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Current Trends

Childhood Lead Poisoning – United States: Report to the Congress by the Agency for Toxic Substances and Disease Registry

INTRODUCTION

The long-term consequences of unabated exposures to environmental lead sources can be serious, particularly for children. Recent scientific studies have shown a progressive decline in the lowest exposure levels of lead at which adverse effects can be reliably detected in children. In recognition of this, Congress directed the Agency for Toxic Substances and Disease Registry (ATSDR), in consultation with the Environmental Protection Agency (EPA), to examine the nature and extent of childhood lead poisoning in the United States. The study was to address such areas as the long-term health implications of environmental lead exposure in children, the extent of lead intoxication of children in terms of geographic areas and sources of lead in the United States, and methods and strategies for removing lead from the environment of U.S. children. This article summarizes the key findings of the report (1).*

EXPOSURE CLASSIFICATION

The degree of exposure to children was classified by blood lead (Pb-B) levels of 25, 20, and 15 μ g/dL. The groupings were based on 1) presence of both 25 μ g/dL Pb-B and elevation of erythrocyte protoporphyrin (EP)[†]; 2) the corresponding value of 20 μ g/dL used recently by the World Health Organization[†] for the European Economic Community; and 3) findings of EPA's Clean Air Scientific Advisory Committee,[†] which concluded that 10–15 μ g/dL of lead is associated with the onset of effects that "may be argued as becoming biomedically adverse" (2). Levels of 25, 20, 15, and 10 μ g/dL were used for grouping pregnant women and women of reproductive age when estimating fetal lead exposure and potential adverse health effects (2–4).

^{*}The complete report is available on request from ATSDR, Mailstop F38, Atlanta, Georgia 30333. *References are available on request from the Office of the Associate Administration, ATSDR, Mailstop F38, Atlanta, Georgia 30333.

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TOXIC EFFECTS OF LEAD

Infants and young children are at highest risk for the adverse health effects of lead (2,3). Exposures of women of childbearing age are also a concern because lead is directly transferred across the placenta; therefore, the developing fetus is exposed at levels proportional to maternal lead stores (5-7).

The toxic effects of lead in children (Table 1) are evident across a broad range of exposures, and some occur at Pb-B levels previously considered noninjurious (i.e., <25 μ g/dL) (4). Further follow-up studies are needed before the impact and persistence of the low-level neurobehavioral effects are fully known.

ESTIMATES OF THE NUMBERS OF CHILDREN EXPOSED TO LEAD

Valid estimates of the total number of lead-exposed children according to standard metropolitan statistical areas (SMSAs) or other appropriate geographic units smaller than the nation as a whole are not possible. The only national data set for Pb-B levels in children comes from the National Health and Nutrition Examination Survey II (NHANES-II). Using this data set, ATSDR quantified the numbers of lead-exposed children (ages 6 months to 5 years) living in all SMSAs according to 30 socioeconomic and demographic strata and selected Pb-B levels.

These estimates for 1984 were projected from data collected in 1976–1980 (the years of NHANES-II). The degree of error in these estimates is difficult to quantify since sources of both overestimation and underestimation are present. In addition, Hispanic, Asian, and other subgroups are omitted because no data are available;

Neurologic effects	Heme synthesis effects	Other effects	Lowest level Pb-B (μg/dL) effect seen
Deficits in neurobe- havioral develop- ment (Bayley and McCarthy Scales); electrophysiological changes	ALA-D inhibition	Reduced gestational age and weight at birth; reduced size up to age 7–8 years	10–15 (prenatal & postnatal)
	EP elevation	Impaired vitamin D metabolism; Py-5-N inhibition	15–20
Lower IQ, slower reaction time (studied cross-sectionally)			<25
Slowed nerve con- duction velocity			30
	Reduced hemoglo- bin; elevated CP and ALA-U		40
Peripheral neuropa- thies	Frank anemia		70
Encephalopathy		Colic, other GI ef- fects; kidney effects	80–100

TABLE 1. Adverse effects of lead*

*Adapted from (2), with updating.

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however, no economic or racial subgrouping of children is exempt from the risk of sufficiently high Pb-B levels to cause adverse health effects.

For all SMSAs, about 400,000 fetuses are exposed to maternal Pb-B levels of more than 10 μ g/dL and are therefore at risk for adverse health effects.

Of the estimated 2,380,600 children exposed to lead at levels above 15 μ g/dL (about 17% of the total 13,840,000 children within SMSAs), an estimated 715,500 (5.2%) and 199,700 (1.4%) children have Pb-B levels >20 μ g/dL and >25 μ g/dL, respectively.

