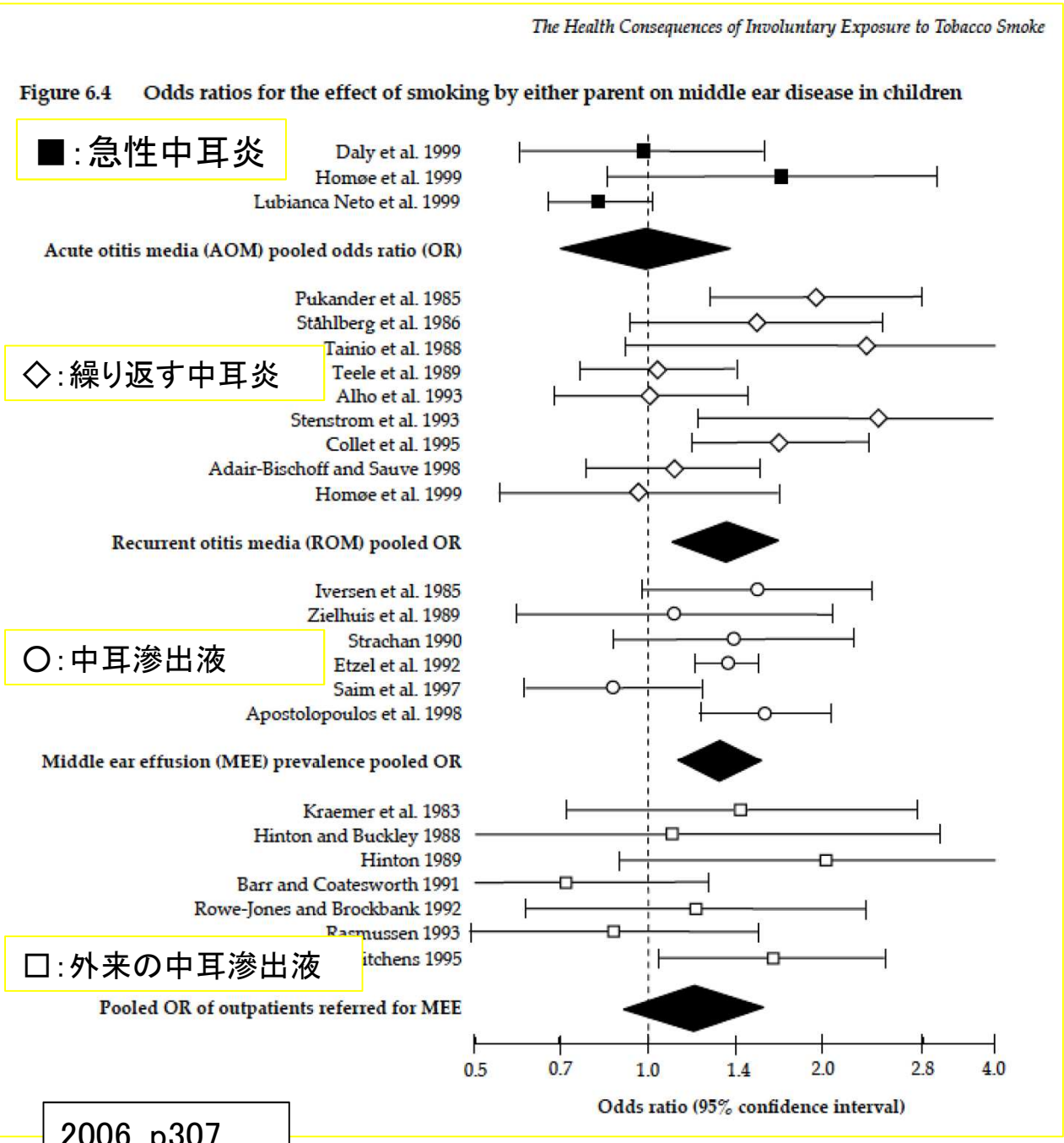
米国公衆衛生総監、2006年報告の主要な結論
2. 乳幼児突然死症候群、急性呼吸器症状、
耳鼻科疾患、重症化する喘息は受動喫煙と明らかな飲食店等のサービス産業が関係がある。
両親の喫煙は呼吸器症状の原因となり、かつ、小児の肺の発達障害の原因となる。

Conclusions

1. The evidence is sufficient to infer a causal relationship between parental smoking and middle ear disease in children, including acute and recurrent otitis media and chronic middle ear effusion.

2. The evidence is suggestive but not sufficient to infer a causal relationship between parental smoking and the natural history of middle ear effusion.

両親からの受動喫煙が小児期の
1. 急性、繰り返す中耳炎、滲出性中耳炎のリスクとなることとは明らか
2. 中耳疾患の発生の原因となることが示唆



2006, p307

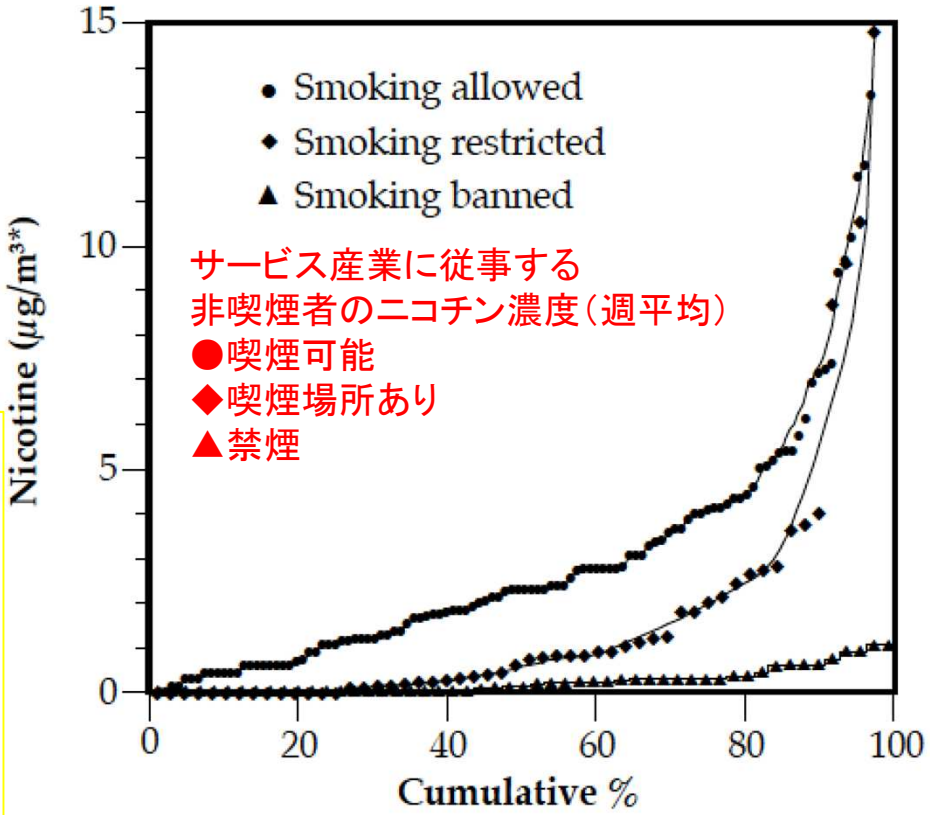
Major Conclusions

5. Many millions of Americans, both children and adults, are still exposed to secondhand smoke in their homes and workplaces despite substantial progress in tobacco control.

6. Eliminating smoking in indoor spaces fully protects nonsmokers from exposure to secondhand smoke. Separating smokers from nonsmokers, cleaning the air, and ventilating buildings cannot eliminate exposures of nonsmokers to secondhand smoke.

米国公衆衛生総監、2006年報告の主要な結論
5. 喫煙対策は進んだものの、多くのアメリカ人（成人、小児）が家庭で、職場で受動喫煙に曝露されている。
6. 受動喫煙の防止には、屋内完全禁煙が必要。区域分け、自然換気、強制換気などの、いわゆる「分煙」では受動喫煙を防止出来ない。

Figure 10.10 Cumulative frequency distributions of weekly average nicotine concentrations in nonsmokers' work areas in shops and other nonoffice settings



