

MORBIDITY AND MORTALITY WEEKLY REPORT

January 12, 1979 / Vol. 28 / No. 1

Current Trends Highlights of the Surgeon General's Report on Smoking and Health

Epidemiologic Notes and Reports 12 Trichinosis Associated with Bear Meat – Alaska, California

Current Trends

Highlights of the Surgeon General's Report on Smoking and Health

In the 15 years that have elapsed since the original Report of the Advisory Committee on Smoking and Health to the Surgeon General, there have been many new scientific studies on the relationship between tobacco consumption and health. There are now more than 30,000 articles in the world literature on this subject. The new volume on smoking and health includes a detailed review and update of the relationships between smoking and health written by various agencies within the U.S. Department of Health, Education, and Welfare, and coordinated and assembled by the Office on Smoking and Health. This issue of the MMWR offers summaries of selected chapters in the new 1979 Surgeon General's Report. The complete edition of the report, when available in Spring 1979, may be requested from the Office on Smoking and Health, Park Bldg., Rm. 158, Rockville, Md. 20857.

Smoke and Physiology: The lighted cigarette generates about 4,000 compounds which can be separated into gas and particulate phases. Carbon monoxide (the gas phase), nicotine, and tar (the particulate phase) are the most likely contributors to the health hazards of smoking. Many other compounds are probable or suspected contributors.

It is generally accepted that nicotine is principally responsible for cigarette smokers' physiologic responses through release of catecholamines, epinephrine, and norepinephrine. Short-term physiologic responses attributable to nicotine and/or catecholamines include increased heart rate, blood pressure, cardiac output, stroke volume, oxygen consumption, coronary blood flow, and arrhythmias. Bronchoconstriction and related pulmonary manifestations, increased mobilization and utilization of free fatty acids, and hyperglycemic effects also occur.

Smoking enhances the breakdown of drugs by increasing the amount and the activity of hepatic microsomal enzymes. Some therapeutic agents whose clinical effects are modified by smoking are phenacetin, antipyrine, theophylline, caffeine, imipramine, pentazocine, and vitamin C.

Tobacco smoking also affects the values of a number of clinical laboratory tests in humans. Such changes include increases in the number of leukocytes, the red cell mass, the levels of hemoglobin and carboxyhemoglobin, and the hematocrit. Cigarette smoking decreases the serum levels of creatinine, albumin, globulin (in females) and uric acid (in males). These levels revert to normal after cessation of smoking.

Mortality: Data from the 7 original prospective studies, from long-term follow-up of 3 of these studies, and from prospective studies in Japan and Sweden yield quantitative results substantially identical to earlier conclusions. Overall, current management is substantially identical to earlier conclusions.

CENTER FOR DISEASE CONTROL U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELATEANEANEANEANEANE

Smoking - Continued

have an approximately 70% greater chance of dying from disease than nonsmokers (i.e., smokers have an overall mortality ratio of 1.7; see Table 1). Specific mortality ratios are directly proportional to the amount smoked and to the years of cigarette smoking, and are higher both for those who initiated their smoking at younger ages and for those who inhale. Although mortality ratios for smokers are highest at the younger ages and decline with increasing age, the actual number of excess deaths attributable to cigarette smoking increases with age.

Former cigarette smokers experience declining mortality ratios as the years of not smoking increase. After 15 years, mortality ratios for former smokers approach those for individuals who never smoked. Mortality ratios for any given age for former smokers are directly proportional to the amount smoked before cessation and inversely related to the age of smoking initiation. Regardless of these factors, cessation does diminish an individual's risk, provided s/he is not ill at the time of cessation. Overall mortality ratios for female smokers are somewhat less than those for male smokers. However, subsets of females with smoking characteristics similar to those of men experience mortality rates similar to those of male smokers.

Calculations from prospective study data have indicated that life expectancy at any given age is significantly shortened by cigarette smoking. However, mortality ratios are decreased in smokers who use cigarettes with a decreased tar and nicotine content. From the detailed data of 2 prospective studies, excess mortality is found to be greatest for the 45- to 54-year age group among women and men, indicating that smoking causes premature mortality. Coronary heart disease is the chief contributor to the excess mortality among cigarette smokers, followed by lung cancer and chronic obstructive lung disease.

individuals who limit their smoking to only pipes and cigars have overall mortality rates that are slightly higher than nonsmokers, but lower than cigarette smokers.

Morbidity: Following the 1964 Report to the Surgeon General, the National Center for Health Statistics began collecting information on smoking, on the basis of probability samples of the population, as part of the National Health Interview Survey. These data have proven valuable in assessing the relationships between tobacco use and health indicators including illness and disability.

In general, male and female current cigarette smokers report more acute and chronic conditions than persons who never smoked. The age-adjusted incidence of acute conditions for males who had ever smoked was 14% higher, and for females 21% higher, than for those who had never smoked cigarettes. Non-physician-dependent indicators of morbidity include measures such as work-days lost, days in bed, and limitation of activity resulting from chronic disease. As derived from 1974 survey data, there are more than 81,000,000 excess work days lost and more than 145,000,000 excess days of bed disability per year due to smoking in the U.S. population. Current and former smokers also reported more hospitalizations in the year prior to interview than did non-smokers. Data on disability and illness often show continued higher risk among former smokers, although these data are not conclusive.

Cardiovascular Disease: Systematic observations on the association between cardir ovascular disease and smoking have been made on considerably more than 1 million individuals (mostly men) in the United States, and have involved many millions of person years of experience. Sample sizes are now extensive in both retrospective and prospective studies, and it can be concluded that smoking is causally related to coronary heart disease for both men and women in the United States.

The data show that smoking is 1 of 3 major, independent risk factors for heart attack. The effect is dose-related (including a lower risk with low tar and nicotine cigarettes).