ATSDR SURVEY OF LEAD SCREENING PROGRAMS

The most current data for lead screening results came from an ATSDR survey conducted in December 1986. All data for 1985 screening programs were voluntarily reported. There was no centrally administered data collection and assessment. Of 785,285 children screened by those programs in 1985, 11,739 (1.5%) had lead toxicity as determined by one of two CDC definitions (1978 criteria: a Pb-B level of 30 μ g/dL and an EP level \geq 50 μ g/dL; 1985 criteria: a Pb-B level 25 μ g/dL and an EP level \geq 35 μ g/dL).

Based on examination of 1980 census data for children and their housing in the 318 SMSAs, for 35 SMSAs, \geq 50% of the children were at high risk of exposure to leaded paint because their housing was built before 1950. For all 318 SMSAs, 4.4 million children were at potential risk because they lived in older housing with high lead content paint.

SOURCES OF LEAD CONTRIBUTING TO HUMAN EXPOSURE

The ATSDR estimates of children exposed to lead by source are shown in Table 2. Because of the interrelating pathways of exposure, the numbers of children exposed to lead on a source-specific basis can only be estimated. In addition, since the type and availability of data for each lead source vary considerably, exposure category definitions are different for each of the major lead sources, i.e., paint, gasoline, stationary sources, dust/soil, water, and food. Since the estimated numbers of children for each source and category are not comparable, they cannot be used to rank the severity of the lead problem by source of exposure.

The total of approximately 12 million children exposed to leaded paint is for children <7 years of age; the estimated 5.9 million children in the oldest housing

Source	No. children* (millions)
Leaded paint [†]	12.0
Leaded gasoline	5.6
Stationary sources	0.233
Dust/soil	5. 9– 11.0
Water/plumbing ^{\$}	10.4
Food	1.0

TABLE 2. Estimated numbers of children exposed to sources of lead

*Numbers in the table are not additive since children are usually exposed to multiple sources of lead in the environment.

[†]Includes children in oldest housing: 5.9 million; children in old/deteriorated housing: 1.8–2.0 million.

[§]Residential service lead connection: 4.8 million; leachable lead solder: 1.8 million; water lead level >20 µg/mL: 3.8 million.

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were <6 years of age. Of the estimated 1.8 to 2.0 million children living in old and deteriorated housing, approximately 230,000 would be expected to have Pb-B levels >30 μ g/dL and 1.3 million >15 μ g/dL because of leaded paint exposure.

MMWR

The estimate of 5.6 million children <7 years of age potentially exposed to lead at some level from gasoline are derived for the number of children in the 100 largest urban areas in the United States where vehicular traffic could be expected to figure significantly in childhood lead exposure. This estimate takes into consideration the phase-down of lead in gasoline required by EPA regulations.

Although data are very limited, an estimated 233,000 children are exposed to lead from stationary sources of all types (8).

Dust/soil lead is lead that has settled from leaded paint, gasoline, and stationary sources; therefore, exposure estimates can only be roughly calculated as the sum of these three categories. Dust/soil lead is the primary long-term repository for lead exposure; this pathway is a major contributor to overall lead exposure because of the hand-to-mouth activity of children.

Children are exposed to lead in drinking water primarily from contamination of the supply system (e.g., from lead pipes or from leachable lead solder). EPA recently estimated that 241,000 children <6 years of age have Pb-B levels >15 μ g/dL because of elevated concentrations of lead in drinking water, including 100 with Pb-B levels >50 μ g/dL, 11,000 with levels 30–50 μ g/dL, and 230,000 with levels 15–30 μ g/dL (9).

Because the use of lead-soldered food cans has decreased dramatically in recent years, lead in food is a declining source of exposure. Nonetheless, dietary exposure remains sufficient to add measurable amounts to the total Pb-B levels of as many as 1 million U.S. children.

REDUCING EXPOSURE TO LEAD

Primary environmental lead abatement has been most effective for gasoline, stationary lead sources, and food. Activities are now under way to bring significant reductions of lead in water. Despite the marked reductions of lead in new paint (in 1977, the Consumer Product Safety Commission mandated the reduction of lead in paint to 0.06%), exposure to lead paint in old housing remains an important problem. In addition, lead from other sources that has been deposited in dust and soil is recognized as an important problem.

Abatement of secondary environmental lead exposure is closely linked to childhood lead-screening programs. These efforts involve environmental evaluation and abatement of exposure for children with recognized lead toxicity. Early screening and detection of exposure and toxicity have reduced the rates of severe lead poisoning. Such effective screening programs will reduce the toxicity and costs of lead poisoning. The success of screening programs in the past has been limited by the 1) difficulty of locating all children with lead toxicity; 2) inability to identify remediable sources of lead for many lead-poisoned children; and 3) incomplete removal of lead from the children's environments. Some methods of removing and disposing of lead from homes and other sites are relatively crude and can endanger both abatement workers and occupants.