2006, p647

Conclusions

屋内全面禁煙の必要性に関する10の結論

＝受動喫煙防止には全面禁煙が必要　（2006年報告, p649）

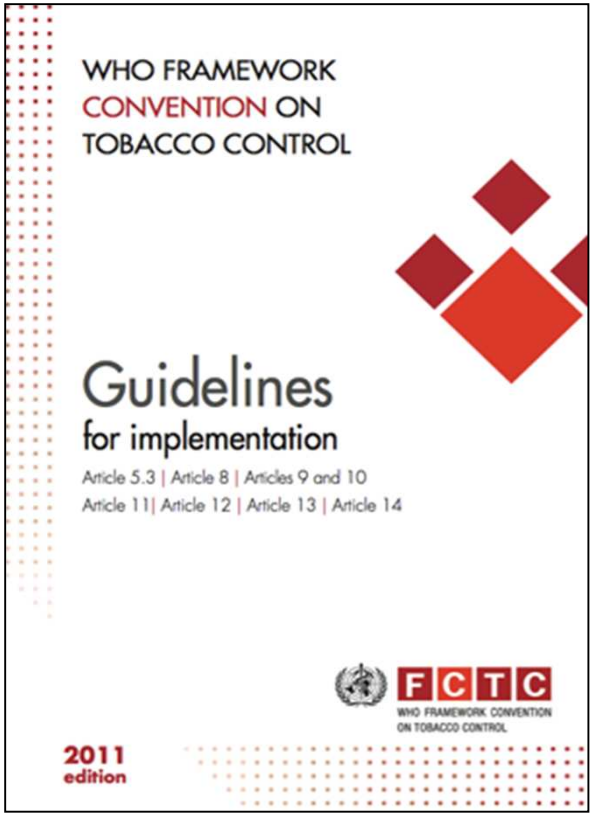
1. Workplace smoking restrictions are effective in reducing secondhand smoke exposure.
   職場の喫煙規制は受動喫煙軽減に有効
2. Workplace smoking restrictions lead to less smoking among covered workers.
   職場の喫煙規制は喫煙者の喫煙本数の減少に繋がる
3. Establishing smoke-free workplaces is the only effective way to ensure that secondhand smoke exposure does not occur in the workplace.
   屋内全面禁煙は受動喫煙完全防止の唯一の手段
4. The majority of workers in the United States are now covered by smoke-free policies.
   現時点で、米国の多くの職場が屋内全面禁煙
5. The extent to which workplaces are covered by smoke-free policies varies among worker groups, across states, and by sociodemographic factors. Workplaces related to the entertainment and hospitality industries have notably high potential for secondhand smoke exposure.
   サービス産業の全面禁煙化は遅れている
6. Evidence from peer-reviewed studies shows that smoke-free policies and regulations do not have an adverse economic impact on the hospitality industry.
   サービス産業を全面禁煙化しても営業収入は減らない
7. Evidence suggests that exposure to secondhand smoke varies by ethnicity and gender.
   人種と性により差が出ている
8. In the United States, the home is now becoming the predominant location for exposure of children and adults to secondhand smoke.
   米国では、家庭が受動喫煙の曝露の場となっている
9. Total bans on indoor smoking in hospitals, restaurants, bars, and offices substantially reduce secondhand smoke exposure, up to several orders of magnitude with incomplete compliance, and with full compliance, exposures are eliminated.
   完全禁煙と不完全な禁煙では数十万倍の差がある
10. Exposures of nonsmokers to secondhand smoke cannot be controlled by air cleaning or mechanical air exchange.
    空気清浄機や強制換気では受動喫煙を防止できない

FCTC第8条「受動喫煙からの保護」履行のためのガイドライン

"Guidelines for implementation Article Article 5.3, 8, 9, 10, 11, 12, 13, 14" (2011)

●喫煙室や空気清浄機の工学的な対策では受動喫煙を防止できない

Approaches other than 100% smoke free environments, including ventilation, air filtration and the use of designated smoking areas (whether with separate ventilation systems or not), have repeatedly been shown to be ineffective and there is conclusive evidence, scientific and otherwise, that engineering approaches do not protect against exposure to tobacco smoke.



●建物内の100%完全禁煙化以外に手段はない

FCTC発効から5年以内(2010年2月27日)に

建物内を100%完全禁煙とする

法律による屋内全面禁煙化を求めている。

諸外国では飲食店のなどサービス産業も含め屋内の全面禁煙化が進行。

Each Party should strive to provide universal protection within five years of the WHO FCTC's entry into force for that Party.

2007年、第2回締約国会議で採択
2011年、ガイドラインとして発表

http://www.who.int/fctc/protocol/guidelines/adopted/guidel_2011/en/index.html

43ヵ国(2012年時点)+ロシア(2014年)+韓国(2015年)は屋内全面禁煙

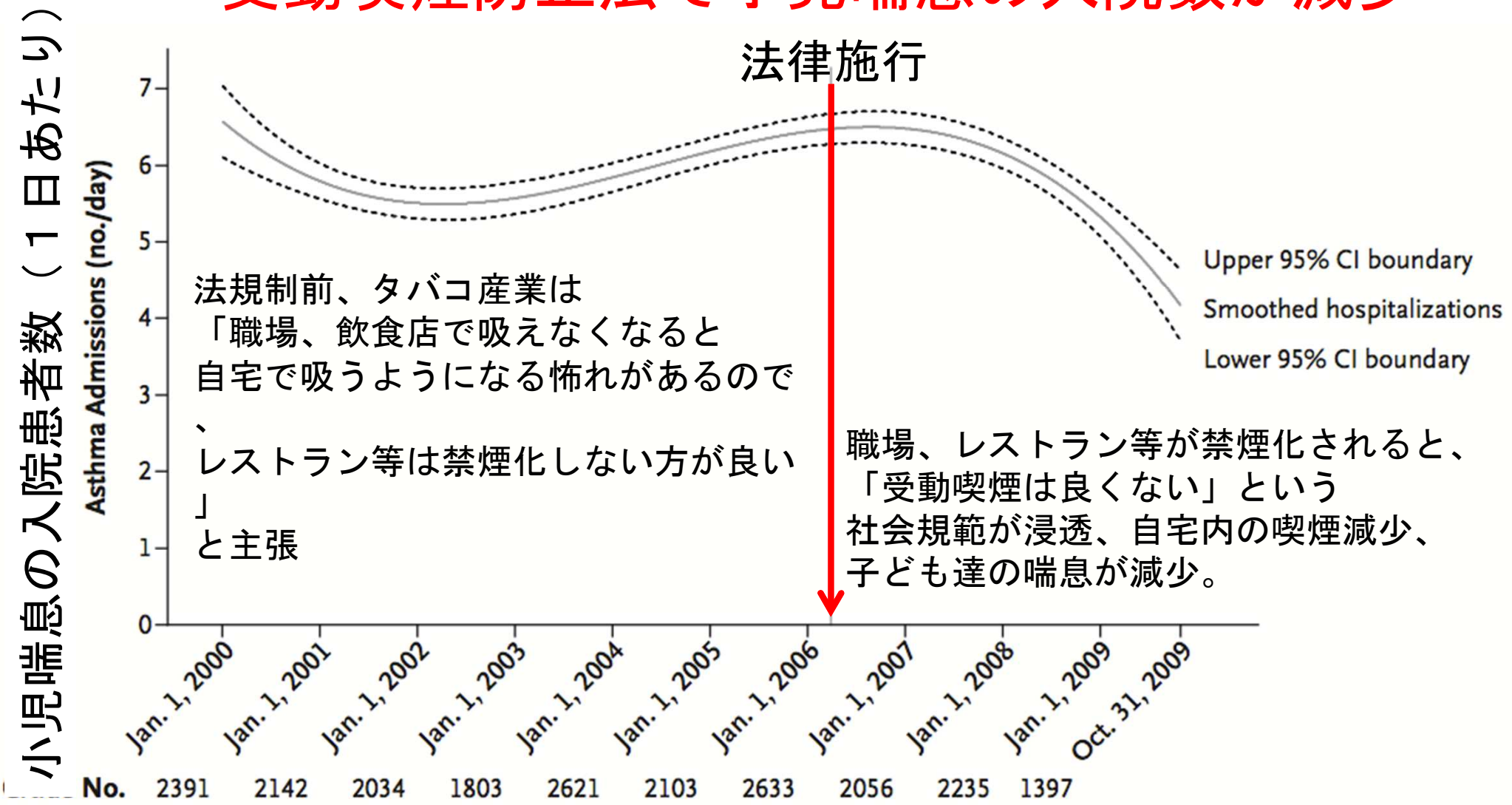
条約により、8つのカテゴリーがすべて全面禁煙の国
医療施設/大学以外の教育施設/大学/官公庁/一般の職場/公共交通機関
食事を主とするレストラン/飲物を主とするカフェ・ パブ、バー （居酒屋）

World Health Organization.
WHO Report on the Global Tobacco Epidemic, 2013より一部改変.