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	Α	В		С	D	Е	F		3	п
		Age in 1	years					M	F	
and provide the second s		45-64	65-79							
All cancers		2.14	1.76	2.21	1.62		1.97	70	4 5	15 9
lung and bronchus	14.0	7.84	11.59	12.14	3.64	14.2	10.73	7.0	4.5	15.5
larynx		6.09	8.99	9.95	13.59		13.10			
buccal cavity	13.0	((4.09	7.04	3.9	2.80			1.0
pharynx		(9.90	2.93	12.54	2.81					
esophagus	4.7	4.17	1.74	6.17	2.57	3.3	6.60			0.7
bladder and other	2.1	2.20	2.96	2.15	0.98	1.3	2.40	1.8	1.6	6.0
pancreas	1.6	2.69	2.17	1.84	1.83	2.1		3.1	2.5	
kidney		1.42	1.57	1.45	1.11	1.4	1.50	• •	<u>.</u>	00
stomach		1.42	1.26	1.60	1.51	1.9	2.30	0.9	2.5	Q .0
Intestines				1.27	1.27	1.4	0.50			0.9
rectum	27	{1.01	{ 1,17	0.98	0.91	0.6	0.80			1.0
	2.1	3		4.75			1 57			
MI cardiovascular		1.90	1.31	1.75	1.06	16	1.57	1.7	1.3	2.0
coronary heart disease	1.6	2.03	1.36	1.74	1 1/	0.0	1 30	1.0	1.1	1.8
cerebrovascular lesions	1.3	1.38	1.00	5.24	1.17	1.8		1.6		
byporte	6.6	2.62	4.92	1.67	2.51	1.6	1.20	1.3	1.4	1.0
Operal enteriosalesesia	1.4	1.40	1.42	1.86		3.3	2.00	2.0	2.0	
	1.4			1.00						
(non respiratory diseases							2.85			
(inclineoplastic)				10.08			2.30	1.6	2.2	4.3
emphysema and/or bronchitis	24.1	6 55	11 41	14 17		7.7	7			
bronchitic		0.55	11.41	4.49		11.3	3			
respiratory tuberculosis	5.0	h		2.12	1.27					
asthma	0.0	•		3.47						
influenza and pneumonia	1.4	1.86	1.72	1.87		1.4	4 2.60)		2.4
Certain other conditions										
Stomach ulcar		4.06	4 13	3 4.13	(
duodenal ulcer	{ 2.5	⁵ 2.86	1.50	2.98	{ 2.06	6.	9 2.16	5		0.5
					1 2 5	; ว	3 1.9	32.	4 0.8	3 4.0
Cirrhosis	3.	0 2.06	1.9	/ <u>3.</u> 33	1.35	, 2.			2.11	
Par Kinsonism	0.	4		0.20			0 17	n 1	4 1	2 1.78
All causes	16	4 1 88	14	3 1.84	1.22	2 1.5	2 1.7	<u> </u>	- I.	

TABLE 1. Mortality ratios of current cigarette-only smokers, by cause of death, in 8 Prospective epidemiologic studies*

Study A: Doll R, Peto R: Mortality in relation to smoking: 20 years' observation on male British doctors. Br Med J 2:1525-1536, 1976; Study B: Hammond EC: Smoking in relation to the death rates of one million men and women, in Haenszel W (ed): Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. U.S. Public Health Service, National Cancer Institute Monograph 19, 1966, pp 127-204; Study C: Kahn HA: The Dorn study of smoking and mortality among U.S. veterans: Report on 8½ years of observation, in Haenszel, Ibid, pp 1-125; Study D: Hirayama T: Smoking in relation to the death rates of 265,118 men and women in Japan. A report on 5 years of follow-up. Presented at the American Cancer Society's 14th Science Writers' Seminar, Clearwater Beach, Florida, 24-29 Mar 1972; Study E: Best EWR: A Canadian study of smoking and health. Ottawa, Dept of National Health and Welfare, 1966; Study F: Hammond EC, Horn D: Smoking and death rates-Report on forty-four months of follow-up on 187,783 men. I. Total mortality. II. Death rates by cause. JAMA 166:1159-1172, 1294-1308, 1958; Study G: Cederlof R, Friberg L, Hrubec Z, et al: The relationship of smoking and some social covariables to mortality and cancer morbidity. A ten-year follow-up in a probability sample of 55,000 Swedish subjects age 18-69. Part 1/2, Sweden. Karolinska Institute, 1975; Study H: Weir JM, Dunn JE, Jr: Smoking and mortality: A prospective study. Cancer 25:105-112, 1970 068683

Smoking - Continued

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synergistic with other risk factors for heart attack, and more strongly associated with younger ages. Smoking also increases the possibility of recurrence of myocardial infarction. Cessation of smoking reduces the risk of mortality from coronary heart disease, and after 10 years of not smoking cigarettes this risk approaches that of the non-smoker.

Smoking is a major risk factor for arteriosclerotic peripheral vascular disease and is associated with more severe and extensive atherosclerosis of the aorta and coronary arteries than is found among nonsmokers. In persons with angina pectoris or with intermittent claudication of peripheral vascular disease, smoking reduces the individual's established threshold for the precipitation of angina or claudication. Although it does not induce chronic hypertension, cigarette smoking in the presence of hypertension acts synergistically to increase the effective risk for coronary heart disease. Women who smoke and use oral contraceptives are at a significantly elevated risk of fatal and non-fatal myocardial infarction and thromboembolism. The relationship of smoking to the incidence of stroke is not established; however, an association with subarachnoid hemorrhage has been reported in women.