REPORT RECOMMENDATIONS

Recommendations contained in the ATSDR report include the needs to 1) integrate comprehensive approaches to controlling lead exposure in high-risk areas of the United States, 2) establish and maintain effective and efficient screening

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programs, 3) develop environmental measurement techniques for field use, and 4) conduct research on childhood lead poisoning and develop effective legal sanctions.

Reported by: Agency for Toxic Substances and Disease Registry and Center for Environmental Health and Injury Control, CDC.

Editorial Note: In the past 2 decades, knowledge of the effects of lead poisoning has changed substantially. When national childhood lead poisoning prevention programs were instituted in the early 1970s, lead encephalopathy and other manifestations of severe overt lead poisoning were common. Today these outcomes are rare—to a great extent because of childhood lead-screening programs in high-risk areas and reduction of lead in the environment (particularly for gasoline, air, and food).

Despite these reductions, childhood lead poisoning is not disappearing. The ATSDR lead report documents three critical developments in lead poisoning (1). First, long-term effects (particularly neurobehavioral, cognitive, and developmental) are increasingly being observed in studies of children with lead levels much lower than previously believed harmful. Second, the numbers of children exposed to lead at these new lower levels of concern (corresponding to Pb-B levels approximately \geq 15 µg/L) are estimated at several million. This estimate is largely based on projections from the data in NHANES-II; NHANES-III will provide updated data in the coming decade. Third, the remaining important sources of lead in the environment (primarily lead paint in older housing and lead in dust and soil from past deposition and from deteriorating housing) will be difficult and expensive to remedy.

Childhood lead poisoning is one of the most common environmental diseases of children in the United States. In concept, it is a totally preventable disease – remove the lead from the child's environment and the disease will disappear. In practice, eliminating childhood lead poisoning will require substantial commitment.

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Syphilis and Congenital Syphilis – United States, 1985–1988

In 1987, 35,241 cases of primary and secondary syphilis were reported in the United States. The incidence of 14.6 cases per 100,000 persons equals that of 1982 - the highest rate since 1950. The 25% increase over the 1986 rate was the largest single-year increase since 1960. Because of this increase, the Public Health Service objective to reduce the incidence of primary and secondary syphilis to 7.0 cases/100,000 persons by 1990 (1,2) is unlikely to be achieved.

The increase in incidence was greatest for blacks and Hispanics – groups for which incidence rates were already high (Figure 1). In all racial/ethnic groups, increases were greater for females than for males. From 1986 to 1987, the rate per 100,000 persons 15–64 years of age* increased 36% for black males (106.2 to 144.9), 43% for black females (55.5 to 79.4), 7% for Hispanic males (66.0 to 70.7), and 24% for Hispanic females (17.8 to 22.0). In contrast, the rate for white males decreased from 6.4 to 5.7, while for white females, rates increased 22% (2.2 to 2.6). The decrease among white males appears to be attributable to continuing decreases in syphilis incidence among homosexual men (3).

In 1987, 57% of all reported U.S. cases were reported from Florida, California, and New York (Table 1). Six additional states and the District of Columbia had 1987 incidence rates >7.0/100,000 and had increases between 1985[†] and 1987 (Table 1). Eleven other states had 1987 incidence rates >7.0/100,000, but incidence did not increase from 1985 to 1987 (Figure 2). In Texas, rates decreased steadily from

*Ninety-nine percent of cases in 1987 occurred in persons 15–64 years old. *1985 was chosen as baseline for this comparison because, in several areas, the increases began during 1986.

FIGURE 1. Incidence of primary and secondary syphillis in persons aged 15–64 years, by race/ethnicity and sex – United States, 1986–1987



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28.4/100,000 in 1985 to 18.4/100,000 in 1987. In Nevada, Oregon, Delaware, Connecticut, and Pennsylvania, syphilis rates were below the 1990 objective of 7.0/100,000 in 1985.

The highest rates were reported in urban areas; this was especially apparent in New York and Pennsylvania. The 1987 rate per 100,000 persons was 63.5 in New York City, compared with 3.4 for the rest of New York, and 41.6 in Philadelphia, compared with 2.5 for the rest of Pennsylvania.