アメリカは50州のうち26州が屋内全面禁煙

注:
フランス、イタリア、フィンランドなどは喫煙室の設置を
容認しているが、設置基準が厳しすぎて、実質的には全面禁煙
台湾、香港も既に屋内全面禁煙法を実施

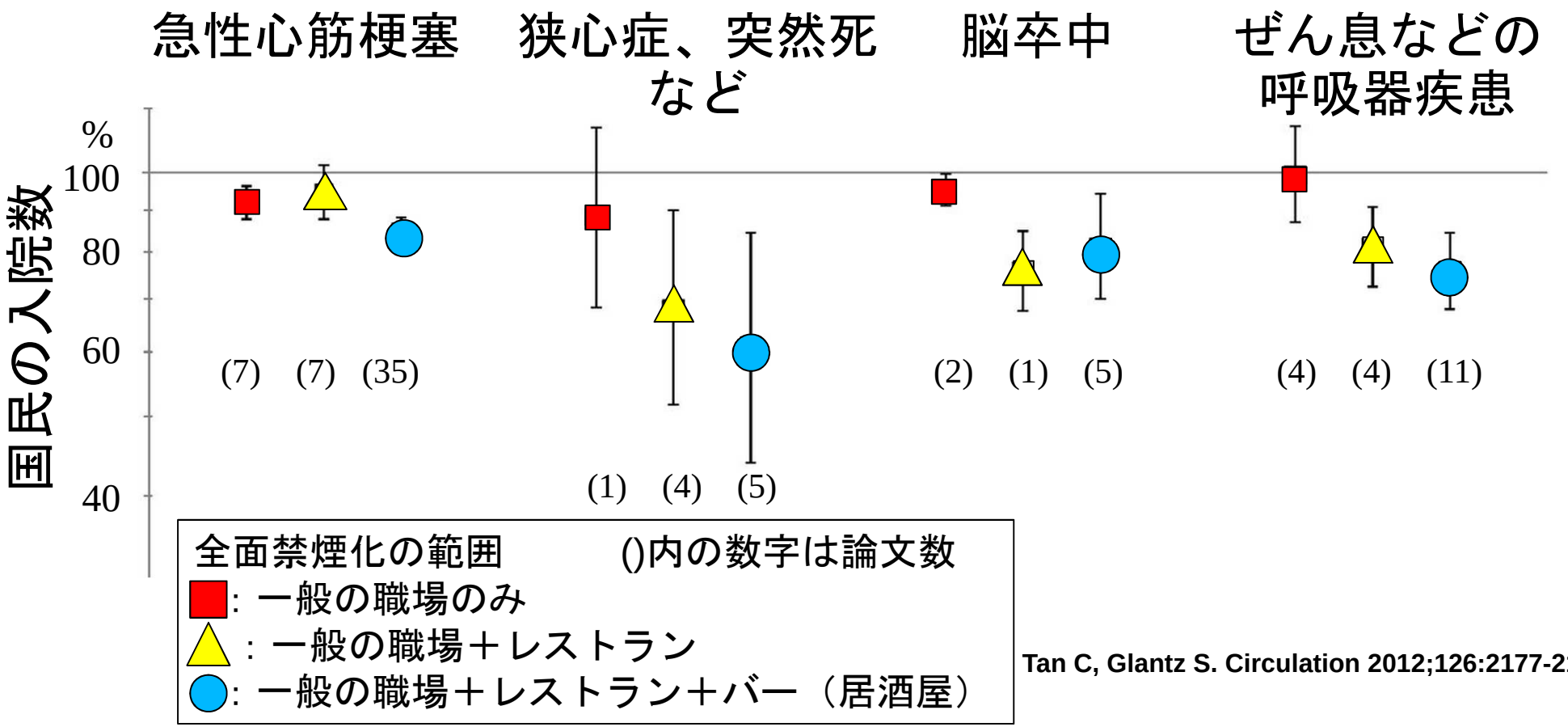
受動喫煙防止法で小児喘息の入院数が減少



Smoke-free Legislation and Hospitalizations for Childhood Asthma.
Mackay D, et al. N Engl J Med 2010;363:1139-45.

法律による屋内全面禁煙の効果（量・反応関係あり）

・法律による屋内全面禁煙化で国民の病気が減少
・禁煙化の範囲が広い（居酒屋・バーを含む）ほど減少が大きい



Tan C, Glantz S. Circulation 2012;126:2177-2183

Major Conclusions

(2006年報告, 11頁）

3. Exposure of adults to secondhand smoke has immediate adverse effects on the cardiovascular system and causes coronary heart disease and lung cancer.

2006年報告書の主要な結論

3.　受動喫煙への曝露は、心血管システム、冠動脈疾患、肺がんに直ちに悪影響を及ぼす

この結論３が、すべての職場、レストラン、バー（居酒屋）を全面禁煙にする法律が施行された国・州では、心筋梗塞など喫煙関連疾患の入院数が減少した、という研究により立証されたことになる。