Cancer: The most definite causal relationship between tobacco use and any disease was shown in the 1950s and 1960s with lung cancer. Since then, additional studies have strengthened the earlier conclusions, particularly with regard to women smokers for whom only preliminary data were then available. Lung cancer mortality rates in women are increasing more rapidly than in men, and if present trends continue, this disease will *(Continued on page 9)*

	1et W	EEK ENDING	1	CUMU	ATIVE EIRST	WEEK
D. 65 A 65			MEDIAN	- Cumu	CATIVE, TINGT	WEEN
DISEASE	January 6, 1979	January 7, 1978*	1974-1978**	January 6, 1979	January 7, 1978°	MEDIAN 1974-1978**
Aseptic meningitis	66	30	45	66	30	45
Brucellosis	1	3	3	1	3	3
Chickenpox	2,338	2,325	2,325	2,338	2,325	2,325
Diphtheria	5	-	2	5	-	2
Encephalitis: Primary (arthropod-borne & unspec.)	6	4	14	6	4	14
Post-infectious	1	3	2	1	3	2
Hepatitis, Viral: Type B	177	2 3 3	229	177	233	229
Type A	359	382	551	359	382	551
Type unspecified	142	131	124	142	131	124
Malaria	3	7	4	3	7	4
Measles (rubeola)	118	156	242	118	156	2 4 2
Meningococcal infections: Total	29	13	24	29	13	24
Civilian	29	13	24	29	13	24
Military	- 1	-	-	-	-	
Mumps	120	229	794	120	229	794
Pertussis	23	22	22	23	22	22
Rubella (German measles)	47	72	119	47	72	119
Tetanus	1	1	2	1	1	2
Tuberculosis	344	2 56	290	344	256	2 90
Tularemia	-	3	3	-	3	3
Typhoid fever	3	4	4	3	4	4
Typhus fever, tick-borne (Rky. Mt. spotted)	1	2	1	1	2	i
Venereal diseases:						-
Gonorrhea: Civilian	16,576	16,306	18,717	16.576	16.306	18.717
Military	670	2 92	413	670	292	413
Syphilis, primary & secondary: Civilian	396	284	410	396	284	410
Military	3	5	5	3	5	5
Rabies in animals	28	42	40	28	42	40

TABLE I. Summary – cases of specified notifiable diseases, United States [Cumulative totals include revised and delayed reports through previous weeks.]

TABLE II. Notifiable diseases of low frequency, United States

	CUM. 1979		CUM. 1975
Anthrax 1		Poliomyelitis: Total	-
Botulism	-	Paralytic	
Congenital rubella syndrome	-	Psittacosis 1	
Leprosy (NYC 1, Calif. 7, Hawaii 2)	10	Rabies in man	-
Leptospirosis † (Hawaii 1)	1	Trichinosis	- E 1
Plague	-	Typhus fever, flea-borne (endemic, murine)	

*Delayed reports received for calendar year 1978 are used to update last year's weekly and cumulative totals.

**Medians for gonorrhea and syphilis are based on data for 1976 1978.

t Delayed reports: Anthrax: Idaho +1 (1978); Leptospirosis: Conn. +1 (1978); Psittacosis: Wash. +3 (1978)

	ASEPTIC	ASEPTIC BAU	CHICK CO.			E	NCEPHALI	TIS	HEPATI	TIS (VIRAL), BY TYPE		
REPORTING AREA	MENIN- GITIS	CEL. Losis	POX	DIPHT	HERIA	Pri	mary	Post-in- fectious	В	A	Unspecified	MAL	ARIA
	1979	1979	1979	1979	CUM. 1979	1979	1978*	1979	1979	1979	1979	1979	CUM. 1979
UNITED STATES	66	1	2,338	5	5	6	÷.	1	177	359	142	3	3
NEW ENGLAND			384		-					10	2		
Maine	2	-	15	2	-	-	÷	2	-	3	-		12
Vt	-		12		-	-	Ξ.	-			-		-
Mass	-	-	3	-	-	-	-	-	1	-	-	-	-
R.I.	1	-	165	-	-	-	1	-	6	1	3	-	-
Conn.		-	161	-	-	-	-	-	1	4	-	-	-
10000	~	-	28	-	-	1	÷.	-	-	2	-	-	-
MID. ATLANTIC	10		103	1.000				1.251			2.2		
NY OF	17	-	13.8	-	2	1			52	14	14		-
N.I.	6	-	38	-	2	-	-	-	11	11	4	-	-
Pa.	-	-	14.14		-	-	-		15	16	4	-	-
	12	-	7	-	-	1	-		NA	NA	NA	-	-
E.N. CENTRAL											_		
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	-		233	-	~		-		-			-	-
W.N. CENTRAL													
Minn.	7	-	95	-	1	-		-	15	40	4		-
lowa	-	-	1	-		-	-	-	6	24	-	-	
Mo.		-	39	-		-	-	-	3	3	_	-	-
N. Dak.	<u>.</u>		2.9		- C	2	- S	-	2	8	2	20	
a Dak.		-	20	-	-	-			-	3	-	-	
Kane	2	-	23		-	-	1	1.1	1	2	2		
inanta,	-	-			-	-	-	-	-	-	-	-	
S. ATLANTIC													
Det	5	1	265		-	1	1	-	24	20	5	-	-
Md.	-	-	-	-	-	-	-	-	-	-	-	-	-
D.C.	2		31			-	1	-	12	8			-
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E.S. CENTRAL													
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W.S. CENTRAL													
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La.	-	-	-		-	-	-	-	1	3	1	1	1
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MOUNT	8	-	150	-			~		15	46	22	-	-
Mont	1	-	132		-		-	-	2	31	44	-	
Idaho	<u> </u>	-	81		÷	-	-	-	-	-	-	-	-
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PACIFIC													
Wash.	25		9.8	5	5	2	2	-	63	111	38	2	2
Oreg.	-		74	5	5	-	8	-	3	28	1	-	-
Calif.		-	-		-	-	-	-	9	15	2	-	-
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TABLE III. Cases of specified notifiable diseases, United States, weeks ending January 6, 1979, and January 7, 1978 (1st week)

Not notifiable.

Not notifiable. NA: Not available. Delayed reports received for 1978 are not shown below but are used to update last year's weekly and cumulative totals.