The national increase was first noted in the last half of 1986 (Figure 3), reflecting

	198	35	198	37
Area	No.	Rate	No.	Rate
District of Columbia	342	55.3	425	69.1
Florida	3679	32.6	7453	62.5
California	4326	16.6	7718	28.2
New York	2530	14.2	5004	28.1
Nevada	64	6.9	180	18.1
North Carolina	672	10.9	752	11.9
Oregon	112	4.2	310	11.4
Delaware	39	6.3	71	11.1
Connecticut	215	6.8	334	10.5
Pennsylvania	513	4.3	941	7.9
Total United States	27,143	11.5	35,241	14.6

TABLE 1. Areas with reported 1987 incidence rates of primary and secondary syphilis >7.0/100,000 persons and an increase in incidence between 1985 and 1987 – United States

FIGURE 2. Rates of primary and secondary syphilis per 100,000 persons in 1987 and change from 1985 - United States



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increases in Florida, California, and New York. The national increase peaked in the third quarter of 1987, then plateaued through the first half of 1988, again reflecting trends in Florida, California, and New York. In other areas, such as Connecticut, Tennessee, and Nevada, rates continued to increase during the first half of 1988. In Pennsylvania, where the incidence remained stable but elevated after a large increase in early 1986, the rate began to increase again in 1988.

In the second half of 1987, the rate of congenital syphilis cases increased 21% to 10.5 cases per 100,000 live births. Most cases occur in areas with high syphilis incidence among adult women; in 1987, 67% of all cases were reported from Florida, California, and New York.

Reported by: Participating city and state health depts and STD control programs. Div of Sexually Transmitted Diseases, Center for Prevention Svcs, CDC.

Editorial Note: Decreases in syphilis and gonorrhea (3–7) in homosexual men reflect changes in sexual behavior related to controlling the spread of human immunodeficiency virus (HIV) in that population. The increases in incidence of syphilis described here suggest that efforts to achieve similar behavioral changes in minority populations have not been successful (8). In addition, the evidence is strong, especially from Africa, that genital ulcer diseases like syphilis increase the efficiency of sexual transmission of HIV (9–12).

In March 1988, CDC reviewed the trends in syphilis with sexually transmitted disease experts from academic/medical institutions and state and local health departments. This group identified the following three research priorities: 1) defining the current epidemiology of syphilis, including the relationship with illegal drug use, 2) evaluating and improving the effectiveness of different intervention methods, and 3) evaluating the effect of HIV coinfection on syphilis transmission.

The following interventions were suggested as being essential if these trends of increased syphilis rates are to be reversed:

FIGURE 3. Rate of primary and secondary syphilis per 100,000 persons, by quarter and year of report – United States, 1984–1988



*Rate for 2nd quarter of 1988 is based on preliminary data reported to MMWR.

Syphilis and Congenital Syphilis - Continued

- 1. Reemphasize the traditional methods of syphilis control-interviews and sex partner notification.
- 2. Conduct screening for sexually transmitted diseases in high-risk populations.
- Assure access to quality clinical care by removing financial barriers and other obstacles (e.g., long waiting times and lack of evening hours).
- 4. Enhance current surveillance systems to allow ongoing evaluation of intervention strategies and effective resource allocation.

Congenital syphilis, a preventable consequence of untreated syphilis in pregnant women, causes fetal or perinatal death in 40% of affected pregnancies (13). Because increases in congenital syphilis lag behind increases in syphilis in women by about 1 year (14), congenital syphilis can be expected to continue to increase in frequency. This may be a particular problem for urban black and Hispanic women, who have a disproportionate increase in incidence and who are less likely than white women to receive adequate prenatal care (15).

Congenital syphilis can be prevented by appropriate treatment of the mother during pregnancy (13). Syphilis screening in pregnant and childbearing-aged women is the best way to identify those who need treatment. In addition, efforts must be made to remove obstacles that prevent women from receiving early prenatal care, especially in areas with high syphilis incidence.

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Update: *Salmonella enteritidis* Infections and Grade A Shell Eggs – United States

Salmonella enteritidis (SE) continues to be an important cause of outbreaks of gastroenteritis. This report describes recent outbreaks of SE infections that have been associated with Grade A eggs.

Fort Monmouth, New Jersey. From May 3 to May 9, 1988, 88 (47%) of 188 students in a New Jersey college preparatory school developed febrile gastroenteritis. Symptoms included diarrhea, abdominal pain, headache, and fever. Twenty-seven (31%) of the ill students were hospitalized, and all recovered; stool cultures from each ill patient yielded SE. An epidemiologic investigation indicated that homemade ice cream prepared with Grade A raw eggs only 2 hours before consumption was the source of the outbreak. A culture of the implicated ice cream yielded SE. The ice cream had been properly cooled, and no food handling errors were identified.