分煙（喫煙室）の問題点

１．受動喫煙を防止できない

・ドアの開閉に伴う漏出
・喫煙者の退出に伴う漏出
・喫煙者の肺にたまったタバコ煙の持ち出し
・喫煙室の掃除業者の受動喫煙

２．サービス産業従業員の受動喫煙

・レストラン・居酒屋では、毎日数時間の職業的受動喫煙
・喫煙室・喫煙店内のPM2.5濃度は北京の数倍

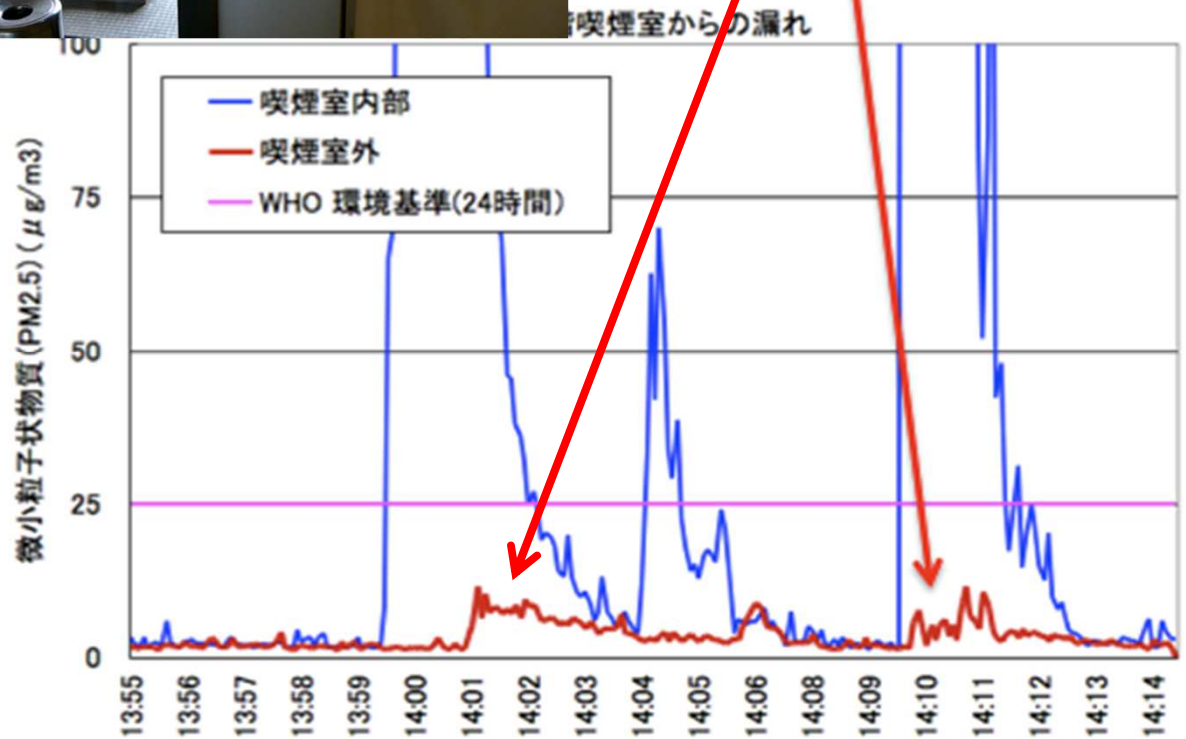
３．オリンピック・パラリンピック大会との関連

「一定の要件を満たす喫煙室」: 換気扇3台でも漏れ

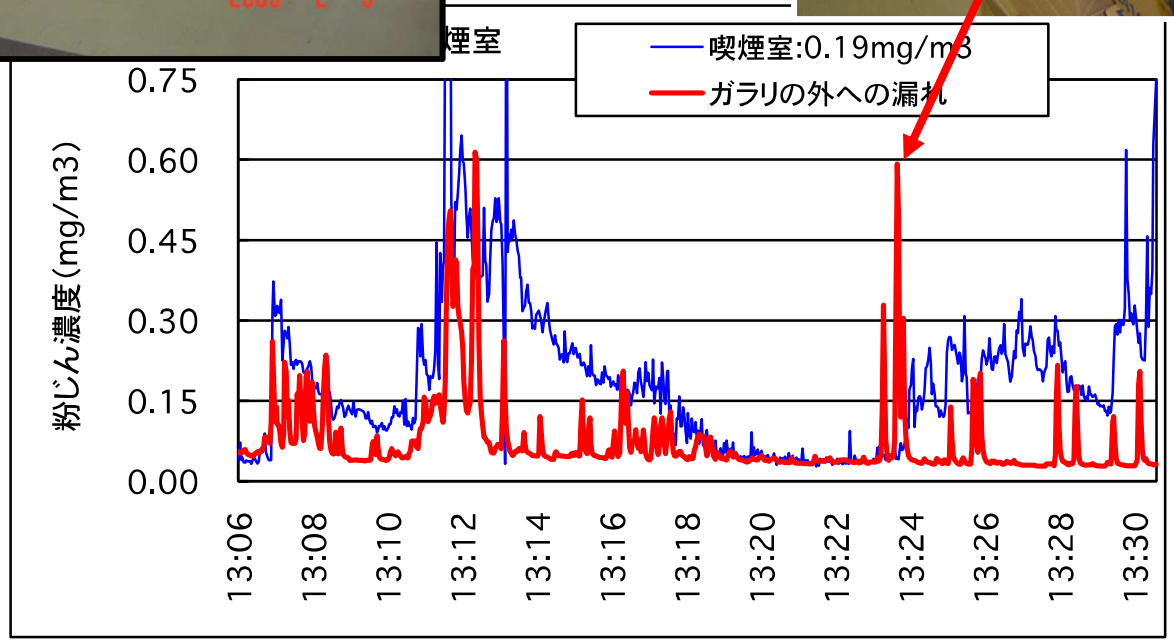
- 漏れの原因

  ドアのフイゴ作用

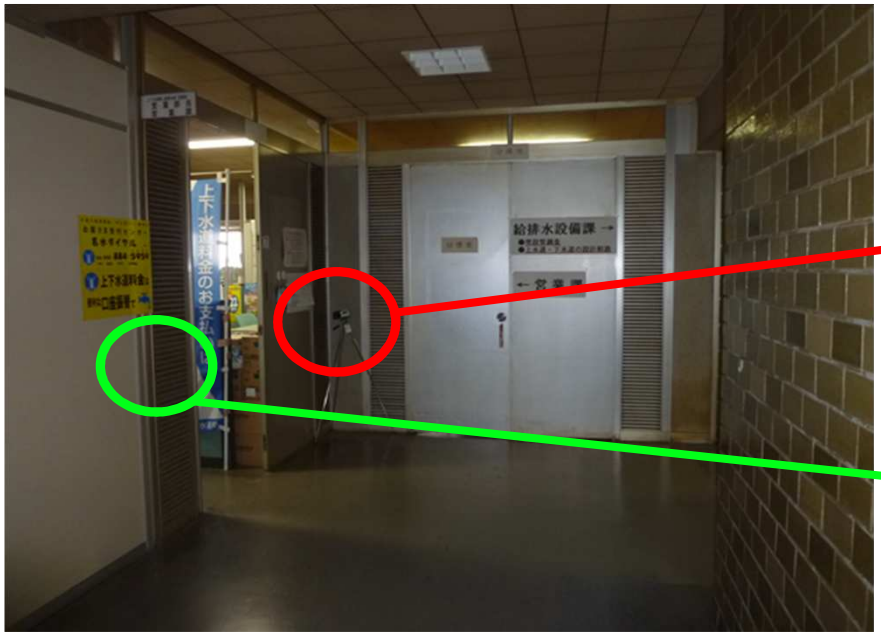
  ドアの開閉により、
  空気の取入口や
  パネルと天井・床との
  隙間から押し出される

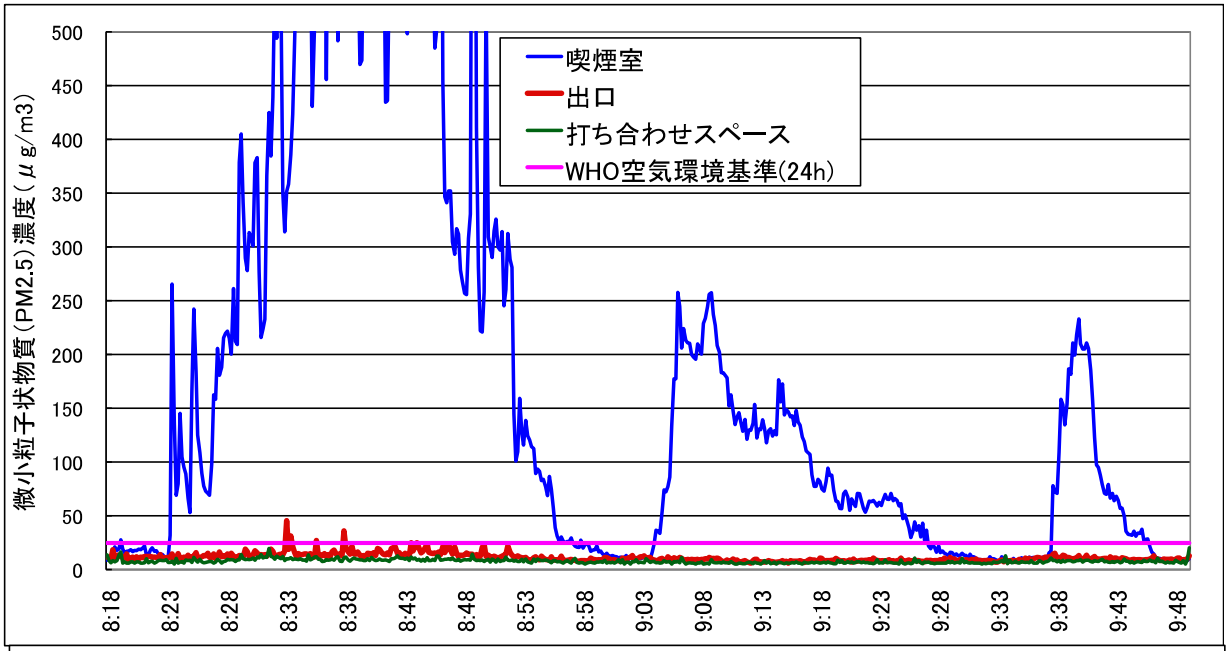


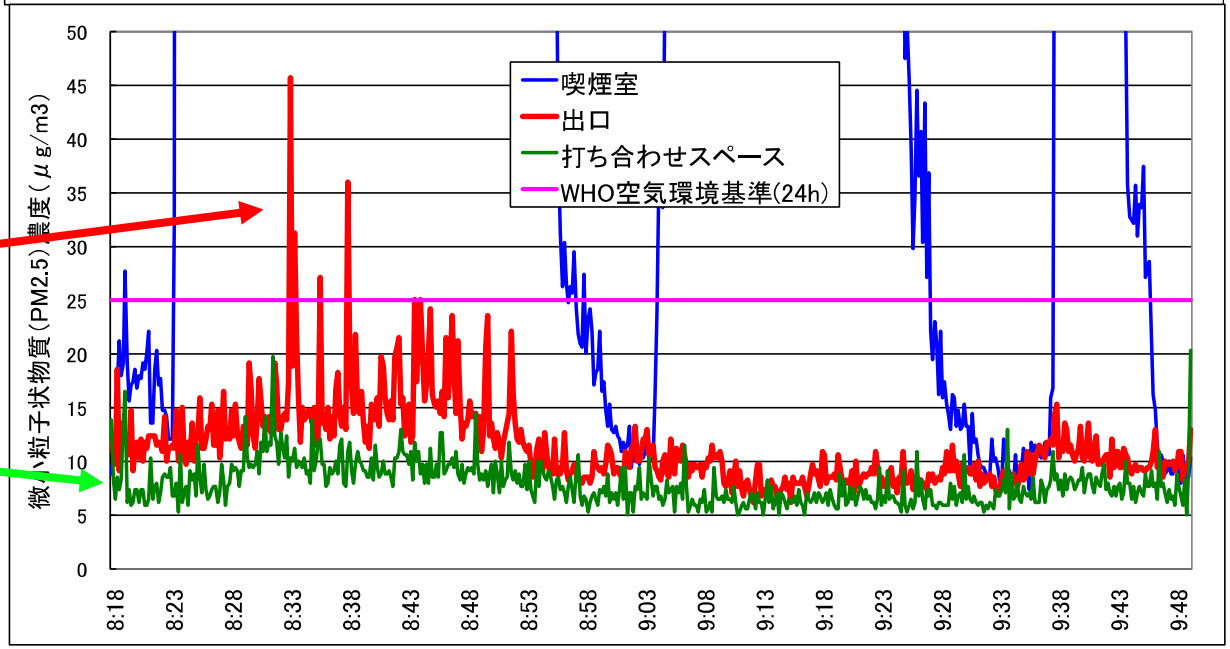
某区役所:ドアのフイゴ作用:ドアを押し入れる際に、瞬間的に喫煙室が陽圧になり、煙が空気取入口から漏れる