	M	EASLES (RU	BEOLA)	MENING	OCOCCAL INI TOTAL	FECTIONS	MUMPS		PERTUSSIS	AUB	ELLA	TETANUS
HEPORTING AREA	1979	CUM. 1979	CUM. 1978*	1979	CUM. 1979	CUM. 1978*	1979	CUM. == 1979	1979	1979	CUM. 1979	CUM. 1979
UNITED STATES	118	118	156	29	29	13	120	1 20	23	47	47	1
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	-	-	3	-	_	-	13	10	_	- <u>-</u>	-	_
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Vt.	-	-	-	-	-	-	-	-	-	2	2	-
Mass.	-	-	-	-	-	-	1	1	-	2	2	- 1
R.I.	-	-	-	+	-	-	4	4	-	-	-	- 1
Conn.	-	-	-	-	-	1	3	3	-	-	-	-
										_	_	
MID. ATLANTIC	12	12	44	5	2	1	12	12	4	9	9	1
Upstate N.Y.	4	6	50	2	2	-	1	:	2	4	4	1
N I	-	-		-	-	-	2	2	2	2	-	-
Pa.	-	-	1	1	1	-	ž	2	-	3	3	
E.N. CENTRAL	44	44	26	4	4	1	41	41	12	16	16	-
Ohio	-	-	_	1	1	_	2	2	1	1	ĩ	- 1
Ind.	1	1	-	-	-	~	5	5	_	-	-	
111.	33	33	1	-	-	-	9	9	10	12	12	-
Mich.	7	7	25	3	3	1	6	6	1	3	3	
Wis.	3	3	-	-	-	-	19	19	-	-	-	- 1
W.N. CENTRAL	4	4	1	1	1	3	1	1	-	2	2	_
Minn.	-	-	-	-	-	-	~	-	-		-	- 1
lowa	-	-	1	-	-	1	1	1	-	-	-	
Mo.	4	4	-	1	1	1	-	-	-	2	2	-
N. Dak.	-	-	-	-	-	-	-	~	-	-	-	-
S. Dak.	-	-	-	-	-	-	-	-	-	-	-	-
Nebr.	-	-		-	-	-	-	-	-	-	-	_
Kans.	-	-	-	-	-	1	-	-	-	-	-	-
S. ATLANTIC	7	7	13	9	9	-	10	10	1	2	2	-
Del.	-	-	1	1	1	-	1	1	-	_	-	
Md.	1	1	-	1	1	-	-	_	-	-	-	-
D.C.	-	-	-	-	-	-	-	-	-	-	-	
Va.	2	2	5	2	2	-	4	4	-	-	-	-
W. Va.	4	4	7	1	1	-	3	3	-	2	2	-
N.C.	-	-	-	-	-	-	2	2	-	-	-	- 1
S.C.	-	-	-	1	ł	-	-	-	-	-	-	-
Ga.	-	-	-	3	3	-	-	-	1	-	-	-
Fla.	-	-	-	-	-	-	-	-		-	-	-
E.S. CENTRAL	-	-	40	2	2	-	23	23	1	1	1	
Ky.	-	-	16	_	-	-	21	21	i	-	-	- 2
Tenn.	-	-	17	1	1	-	_		-	-	-	
Ala.	-	~	-	1	1	-	1	1	_	1	1	2
Miss.	-	-	7	-	-	-	1	1	-	-	-	-
W.S. CENTRAL	23	20	3	z	2	4	12	12	2	4	4	-
Ark.	_	_	_	1	1	1	_	-	-	-		-
La.	-	-	-	-	_	_	-	-	-	-	-	-
Okla.	-	-	-	-	-	-	-	-	-	-	-	1
Tex.	20	20	3	1	1	3	12	12	2	4	4	1
MOUNTAIN	4	4	18	3	3	-	2	2	_	3	٦	1.2.1
Mont.	2	2	18	_	-	-	1	ĩ	-	3	2	-
Idaho	_	_	_	-	-	-	_		-	<u> </u>	-	-
Wyo.	-	-	-	-	-	-	-	-	-	-	-	1
Colo.	-	-	-	-	-	-	-	-	-	-	~	-
N. Mex.	-	-	-	-	-	-	-	-	-	-	-	1
Ariz.	-	-	-	3	3	-	-	-	-	-	-	
Utah	-	-	-	-	-	-	-	-	-	-	-	11
Nev.	2	2	-	-	-	-	1	1	-	-	-	- 1
PACIFIC	27	27	8	2	2	3	9	q	2	*	4	
Wash.	23	23	ĩ	-	-	-	í	1	1	<u> </u>	0	
Oreg.	1	1	-	-	-	-	2	2	-	-	-	- 1 I.
Calif.	3	3	7	1	1	3	6	6	2	6	-	
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Hawaii	-	-		1	1	-	-	-	-	-		1
Guam	NA	-	-	-	-	-	ŇΑ	8 7 0	NA	N A		
P.R.	-	-	1	-	-	-	-	-	-	-	-	-
V.I.		-	-	-		-	-	-	-	-	-	
rac. Irust lerr.	1	1	10	-	-	-	2	2	-	-	-	- 2

TABLE III (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending January 6, 1979, and January 7, 1978 (1st week)

NA: Not available. *Delayed reports received for 1978 are not shown below but are used to update last year's weekly and cumulative totals.