Asbury Park, New Jersey. An outbreak of SE infections was reported in a group of 100 service organization trainees who had stayed at the same hotel in Asbury Park, (Continued on page 495)

	32	nd Week End	ling	Cumulative, 32nd Week Ending			
Disease	Aug. 13,	Aug. 15,	Median	Aug. 13,	Aug. 15,	Median	
	1988	1987	1983-1987	1988	1987	1983-1987	
Acquired Immunodeficiency Syndrome (AIDS) Aseptic meningitis Encephalitis: Primary (arthropod-borne	496 178	U* 441	155 418	19,322 2,930	11,998 5,119	4,621 4,424	
& unspec) Post-infectious	22	50 3	34 2	437	648 75	618 75	
Gonorrhea: Civilian Military	13,/90 343	14,815 602	17,437	416,456 7,569	4/9,49/ 10,356	530,774	
Hepatitis: Type A	4/5	365	406	14,808	15,095	13,229	
Type B	514	454	489	13,469	15,787	15,434	
Non A, Non B	56	45	70	1,573	1,948	2,241	
Unspecified	54	37	100	1,291	1,898	2,968	
Legionellosis	41	16	18	537	571	438	
Leprosy Malaria Messies: Total [†]	4 24 61	23 17	3 23 27	493 2 014	514 3 149	155 537 2 236	
Indigenous Imported	56 5	13 4	20 4	1,804	2,777	1,924 255	
Meningococcal infections	39	32	32	1,965	1,981	1,893	
Mumps	33	85	34	3,279	9,987	2,335	
Pertussis	87	80	80	1,375	1,275	1,318	
Rubella (German measles)	4	8	9	141	273	472	
Syphilis (Primary & Secondary): Civilian	733	979	565	23,416	21,273	16,932	
Military	2	7	4	105	107	113	
Toxic Shock syndrome	10	9	9	198	196	245	
Tuberculosis	443	372	397	12,402	12,799	12,910	
Tularemia	7	5	8	114	122	122	
Typhoid Fever	9	7	10	205	184	201	
Typhus fever, tick-borne (RMSF)	26	24	27	393	389	440	
Rabies, animal	60	80	107	2,582	3,028	3,257	

TABLE I. Summary - cases of specified notifiable diseases, United States

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1988		Cum. 1988
Anthrax Botuliam: Foodborne Infant Other Brucellosis (Upstate N.Y. 1) Cholera Congenital rubella syndrome Congenital syphilis, ages < 1 year Diphtheria	- 15 22 3 38 1 3 171 -	Leptospirosis Piague Poliomyelitis, Paralytic Paittacosis (Iowa 1, N.C. 1) Rabies, human Tetanus (N.C. 2) Trichinosis	19 7 - 52 - 29 38

*Because AIDS cases are not received weekly from all reporting areas, comparison of weekly figures may be misleading. 'Four of the 61 reported cases for this week were imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