N市役所の喫煙室：大型換気扇、紙製の弁（ダンパー）でも漏れ







N市役所の喫煙室：大型換気扇、紙製の弁（ダンパー）でも漏れ

ドアを押し込むときに、煙が漏れないように、空気取入口（ガラリ）に、紙製の弁（ダンパー）を設置しても漏れあり

→対策が無ければ、漏出はさらに増大

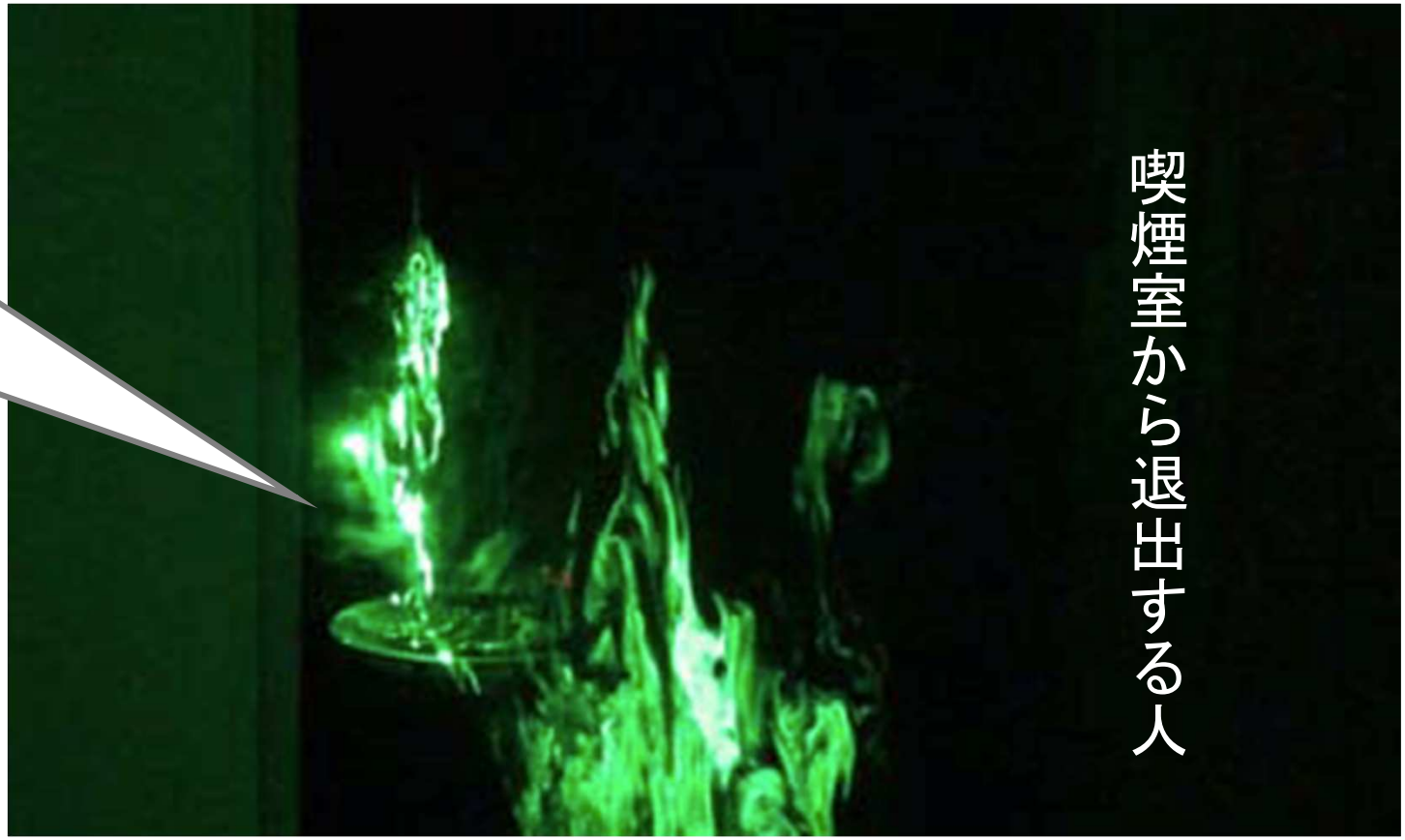
ドア閉（内部は陰圧）時には、
空気取入口から空気が流入

ドア閉（内部は陰圧）時には、
空気取入口から空気が流入

喫煙室から出てくる人の身体の後にできる渦に巻き込まれて タバコ煙が持ち出される＝「完全分煙」は不可能

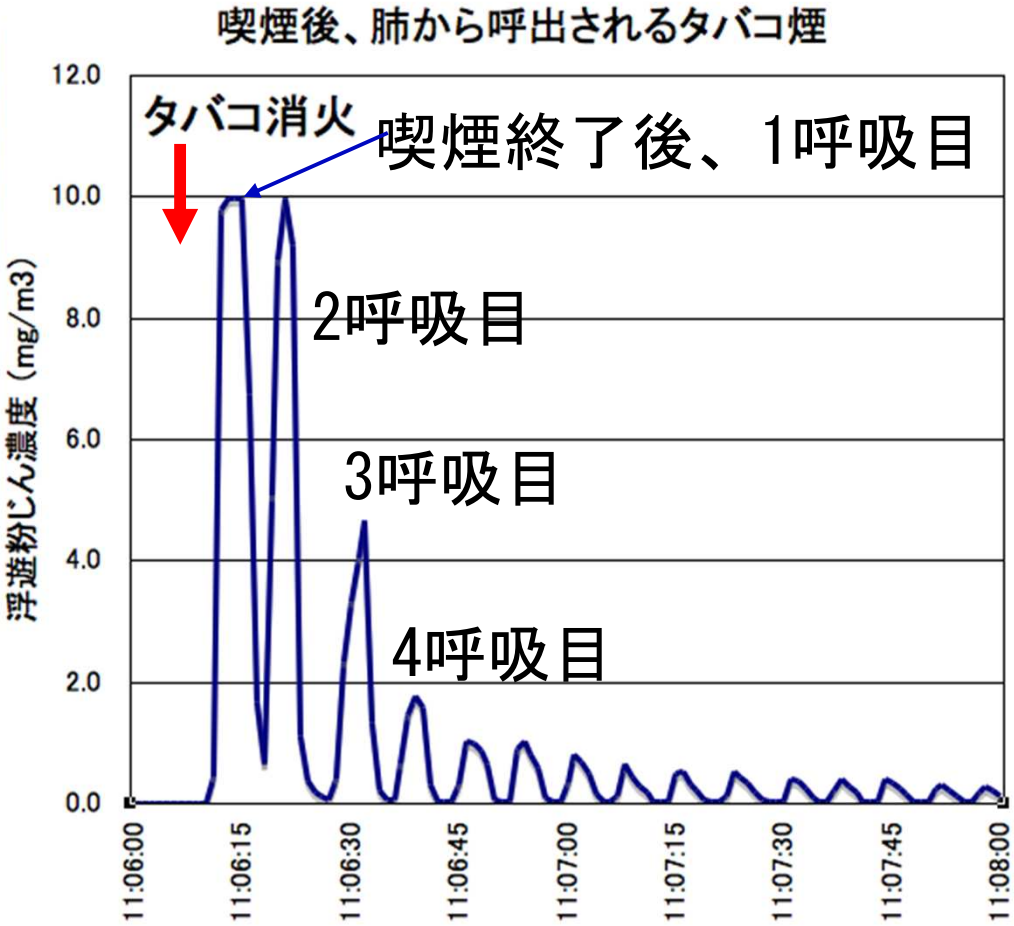
漏れの原因

「一定の要件を満たす喫煙室」の基準＝
開口部分の
風速0.2 m/sのよりも、
喫煙者の歩く速度
0.7m/sの方が速い。