	TURES	RCUI DSIS	TULA	TYP	ного	TYPHU	SFEVER	FEVER VENEREAL DISEASES (Civilian)						RABIES
REPORTING AREA			REMIA	FE	VER	(R/	MSF)		GONORRHEA		SYP	HILIS (Pri.	& Sec.)	Animals
	1979	CUM. 1979	CUM. 1979	1979	CUM. 1979	1979	CUM. 1979	1979	CUM. 1979	CUM. 1978°	1979	CUM. 1979	CUM, 1978*	CUM. 1979
UNITED STATES	344	344	-	3	3	1	1	16,576	16,576	16,306	396	396	284	28
NEW ENGLAND								201	201	201				
Maine	1	1	_	-	_	-	_	391	391	28	- 13	-	-	_
N.H.	-	-	~	-	-	-	-	17	17	12	-	-	-	-
Mass	2	2	-	-	-	-	-	8	8	4	-	-	-	-
R.I.	3	3	_	_	-	-	_	122	122	172	11	11	6	_
Conn.	2	2	-	-	(e:	_	-	164	164	159	ż	2	2	-
MID ATLANTIN														
Upstate N.Y	71	71	-	-	-	-	-	1,952	1,952	2,045	47	47	42	-
N.Y. City	17	17	-		_	_	_	538	538	718	42	42	25	
Pa	17	17	-	-	-	-	-	558	558	830	2	2	8	-
10	22	22	-	-	-	-	-	476	476	497	3	3	9	-
E.N. CENTRAL	37	27	_	_	_	_	_	2 360	2.050	1 613	66	66	22	,
Ohio	-		_	_	_		-	7.03	2,059	488	19	19	1	-
14.	7	7	-	-	-	-	-	15	15	193	-	-	ī	-
Mich.	30	3)	-	-	-	-	-	588	588	449	24	24	29	1
Wis.	_	-	-	_	-	_	-	583	583	483	10	10	-	_
W.N. CENING							-	170	115	-	2	د		
Minn.	8	а	-	-	-	-	-	867	867	643	7	7	4	5
lowa	1	1	-	-	-	-	-	99	99	174	2	2	3	1
Ma.	-	-	_	_	-	_	-	310	54 317	367		-	1	1
S. Dak.	ĩ	ĩ	-	-	-	_	-	19	19	17	-	-	-	_
Nebr.	-	-	-	-	-	-	-	16	16	30	-	-	-	~
Kans.	-	-	100	-	-	-	-	35	35	55	-	-		Ē.
S AT.	د	3	-	-	-	-	-	3 3 4	334	-	5	2	-	_
Del	89	89	-	-	-	1	1	3,723	3,723	4,035	75	75	100	6
Md,	-	-	-	-	-	-	-	84	84	18	2	2	1	-
D.C.	25	25	-	-	-	1	1	474	474	614	6	6	11	
Va. W.V.	-	-	_	_	_	_	-	263	263	350	11	11	6	_
N.C.	6	6	-		-	-	-	48	48	68	1	1	-	-
S.C.	8	8	-	-	-	-	-	405	405	482	14	14	4	-
Ga.	17	17	-	-	_	_	_	316	316	201	26	26	20	-
· (a.		-	-	_	_	_	-	1.250	1+250	1,370	11	11	33	~
E.S. CENTRAL														
Ky.	31	31	-	1	1	-	-	1,613	1,613	1,115	12	12	15	-
Ala	1	1	_	-	-	-	_	194	194	664	2	2	-	-
Miss.	ĝ	ĝ	-	1	1	-	-	633	600	319	3	3	5	-
March 1	21	21	-	-	-	-	-	286	286	79	7	7	4	-
ALL CENTRAL	1.4	1.4	_	_	_	_	_	2.051	2 0 5 3	3 (50)	4.6	4.5	30	1.2
La.	2	2	-	-	_	_	-	192	192	2,050	5	5	-	2
Okla.	10	10	-	-	-	-		62	62	436	_	_	-	-
lex,	2	2	-	-	-	-	-	185	185	213	1	1	-	4
MOUNT	_	-	-	-	-	-	-	2:014	2:014	11424	24	24	39	0
Mont	4	٠	-	-	-	-	-	476	476	534	8	8	-	-
Idaho	-	-	-	-	-	-	-	3	3	60	-	-	-	-
Colo		1.00		-			-	17	17	1.0				
N. Mex	_	-	_	-	-		-	159	158	161	6	6	-	-
Ariz	2	2	-	-	-	-		97	97	58	-	-	-	-
Otah Neu	-	-	-	-	-	-	-	69	69	135	-	-	-	-
	-	-	-	_	_	-	-	26	26	16	2	~	-	_
PACIFIC	٤.	٤				-	-	103	100		2	1		
Wash	81	81	-	2	2	-	-	2,542	2,542	3,277	114	114	44	4
Calif	NA	-		-	÷.	-	-	150	150	158	NA	-	4	-
Alaska	74	74	-	2	2	1	2	258	258	2.914	117	111	36	-
Hawari	-	12	-	2	2		-	72	72	30	-			
	7	7	-	-	-	-	-	45	45	48	2	2	3	-
Guara														
P.R.	NA	-	-	NA:	-	NA	~	NA		-	NA		-	-
V.I.	-	-	-	-	-	-	-	. 4	4	47	13	13	6	-
Tac. Trust Terr	17	-	- T	-	-	-	- 5	2	2		-	- E	-	
NA: No						100	-	7						

TABLE III (Cont.'d). Cases of specified notifiable diseases, United States, weeks ending January 6, 1979, and January 7, 1979 (1st week)

The Not available. *Delayed reports received for 1978 are not shown below but are used to update last year's weekly and cumulative totals.

TABLE IV. Deaths in 121 U.S. cities,* week ending January 6, 1979 (1st week)