		Aseptic	Encephalitis		0		н	Hepatitis (Viral), by type				
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious	Gond (Chv	rilian)	A	В	NA,NB	Unspeci- fied	Legionei- Iosis	Leprosy
	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	19,322	2,930	437	77	416,456	479,497	14,808	13,469	1,573	1,291	537	103
NEW ENGLAND	839	150	17	2	12,736	14,606	551	772	94	71	24	13
Maine	23	10	1	-	248	430	16	35	3	1	3	-
N.H.	19	19	1	1	164	250	35	46	7	4	3	-
VI. Maee	462	10	7	;	4 465	125 5 210	262	22	62	2	1	
R.I.		35	<u>'</u>		1.062	1,221	203	63	9		3	1
Conn.	269	18	3	•	6,712	7,261	169	129	7	13	-	-
MID. ATLANTIC	6,368	260	38	4	62,757	78,613	937	1,781	98	146	129	8
N.Y. City	3 427	104	20	3	26 303	41 279	400	4/0	44	103	19	
N.J.	1.558	47	5		9,213	10.045	152	389	33	26	20	í
Pa.	530	-	-	-	18,266	16,713	110	123	11	2	29	-
E.N. CENTRAL	1,425	400	108	11	67,119	70,551	976	1,450	138	73	110	1
Ohio	305	134	29	3	15,266	15,577	216	349	23	11	46	•
ind.	80	42	13	:	5,178	5,690	96	203	13	19	8	-
III. Mich	6/4	127	26	8	19,932	21,604	2/8	281	48	18	42	-
Wis.	255	22	11	:	4,954	8,420	150	168	19		13	1
W.N. CENTRAL	447	129	26	7	17.313	19,452	852	652	71	23	54	1
Minn.	88	24	2	3	2,337	3,035	69	87	15	3	2	-
lowa	25	19	8	•	1,311	1,856	34	63	11	1	13	-
Mo.	236	43	1	-	9,827	10,175	488	378	31	12	12	•
N. Dak. S. Dak	4	-	4	:	9/	184	4	6 2	2	4	1	-
Nebr	25	5	4	2	1.000	1.236	42	34	1	-	5	
Kans.	64	26	6	ī	2,410	2,609	209	81	9	3	7	1
S. ATLANTIC	3.367	673	66	26	121.928	125.529	1.304	2.794	236	181	91	1
Del.	40	13	2		1,787	2,016	24	81	6	2	8	-
Md.	358	72	5	3	12,111	14,027	183	422	22	13	13	1
D.C.	314	13	1	1	8,649	8,344	11	29	3	1	1	-
va. W.Ve	225	12	11	3	857	944	201	205	2	3	0	
N.C.	179	88	16	-	19,179	18,942	207	507	58		26	-
S.C.	105	11	-	1	10,027	10,253	30	334	8	5	13	-
Ga.	474	77	1		22,692	21,989	251	404	-9	3	12	-
	1,002	315		18	30,249	40,074	320	//0		40	12	-
E.S. CENTRAL	496	208	36	1	32,965	30,200	353	139	114	2	23	1
Tenn.	235	16	10		11.084	12,697	52	423	29	-	7	-
Ala.	122	107	16	2	10,444	11,647	20	205	36	5	4	1
Miss.	83	25	-	3	8,198	8,233	16	52	8	-	3	-
W.S. CENTRAL	1,593	371	48	3	46,238	54,340	1,710	1,122	120	332	14	19
Ark.	54	50	12	-	4,606	6,119	198	206	17	10	3	:
Okla.	207	32	4		4,263	6.024	326	117	30	21	7	
Tex.	1,249	275	29	2	28,062	32,375	1,102	735	72	290	-	18
MOUNTAIN	582	113	22	2	9,110	12.678	2.096	1.058	170	108	28	1
Mont.	9	2	•	-	294	353	25	35	9	3		-
Idaho	7	1	•	-	238	451	107	73	5	3	-	-
vvyo. Colo	222	1		-	2 012	283	144	10	3	- 52	2	;
N. Mex.	30		2	-	848	1.369	384	157	12	1	í	
Ariz.	169	34	8	1	3,289	4,367	1,067	418	52	31	12	-
Utah	46	14	4	1	360	392	219	88	28	14	3	-
Nev.	95	9	5	-	1,936	2,693	146	147	14	4	3	-
PACIFIC	4,205	626	76	16	46,290	67,473	5,941	3,021	532	350	64	58
VVasn. Oreg	246	•	6	4	3,931	5,206	1,305	484	117	38	14	3
Calif.	3,745	550	67	12	39 354	2,000 58 144	3.49F	2,006	354	297	47	1
Alaska	14	13	2		666	1,018	252	37	5	5	-+/	1
Hawaii	69	63	1	-	391	545	7	34	4	5	3	7
Guam	1	-	-	-	87	138	9	9		2	1	4
P.R.	769	28	2	1	844	1,313	28	158	26	29	:	3
V.I. Amer Samaa	25	•	-	-	265	157	1	5	2	-	•	•
C.N.M.I.	•	•	•	-	59	52	:	2	-	5	•	2
	-	-	-	•		-		2	-	4	-	1

TABLE III. Cases of specified notifiable diseases, United States, weeks ending August 13, 1988 and August 15, 1987 (32nd Week)