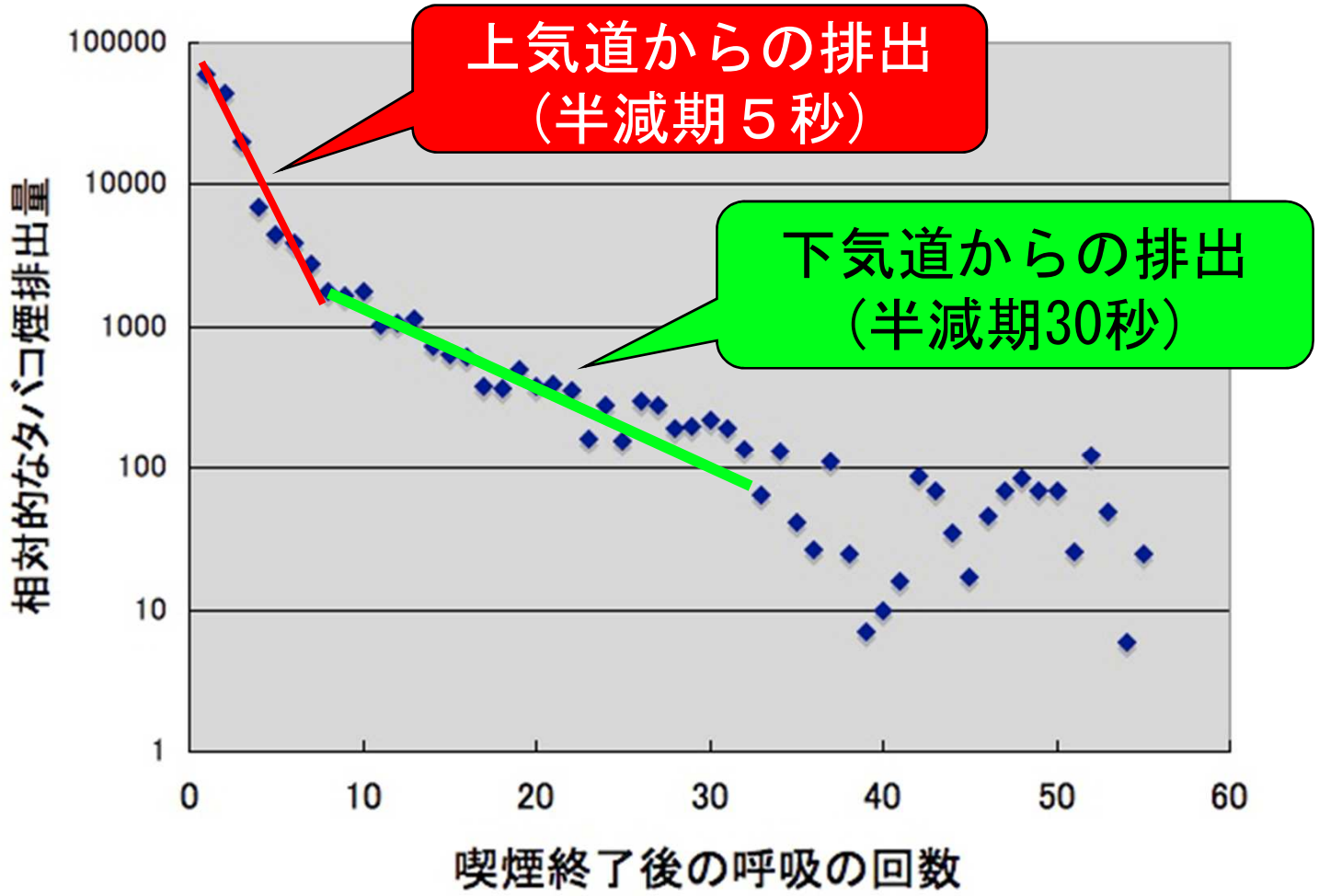
退出する喫煙者の身体の後ろにできる
空気の渦に巻き込まれて煙が持ち出される。

喫煙室退出後、肺内のタバコ煙を禁煙区域で吐出
→受動喫煙の原因



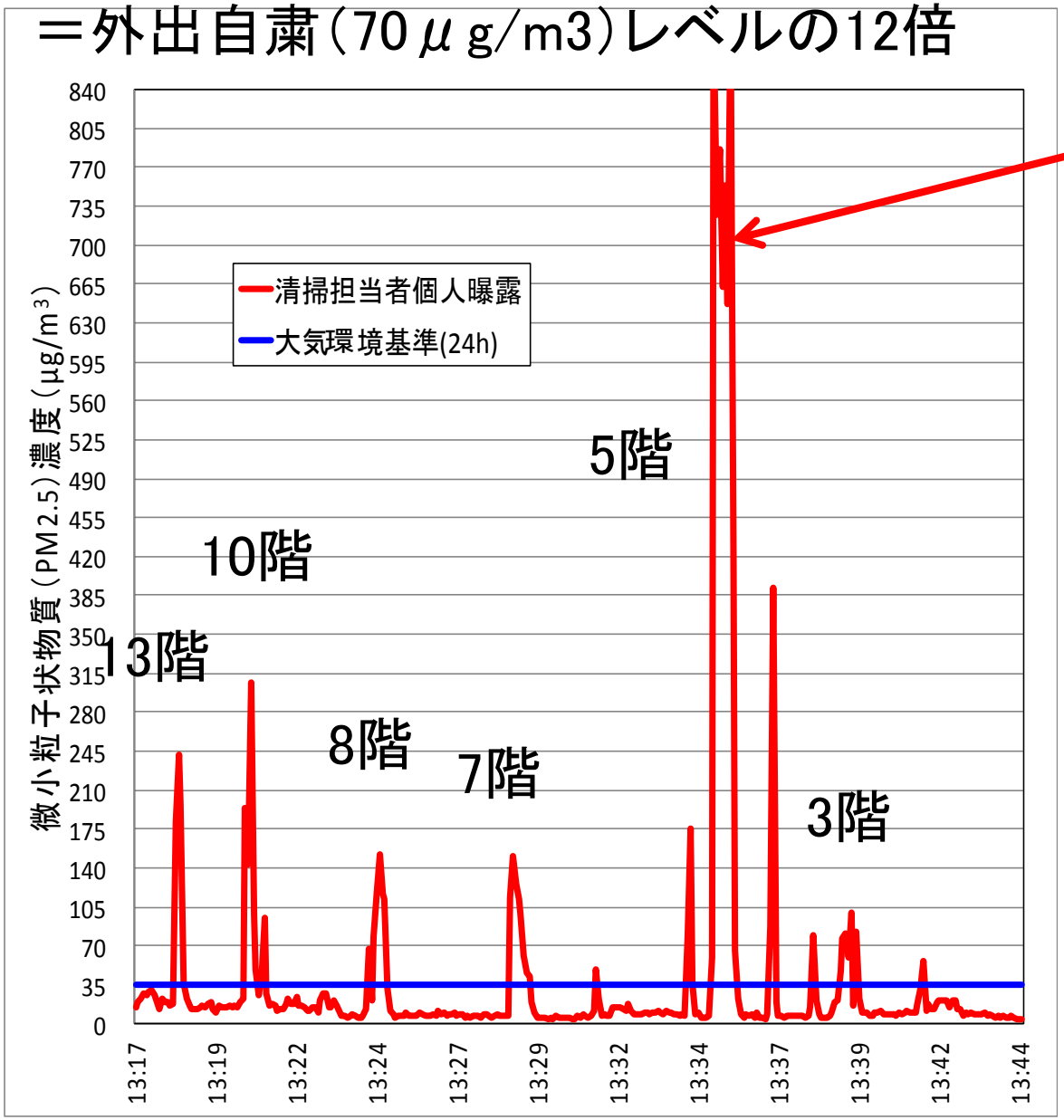
喫煙後、約40回分の呼気（約200秒間）に粒子状物質を検出

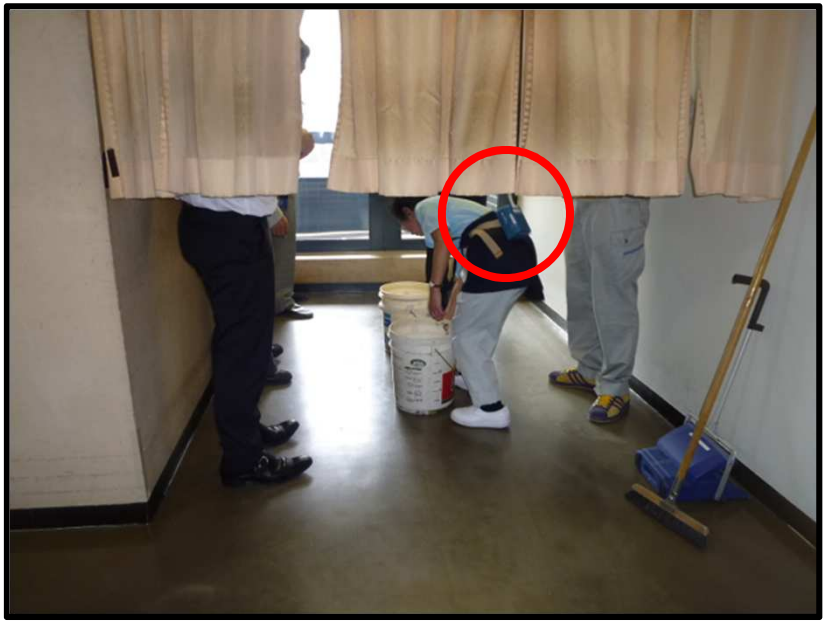
喫煙終了後の呼気に含まれるタバコ煙
40呼吸＝200秒は呼気に煙粒子が含まれる



さらに、ガス状成分（三次喫煙）は洋服や口臭から数時間発生

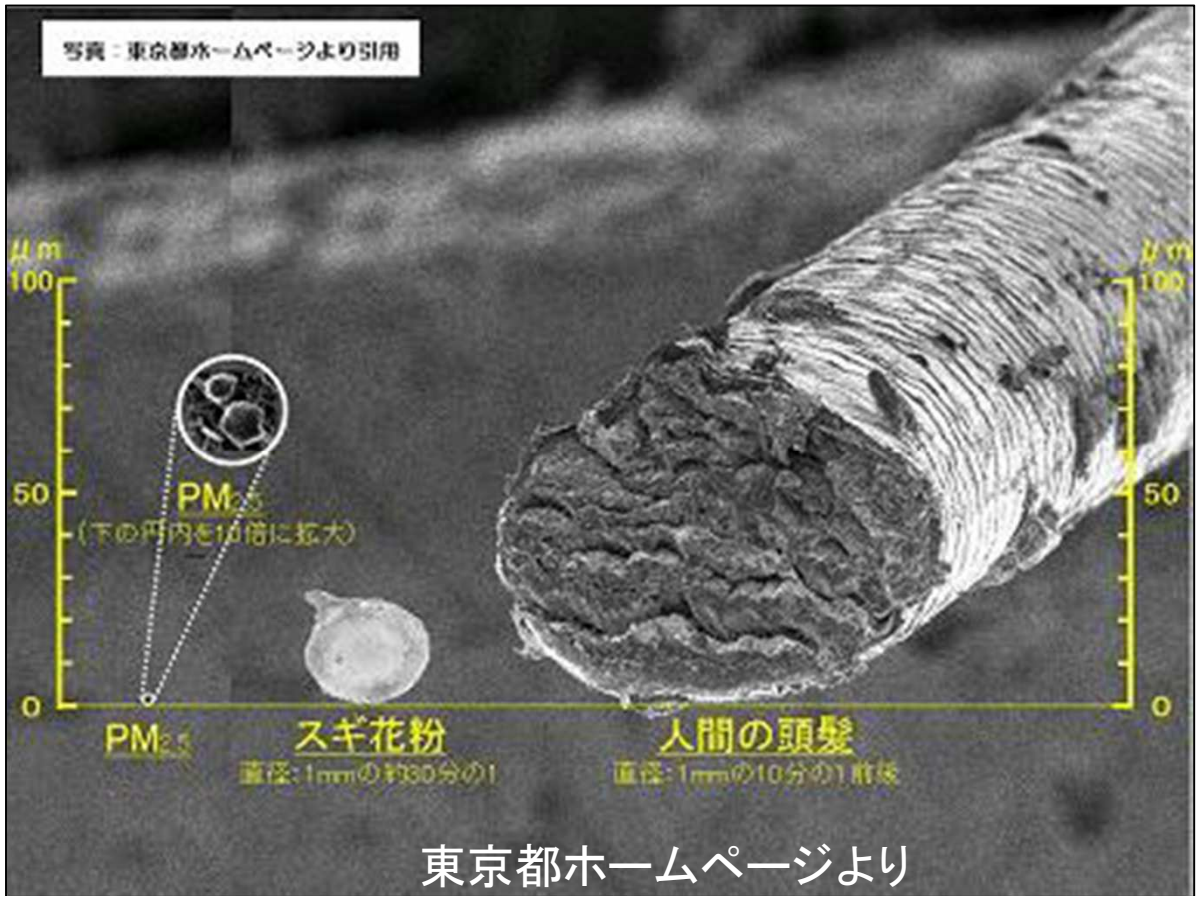
喫煙室の掃除担当業者の職業的な受動喫煙
PM2.5最高値845μg/m3＝大気環境基準(24h)の24倍
＝外出自粛(70μg/m3)レベルの12倍