		ALL CAUS	ES, BY AGE	(YEARS)			2423		ALL CAU	ISES, BY AG	E (YEARS)		P.8.1**
REPORTING AREA	ALL	>66	45-64	25-44	<1	P&I** TOTAL	REPORTING AREA	ALL AGES	>65	45-64	25-44	<1	P & I** TOTAL
NEW ENGLAND	658	441	165	21	14	31	S. ATLANTIC	1,020	609	276	72	37	44
Boston, Mass	54	34	45	3	2	8	Atlanta, Ga. Baltimora Md	141	89	34	14	3	6
Cambridge, Mass.	35	27	5	2	1	3	Charlotta, N.C.	62	26	20	10	5	3
Fall River, Mass.	32	27	5	-	-	-	Jacksonville, Fla.	79	37	25	3	9	3
Hartford, Conn.	58	39	13	2	-	2	Miami, Fla.	47	32	6	6	2	2
Lowell, Mass.	18	15	3	~	-	ĩ	Richmond, Va.	61	35	21	5	1	4
New Bedford, Mass.	29	21	6	1	-	2	Savannah, Ga.	41	20	13	6	ĩ	4
New Haven, Conn.	31	22	5	1	1	-	St. Petersburg, Fla.	100	62	16	5	-	6
Providence, R.I.	55	32	20	-	1	ĩ	Tampa, Fia. Werbington, D.C.	177	55	13	5	2	6
Springfield Mass.	45	28	14	1	2	5	Wilmington, Del.	79	53	18	2	3	3
Waterbury, Conn.	39	23	13	1	-	4	-						
Worcester, Mass.	55	40	10	2	1	د	C.C. OFNERAL			1.52			
							Birmingham, Ala.	551	522	155	ود 2	- 21	30
MID. ATLANTIC	2,295	1.463	537	149	69	105	Chattanooga, Tenn.	57	29	16	6	2	6
Albany, N.Y.	53	37	12	-	4	1	Knoxville, Tenn.	42	27	11	3	1	1
Allentown, Pa. Buffalo, N.V.	20	24	6	1	-	4	Louisville, Ky. Memobis Teon	97	67	22	5	2	1
Camden, N.J.	50	28	18	1	2	3	Mobile, Ala.	49	33	14	1	2	1
Elizabeth, N.J.	26	19	7	-	-	_	Montgomery, Ala.	26	11	6	2	-	1
Erie, Pa.1	وو . ب	20	10	1	1	2	Nashville, Tenn.	87	40	33	4	4	*
Newark, N.J.	73	30	21	11	8	5							
N.Y. City, N.Y.	1.514	962	349	104	÷3	64	W.S. CENTRAL	929	534	236	63	51	36
Paterson, N.J.	47	33	10	2	2	3	Austin, Tex.	38	26	2	3	3	3
Philadelphia, Pa. T	61	33	22	6	-	2	Baton Rouge, La.	35	18	8	1	6	4
Reading, Pa.	47	34	7	4	-	-	Dallas, Tex.	146	86	43	8	4	3
Rochester, N.Y.	136	\$3	32	11	3	11	El Paso, Tex.	43	25	8	2	3	6
Schenectady, N.Y.	25	21	2	1	_	2	Fort Worth, Tex.	67	42	14	6	3	1
Scranton, Pa. I Svracusa N.Y.	111	72	30	3	5	3	Little Bock Ark	141	68	39	15	3	2
Trenton, N.J.	46	24	14	5	-	3	New Orleans, La.	132	11	41	5	3	3
Utica, N.Y.	28	23	4	-	-	2	San Antonio, Tex.	140	79	37	8	11	3
Yonkers, N.Y.	42	30	0	-	1		Shreveport, La. Tulsa, Okla.	39 76	23 53	8 13	6 4	1	2
E.N. CENTRAL	2.284	1,376	620	136	82	80							
Akron, Ohio	55	36	10	2	5	-	MOUNTAIN	579	360	144	32	20	20
Canton, Ohio	51	29	16	4	1.6	4	Albuquerque, N. Mex.	49	30	14	2	1	5
Cincinnati, Ohio	125	302	32	7	4	8	Denver, Colo.	96	60	18	8	7	4
Cleveland, Ohio	170	104	53	9	2	4	Las Vegas, Nev.	53	28	19	4	-	1
Columbus, Ohio	88	51	23	6	6	3	Ogden, Utah	25	16	5	1	-	4
Dayton, Onio Detroit, Mich.	3.27	191	20	24	10	2 4	Phoenix, Ariz.	21	12	30	-	_	2
Evansville, Ind.	38	25	11	1	1	3	Sait Lake City, Utah	53	31	12	4	3	
Fort Wayne, Ind.	64	33	20	5	1	2	Tucson, Ariz.	111	77	26	5	2	1
Grand Banids Mich	26	13	12	1 7	1	1							
Indianapolis, Ind.	165	85	60	6	7	3	PACIFIC	1.700	1.071	414	100	67	57
Madison, Wis.	40	20	14	1	4	2	Berkeley, Calif.	16	10	- 4	2	-	-
Milwaukee, Wis. Peoria III	153	105	41	3	2	3	Fresno, Calif.	76	45	23	3	- 4	4
Rockford, III.		32	10	3	4	2	Honolulu Hawaii	11	27	20	5	-	1
South Bend, Ind.	37	27	4	4	1	3	Long Beach, Calif.	127	81	30	6	8	4
Toledo, Ohio	110	70	30	7	2	1	Los Angeles, Calif.	379	247	82	26	13	13
roungstown, onio	62	42	17	1	2	2	Dakland, Calif. Pasadena, Calif.	61 33	33 23	16	8	3	2
W.N. CENTRAL	721	468	1 4 1	44	45	37	Sacramento. Calif	128	86	31	6	4 6.	2
Des Moines, Iowa	41	32	5	1	1	1	San Diego, Calif.	157	97	40	8	6	1
Duluth, Minn. Kansas City, Kana	36	24	6	2	3	4	San Francisco, Calif.	175	123	34	1.0	6	9
Kansas City, Mo.	136	16	31	-	1 7	Z	San Jose, Calif.	169	103	47	6	2	5
Lincoln, Nebr.	27	15	10	_	_	4	Spokane, Wash.	149	97	37	5	4	5
Minneapolis, Minn.	85	60	11	5	5	5	Tacoma, Wash.	39	24	12	2	-	2
Omaha, Nebr. St. Louis Mo	66	42	19	1	4	-					-		
St. Paul, Minn.	85	63	10	8	16	10		0 72 7					
Wichita, Kans.	54	27	14	5	5	3	Expected Number	11.554	7.135	2.686	650	406	440
									11132	6 0 0 7 7	oof	419	7000

*Mortality data in this table are voluntarily reported from 121 cities in the United States, most of which have populations of 100,000 or more. A death is

reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included. **Pneumonia and influenza

Telecause of changes in reporting methods in these 4 Pennsylvania cities, there will now be 117 cities involved in the generation of the expected values used ^(c) monitor pneumonia and influenza activity in the United States. Data from these 4 cities will appear in the tables but will not be included in the totals for the United States and the Middle Atlantic Region.

Smoking - Continued

be the leading cause of cancer deaths in women in the next decade.