N: Not notifiable

•••••		Measles (Rubeola)					Menin-						2.1.11		
Reporting Area	Malaria	Indig	enous	impo	rted*	Total	gococcal Infections	Mu	mps		Pertussi	8		Rubella	3
	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	1988	Cum. 1988	Cum. 1987
UNITED STATES	493	56	1,804	5	210	3,149	1,965	33	3,279	89	1,375	1,275	4	141	273
NEW ENGLAND	40	-	80	-	50	251	171	2	104	3	110	59	3	5	1
Maine	2	-	7	-	-	3	7	-	-	-	11	7	-	-	1
N.H. Vt	1	-	66	-	44	151	20	1	95	-	33	12	3	3	-
Mass.	21	-	1	-	2	48	77	1	7		45	23	-	1	-
R.I.	5	-	-	-	-	2	21	-	-	2	6	1	-	1	-
Conn.	9	-	6	-	4	21	34	-	-	-	12	12	-	-	-
MID. ATLANTIC	67	18	739	-	39	555	189	1	268	10	81	143	-	12	11
Upstate N.Y.	22	1	16	-	16	39	93	-	72	4	45	105	-	2	9
N.J.	5	15	192	-	11	35	45	-	31		4	8		í	1
Pa.	6	1	491	-	10	38	1	1	73	5	30	30	-	2	-
E.N. CENTRAL	30	-	132	1	46	296	268	1	662	2	129	163		23	32
Ohio	6	-	2	1†	22	5	91	-	97	-	25	40	-	-	-
ina. Ili	2	:	57	-	15	125	21	1	66 220	1	59	13	-	- 10	-
Mich.	19	-	18	-	5	29	60	-	173	1	25	34		19	23
Wis.	2	-	•	-	4	137	37	-	87	-	18	63	-	-	
W.N. CENTRAL	13	-	11	-	1	224	76	1	117	2	84	72	-	-	1
Minn.	5	-	10	-	1	39	16	-	-	-	37	10	-	-	-
IOW8 Mo	1	:	1	-	-	182	- 29	-	31	-	19	16	-	-	1
N. Dak.		-		-	-	105	- 20	-	- 30		7	23	-	-	-
S. Dak.	-	-	-	-	-	-	3	-	1	1	5	3	-	-	-
Nebr. Kane	1	-	-	-	-		10	-	11	-	÷	1	-	-	-
		-	-	-	-		19		44		5	12	-	-	-
Del	/0	12	2/5		13	129	343	10	534	10	154	215	-	16	13
Md.	9	1	10	15	3	5	37	-	95		26	6	-	1	2
D.C.	11	-		-	-	1	7	-	200	-	-	-	-	-	-
Va. W.Ve	10	:	141	-	2	1	38	10	142	-	27	44	-	11	1
N.C.	11	-		-	1	5	59	-	38	3	40	33	-		1
S.C.	8	-	-	-	-	2	33	-	4	-	1	-	-	-	:
Ga.	17	11	110	-	÷	1	49	-	25	-	21	22	-	1	1
r 1d.				-	'	02	114	-	22	4	28	19	-	3	6
E.S. CENTRAL	7	1	52	-	-	2	183	2	383	3	37	24	-	-	3
Tenn.	-	-	-	-	-	-	108	1	195	1	16	1			2
Ala.	4	1	1	-	-	-	25	1	11	ż	14	12	-	-	
Miss.	3	-	16	-	-	2	11	N	N	-	1	5	-	-	-
W.S. CENTRAL	49	-	11	-	3	398	128	10	643	4	78	118	-	7	10
Anx. La	8	:	:	-	1	-	1/	3	246	1	9	. 9	-	3	2
Okla.	8	-	8	-	-	3	13	-	173	-	28	81	-	1	5
Tex.	32	-	3	-	2	395	61	3	142		27	-	-	3	3
MOUNTAIN	24	4	116	3	21	491	58	1	150	32	427	115	-	5	24
Mont.	4	4	4	3†	19	128	2	-	2	-	1	6	-	-	8
Idano Wyo	-	-	:	-		- 2		-	2		258	33	-	-	1
Colo.	9	-	112	-	1	9	14	-	28	-	14	39	-	1	
N. Mex.	1	-	•	-	-	317	10	N	N	5	19	8	-	-	-
Ariz. Liteb	3	-	-	-	-	31	15	1	102	20	113	23	-	-	4
Nev.	1	-	-	-	-	3	1	-	11	-	1		-	1	10
PACIFIC	193	21	388		37	803	549	5	418	23	275	366	,	72	170
Wash.	12	-:	2	-	-	39	48	2	40	2	56	62		/3	1/8
Oreg.	11	-	3	-		74	30	N	N	1	16	49	-	-	2
Calif. Alaska	163	21	380	•	29	686	450	3	348	19	152	131	1	53	112
Hawaii	5		3	-	8	4	15	-	11		45	119	-	- 20	61
Guam	-	-	-	-	1	2	-	-	2				_		4
P.R.	1	•	191	-	-	705	8	1	7	1	11	14	-	1	2
V.I.	-	-	-	-	-	-	:	-	28	-	-	-	-	-	-
Amer. Samoa C.N.M.I.	1	2	-		:	-	2	-	3	-	-	-	•	-	-
U.I.T.I.T.I.		-	-	-	-	•		-	2	-	-	-	-	-	-

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending August 13, 1988 and August 15, 1987 (32nd Week)

*For measles only, imported cases includes both out-of-state and international importations.