環境省 微小粒子状物質（$PM_{2.5}$）に関する基準値　2009年9月9日告示

- １年平均値が 15μg/m³ 以下
- １日平均値が 35μg/m³ 以下
- 外出を自粛するレベル：70μg/m³ 以下(2013年）

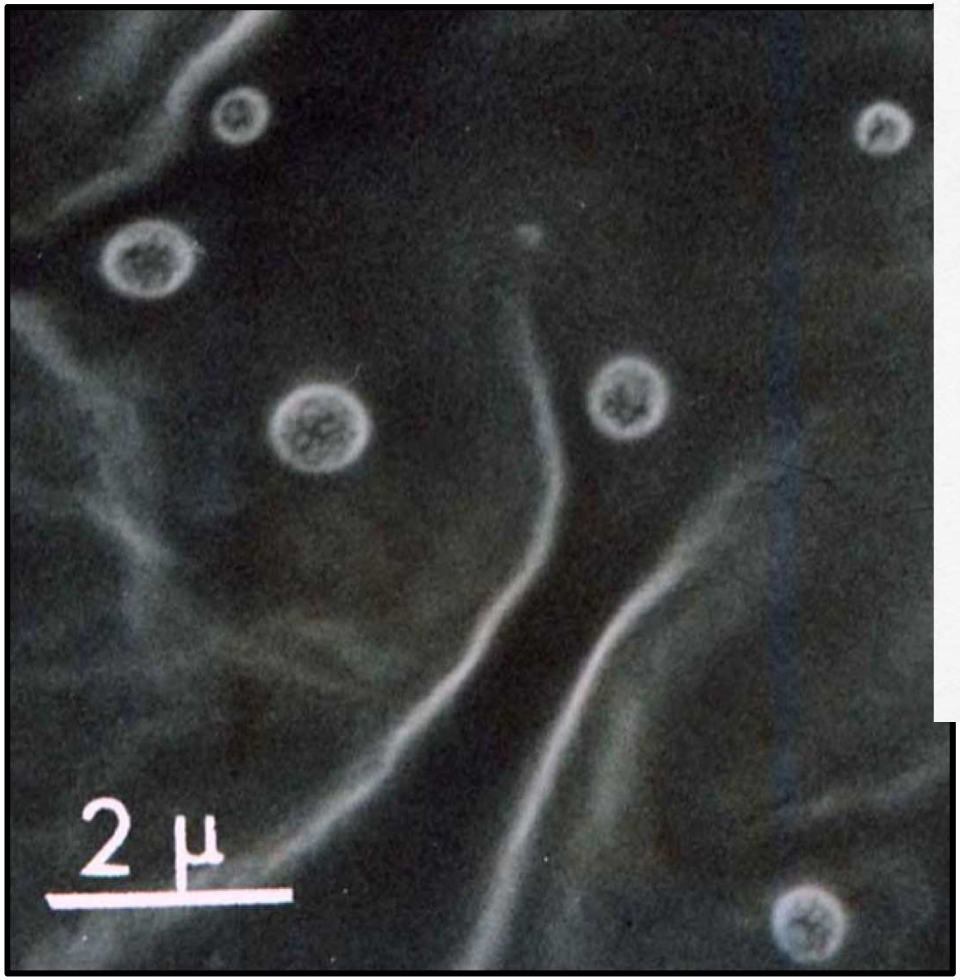


東京都ホームページより

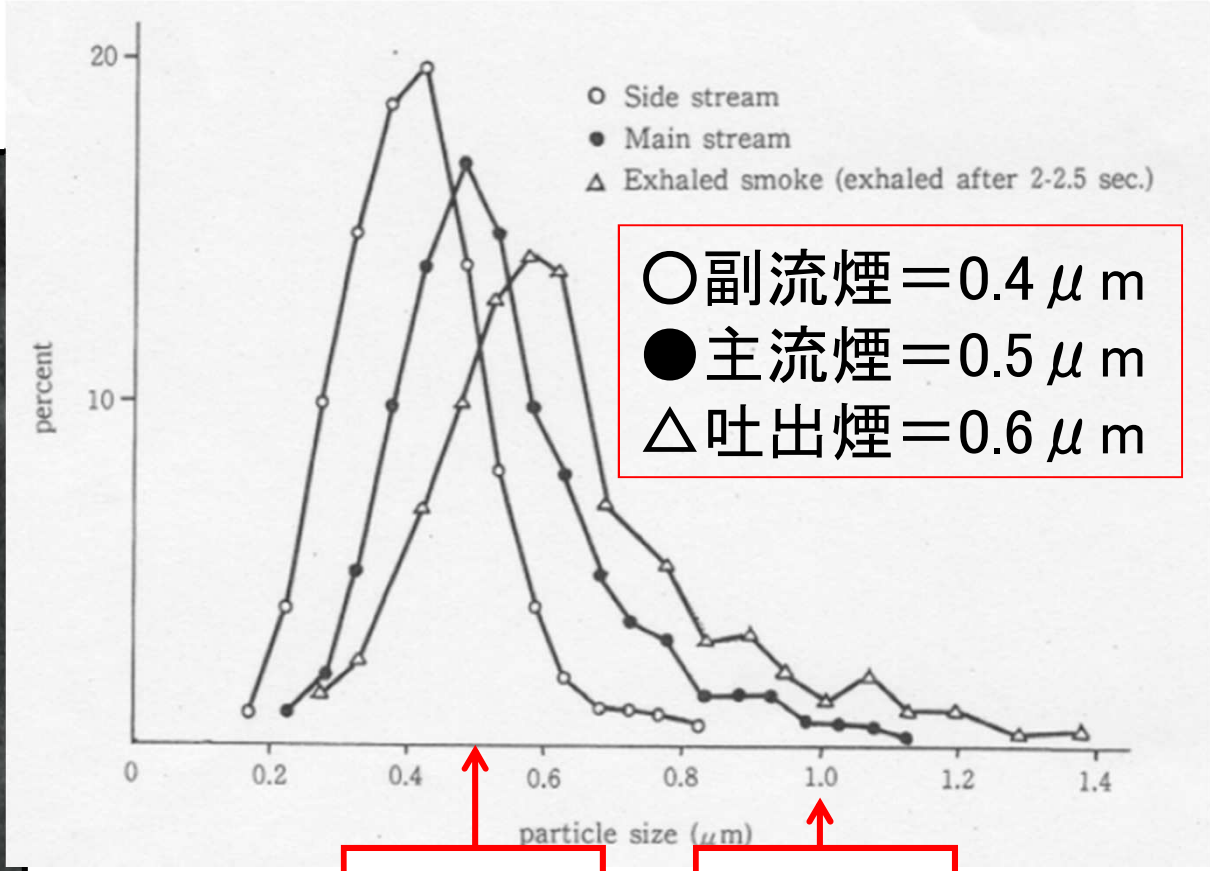
微小粒子状物質は肺の最深部まで吸入＝表面まで黒くなる

タバコ煙の粒子径は1マイクロメーター以下⇒PM 0.5

副流煙の電子顕微鏡写真



「タバコ煙粒子の捕集、観察と気道内での観察」
東 敏昭(産業医大学長), 他.
日本公衆衛生雑誌, 32, 17-23, 1985

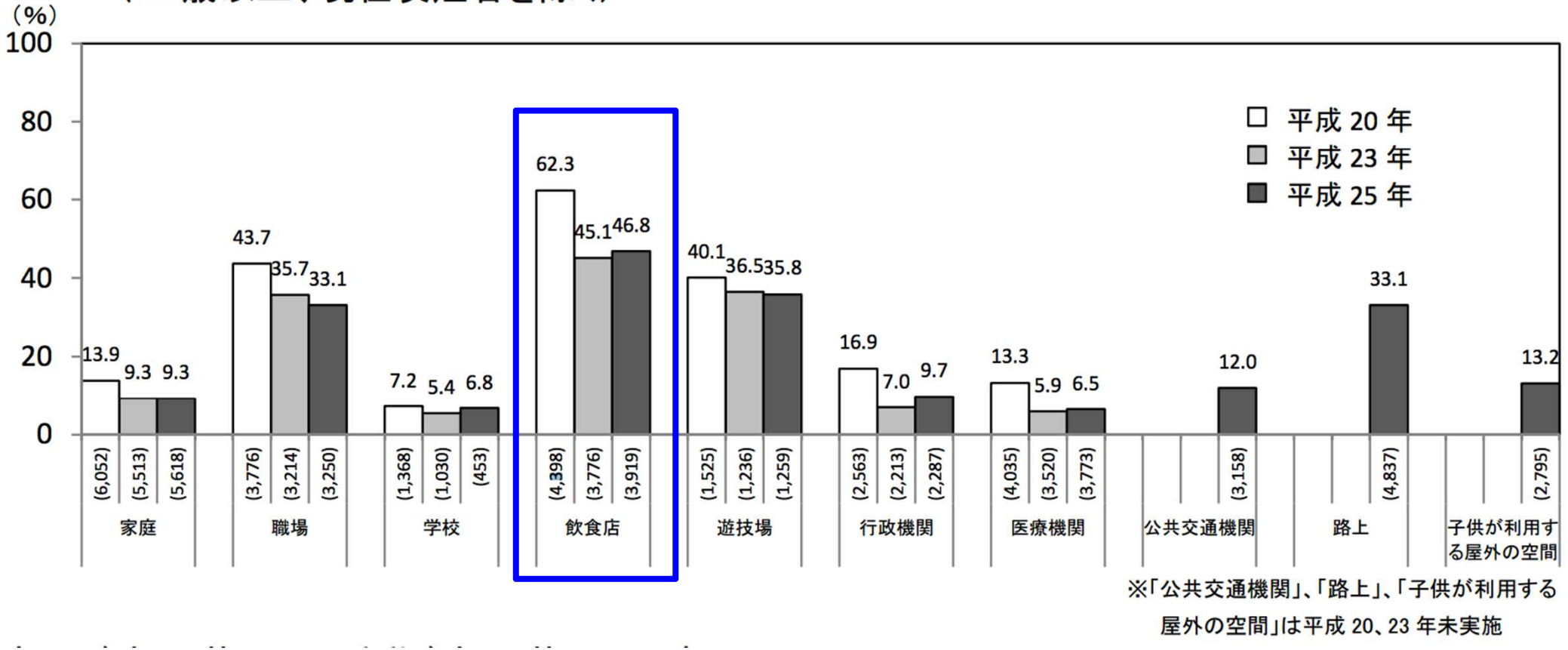


肺の最深部まで吸入、異物反応
⇒肺の炎症
⇒血流に乗って全身の血管の炎症
⇒動脈硬化

受動喫煙の曝露を受ける場所の1位は飲食店46%