Use of filter cigarettes and low tar and nicotine cigarettes decreases lung cancer mortality rates among smokers although not to the low rates for nonsmokers. Exsmokers ^{experience} decreasing lung cancer mortality rates which approach the rates of nonsmokers after 10 to 15 years of cessation.

Evidence has accumulated about the relationships between tobacco use and cancer of the larynx, oral cavity, esophagus, urinary bladder, kidney, and pancreas. Cigarette smoking is a significant causative factor in laryngeal, oral, and esophageal cancers, as well as in lung cancer, and in all of these there is a dose-response effect. For each of these cancers there also appears to be a synergistic effect between smoking and alcohol intake. Pipe and cigar smokers experience approximately the same risk as cigarette smokers for all of these cancers.

Epidemiologic studies have demonstrated a significant association between cigarette smoking and bladder cancer in both men and women, with smoking acting both independently and synergistically with other factors to increase the risk of developing bladder cancer. Cigarette smoking is also associated with cancer of the kidney in men (mortality ratio of 1.5) (Table 1), and with cancer of the pancreas.

Non-neoplastic Bronchopulmonary Disease: Both female and male cigarette smokers have a significantly higher prevalence of chronic bronchitis and emphysema than nonsmokers, and they have an increased chance of dying from these diseases. Smokers, including teenagers who smoke, also have more respiratory symptoms, and at least some of these symptoms are dose-related. Pulmonary function abnormalities are greater among cigarette smokers than nonsmokers, with impairment of pulmonary function detectable even among smokers in young age groups. Cessation of smoking improves pulmonary function, and decreases the prevalence of respiratory symptoms as well as the chance of premature death from non-neoplastic bronchopulmonary disease. Autopsy data have demonstrated dose-related abnormalities in macroscopic and microscopic lung sections among smokers.

Interaction with Occupational Exposures: Significant excess lung cancer mortality has been noted for chromate, nickel, coal gas, and asbestos workers and for uranium miners; exposures to arsenic, hematite, beryllium, and copper are suspect. However, as noted in the 1964 Report, only a small part of the smoking population is exposed to industrial carcinogens and these agents cannot account for the rising lung cancer risk in the general population. Unfortunately, few studies of occupational hazards include smoking histories, and therefore smoking, as a contributory or etiologic factor, cannot be fully evaluated.

Six ways have been identified in which smoking and physical and chemical agents in the workplace interact to produce or increase adverse health effects. Tobacco products may serve as vehicles for toxic agents, thus facilitating bodily entry by the agents. Workplace chemicals can be transformed into more harmful agents by smoking. Certain toxic agents in tobacco products and/or smoke can also be present in the workplace (carbon monoxide, for example), thus increasing exposure to the agent. Or, smoking can cause an effect comparable to that which results from a toxic agent and thus cause an additive biological effect. Alternatively, smoking can act synergistically with toxic agents, as is workplace.

Those who have the highest risk of occupational exposure to toxic agents in general also have the highest smoking rates. Surveys have shown that male, blue-collar workers are much more likely to smoke than white-collar workers.

Pregnancy and Infant Health: Birth weight and fetal growth are significantly affected

Smoking - Continued

by smoking during pregnancy. Babies born to women who smoke during pregnancy are on the average 200 g lighter than babies born to comparable women who do not smoke. Twice as many of these babies weigh less than 2,500 g compared with babies of nonsmokers. Strong evidence indicates that this reflects the fetal growth rate since there is no overall reduction in the duration of gestation when the mother smokes. The ratio of placental weight to birth weight increases with increasing levels of maternal smoking, possibly as a compensatory mechanism for the reduced oxygen availability due to carbon monoxide.

Epidemiologic studies do not provide conclusive data on the effect of maternal smoking on adequacy of lactation, although some animal studies reveal diminished milk production following administration of nicotine. A direct dose-response relationship does exist between the number of cigarettes smoked and the level of nicotine in breast milk.

Accumulated evidence does not indicate that maternal smoking increases the incidence of congenital malformations.

Peptic Ulcer Disease: Six studies have shown a greater prevalence of peptic ulcer disease among male cigarette smokers than among nonsmokers, the median ratio being 1.7 (i.e., their risk is increased by 70%); findings are comparable in women. There is a positive dose-response relationship between smoking and the incidence of peptic ulcer disease, and the risk of dying from peptic ulcer disease is, on the average, twice as high for smokers as for nonsmokers.

Allergy: Tobacco and tobacco smoke extracts have been found to act as antigens and to sensitize T-lymphocytes. However, the precise role of tobacco in immune and allergic processes cannot yet be delineated. Tobacco smoke does exert a variety of effects on respiratory tract structures; chronic smoking leads to consistent histologic changes in the respiratory tract including an adverse long-term effect on the mucociliary transport mechanisms and mucus composition, and morphologic and physiologic changes in macro phages. Alterations in cell-mediated immunity are noted locally and systemically in smokers.

Behavioral Aspects: There is some evidence that the smoking patterns of chronic smokers may be dependent on the nicotine content of their cigarettes. It is noted that the biological half-life of nicotine in the human body is approximately 20-30 minutes and that regular smokers commonly consume 1 cigarette every 30-40 minutes, possibly in an attempt to maintain a constant nicotine level in the blood.

Abrupt and total withdrawal from tobacco is associated with symptoms that subside more quickly and are no worse than those seen in partial abstinence. There is fragmentary evidence suggesting that the abstinence syndrome is more severe in women than in men

Estimates from the 1978 Health Interview Survey indicate that 33.2% of the U.5 population 17 years of age and older smoke (Table 2). It has been estimated that 95% of the 29 million smokers who have quit since 1964 have done so on their own. Survey dati show that only one-third or less of smokers motivated to quit are interested in formal programs, and only a small minority of those actually attend programs when offered Objective data are lacking on most of the smokers who have attended formal programs and controlled research has yet to produce a clearly superior intervention strategy. The current trend in adult-education programs is an emphasis on personal responsibility for health and on adoption of a health-promoting lifestyle.