厚労省、平成25年度 国民健康・栄養調査

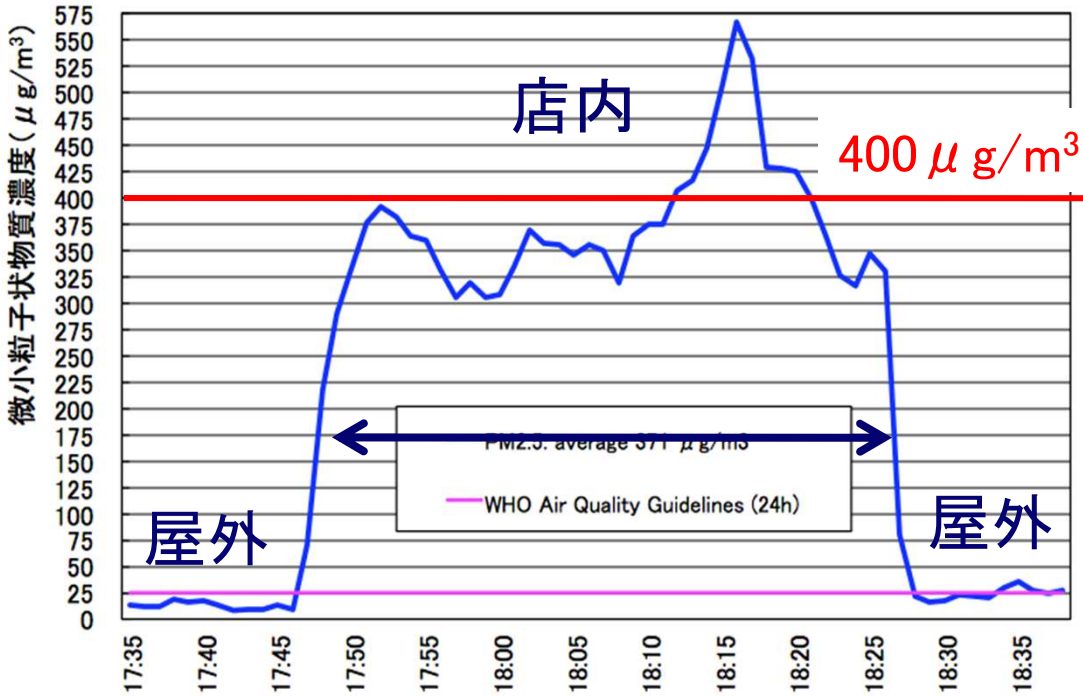
図 38　自分以外の人が吸っていたたばこの煙を吸う機会(受動喫煙)を有する者の割合
（20 歳以上、現在喫煙者を除く）



※「公共交通機関」、「路上」、「子供が利用する屋外の空間」は平成 20、23 年未実施

飲食店のPM2.5(⇒PM0.5)は北京並み

5 in this study measured in Cafe



博多の大通りに面した喫茶店

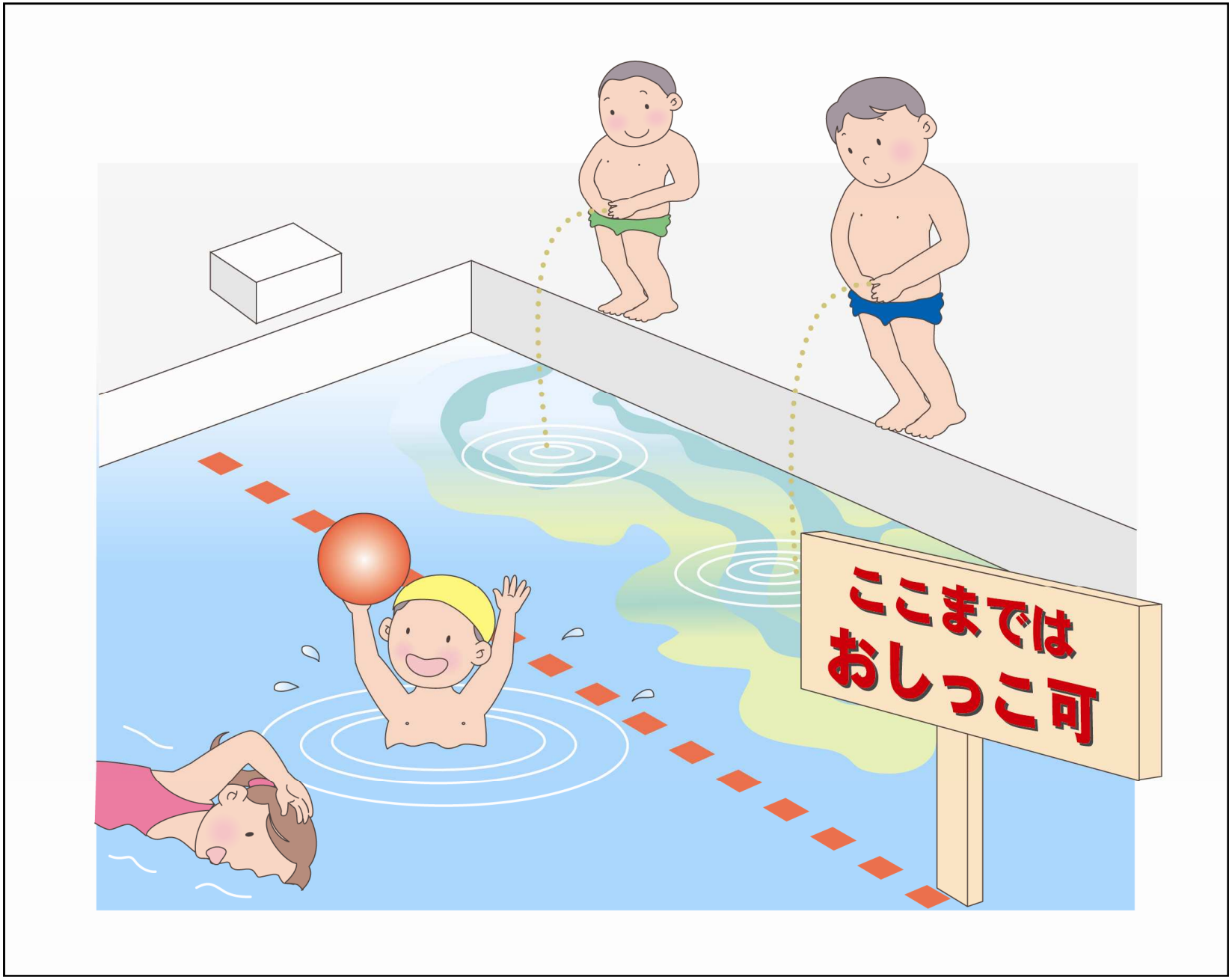
北京市内のPM2.5
2013年1月1～31日

米国大使館測定データ 国立環境研究所提供)

分煙ではダメな理由：ファミレスなどの禁煙・喫煙区域分け

喫煙席の煙はエアコンで攪拌され、数分後には禁煙区域に拡散

区域を分けただけの対策では効果なし



分煙では、従業員の職業的な
受動喫煙を防止出来ない。
個人曝露をPM2.5で評価

喫煙席の粉じん計