Very few carefully designed and implemented longitudinal studies exist in the are of smoking in children and adolescents. The critical level for onset of habitual smokin^a appears to be in high school. Therefore, the true impact of any anti-smoking program with adolescents may not even be measurable until after the adolescent has entered high school.

10

Smoking - Continued

TABLE 2.	Estimates	of the per-	centage of	current,	regular	cigarette	smokers,	adults,
United Sta	tes. 1964-	1978	-		•			

Year	Inte (17	Health erview Su yrs. and	over)	National Clearinghouse for Smoking & Health (21 yrs. and over)				
10.5	Total	Mate	Female	Total	Male	Female		
1964 1965	41.7	51.1	33.3	40.3	52.9	31.5		
1966 1967				42.2	51.9	31.7		
1968 1969								
1970 1971	36.9	43.5	31.1	36.2	42.3	30.5		
1972								
1973 1974 1975	37.0	42.7	31.9	22.0	20.2	26.0		
1976 1977	36.7	41.9	32.0	33.8	39.3	20.9		
1978	33.2	37.5	29.6					

A promising new approach to offset adolescent pressures to smoke involves strategies to resist peer pressure, to understand how advertising and mass media work to influence smoking, and to provide information on ways to resist the models of parents, siblings, and older students who smoke. Also included is a focus on the immediate physiologic effects of smoking, rather than on long-term effects. Researchers find that "significant adults" (such as physicians) are powerful influences on teenage smoking.

Involuntary Smoking: Only a limited amount of systematically collected information regarding the health effects of involuntary (passive or second-hand) smoking on the nonsmoker is available, and much of that centers on exposure to carbon monoxide (CO). Cigarette smoking in enclosed spaces can produce CO levels well above the Ambient Air Quality Standard (9 ppm), even where ventilation is adequate. These levels of CO have been shown to decrease the exercise threshold for angina in patients with coronary artery disease and for intermittent claudication in patients with peripheral vascular disease. In healthy adults, levels of carboxyhemoglobin produced in involuntary smoking situations are functionally insignificant.

Substantial proportions of healthy adults experience irritation of eyes and nose when exposed to cigarette smoke. Changes in psychomotor function, especially attentiveness and cognitive function, have been noted. In children of parents who smoke, bronchitis and pneumonia are more common during the first year of life than in children of nonsmokers. Reported by the Office on Smoking and Health, U.S. Department of Health, Education, and Welfare, Office of the Assistant Secretary for Health.

Send mailing list additions, deletions, and address changes to: Center for Disease Control, Attn: Distribution Services, GSO, 1-SB-36, Atlanta, Georgia 30333. When requesting changes be sure to give your former address, including zip code and mailing list code number, or send an old address label.

The Morbidity and Mortality Weekly Report, circulation 84,000, is published by the Center for Disease Control, Atlanta, Georgia. The data in this report are provisional, based on weekly telegraphs to CDC by state health departments. The reporting week concludes at close of business on Friday.

Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other nublic health problems of current interest to health officials. Send reports to: Center for Disease Control, Attn: Editor, Morbidity and Mortality Weekly Report, Atlanta, Georgia 30333.

Epidemiologic Notes and Reports

Trichinosis Associated with Bear Meat – Alaska, California

Thirteen Alaska Natives and persons of oriental descent have become ill recently with trichinosis after eating Alaskan black bear meat. The meat was eaten on multiple occasion^s in late October and November, sometimes after being roasted for several hours, and of some occasions after preparation in an oriental wok. Thus far, all cases have been asso ciated with the wok-prepared meat.

The index patient, a 39-year-old man, ate the meat in early November while visiting in Anchorage, and then returned to his home in Los Angeles. On December 7, he presented to the emergency room of a hospital there with at least a 1-week history of feverretro-orbital pain, orbital puffiness, and muscle tenderness. He was found to have 50% eosinophilia. A diagnosis of trichinosis was made, and he was admitted to the hospital When he gave a history of eating bear meat with his relatives in Alaska, authorities notified the Alaska State Department of Health and Social Services. Investigation by thistate health department uncovered the other 12 cases.

Samples of the implicated bear meat, when tested by the U.S. Department of Ag^{rl} culture, were found to contain up to 1200 larvae/gram. Portions of the bear were take^{fl} to Los Angeles and Chicago by visitors returning home from Alaska. An epidemiolog^{il} investigation of potentially exposed persons in those areas and in Alaska is in progress Reported by J Cinqué, MD, LAC-USC General Hospital, Los Angeles; S Fannin, MD, Los Angel^{fl} County Dept of Health; R Brodsky, MD, Alaska Native Medical Center, Anchorage; J Farrell, ^T. Woodard, MD, Acting State Epidemiologist, Anchorage; U.S. Dept of Agriculture, Palmer, Alask^{fl} Alaska Investigations Div, Field Services Div, Bur of Epidemiology, CDC.

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The index patient, a 39-year-old man, ate the meat in early November while visitin! in Anchorage, and then returned to his home in Los Angeles. On December 7, he pre sented to the emergency room of a hospital there with at least a 1-week history of fever retro-orbital pain, orbital puffiness, and muscle tenderness. He was found to have 50[%] eosinophilia. A diagnosis of trichinosis was made, and he was admitted to the hospital When he gave a history of eating bear meat with his relatives in Alaska, authorities notified the Alaska State Department of Health and Social Services. Investigation by thi state health department uncovered the other 12 cases.

Samples of the implicated bear meat, when tested by the U.S. Department of Agri culture, were found to contain up to 1200 larvae/gram. Portions of the bear were taker to Los Angeles and Chicago by visitors returning home from Alaska. An epidemiolog^{ic} investigation of potentially exposed persons in those areas and in Alaska is in progress Reported by J Cinque, MD, LAC-USC General Hospital, Los Angeles; S Fannin, MD, Los Angele County Dept of Health; R Brodsky, MD, Alaska Native Medical Center, Anchorage; J Farrell, TL Woodard, MD, Acting State Epidemiologist, Anchorage; U.S. Dept of Agriculture, Palmer, Alaska Alaska Investigations Div, Field Services Div, Bur of Epidemiology, CDC.

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