N: Not notifiable U: Unavailable [†]International [§]Out-of-state

Reporting Area	Syphilis (Primary &	(Civilian) Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	23,416	21,273	198	12,402	12,799	114	205	393	2,582
NEW ENGLAND	672	347	17	308	396	2	16	8	10
Maine	9	1	4	17	18	-		-	1
N.H.	6	3	3	6	12	-	-	-	3
Vt. Mase	3	1	2	177	220	-	1	-	-
R.I.	21	8	-	30	30		-	2	
Conn.	371	170	-	76	107	1	5	2	6
MID. ATLANTIC	4.718	4.021	30	2,255	2,181	-	38	15	330
Upstate N.Y.	304	142	15	329	325	-	5	8	17
N.Y. City	3,009	2,917	5	1,132	1,040	-	22	5	-
N.J. Pa	500 845	423	3	389	300 428	-		2	304
	070	555		4 00 4	1 400		22	-	004
Chio	6/8	559	22	265	279		23	27	90
Ind.	36	41		144	143	-	2		17
111.	326	300	1	574	647	•	11	2	16
Mich.	231	108	8	344	349	1	4	2	26
WIS.	20	42	•	0/	04	-			28
W.N. CENTRAL	143	104	23	325	389	56	- 4	62	321
Minn.	15	13	5	52 31	25	3	2	2	9/
Mo.	84	54	7	164	212	34	2	37	14
N. Dak.	1	-	2	5	6	-	•	-	68
S. Dak.		8	1	24	21	13	-	7	95
Kans	21	4	2	40	29	4		15	24
	0.000	7 050	16	2 606	0 770		24	100	925
5. ATLANTIC	8,622	/,253	10	2,090	2,772	1	24 -	122	37
Md.	472	368	3	271	243	-	1	19	209
D.C.	410	209	-	119	88	-	1		5
Va.	261	186	-	244	283	2	9	12	232
vv. va. N.C.	490	405	7	245	286	-	1	59	4
S.C.	463	485	2	300	283		-	14	56
Ga.	1,414	1,005	-	439	467	1	2	10	169
Fla.	5,033	4,542	3	1,005	1,021	-	10	5	59
E.S. CENTRAL	1,200	1,167	16	1,054	1,110	7	3	51	186
Ky. Tann	40 520	11	6	243	208	2	1	15	/2
Ala.	355	294	3	329	337	•	1	7	57
Miss.	285	383	-	173	188	1	1	4	2
W.S. CENTRAL	2.675	2.614	17	1,572	1,500	32	7	96	349
Ark.	147	163	1	170	172	21	-	15	60
La.	514	463	-	190	180		3	1	3
Ukla. Tev	98	92 1 896	10	1.061	1.003		4	70 10	24
100.	1,310	1,000		.,	.,	•	-	10	
MOUNTAIN	453	426	23	316	387	8	1	10	224
Idaho	2	5	3	11	23	-	-	ĭ	4
Wyo.	1	1	-	2	2	1	-	3	28
Colo.	72	74	3	27	101	5	3	-	13
Ariz	35	206	8	149	153		2	-	27
Utah	11	16	9	18	16	1	-		4
Nev.	221	81	-	32	19	-	-	-	-
PACIFIC	4,255	4,782	25	2,482	2,582	4	83	2	237
Wash.	116	82	3	128	164	-	5	•	-
Oreg.	178	163	1	93	64	-	6	1	-
Alaska	3,93 I R	4,525	Z I -	2,136	2,196	2	60	1	229
Hawaii	22	9	-	97	124	:	3	-	-
Guam	2	2		14	25	-	_	_	
P.R.	370	595	-	126	186	-	4	-	44
V.I.	1	4	-	4	2	-	-	-	-
Amer. Samoa	:	-	-	3	3	-	1	-	-
GIN.IVI.I.	1	-	-	17	-	-	-	-	-

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending August 13, 1988 and August 15, 1987 (32nd Week)

U: Unavailable

	All Causes, By Age (Years)								T	All Causes, By Age (Years)					
Reporting Area	AH		48.04				P&I**	Reporting Area	All				(a a a		P&I**
	Ages	-00	40-04	20-44	1-24		Total		Ages	≥05	40-64	25-44	1-24	<1	Total
NEW ENGLAND	652	445	127	50	13	16	57	S. ATLANTIC	1,414	846	283	152	58	73	50
Bridgeport Conn	108	92	42	23	3		21	Atlanta, Ga.	141	76	34	23	5	3	4
Cambridge, Mass.	25	19	5	1			2	Baltimore, Md.	340	222	69	33	8	8	14
Fall River, Mass.	23	17	6	-	-	-	-	Jacksonville, Fla	117	65	25	17	5	ŝ	3
Hartford, Conn.	46	27	14	4	•	1	3	Miami, Fla.	156	82	35	25	ğ	5	ĭ
LOWell, Mass.	32	26	3	1	2	:	2	Norfolk, Va.	70	39	9	7	5	10	3
New Bedford Mass	28	23	3	1	4			Richmond, Va.	74	43	13	8	4	6	2
New Haven, Conn.	39	26	ğ	3	:	1	3	Savannan, Ga. St Petersburg Fla	79	35	19	5	1	1	2
Providence, R.I.	51	39	6	3	2	1	1	Tampa, Fla.	74	44	20	5	2	1	6
Somerville, Mass.	7		-		-	:	-	Washington, D.C.	200	115	32	19	12	22	5
Waterbury Conn	36	40	6	2		2	2	Wilmington, Del.	27	20	4	2	1	-	1
Worcester, Mass.	61	42	13	4		2	8	E.S. CENTRAL	742	461	168	52	33	28	42
MID ATI ANTIC	2 601	1 664	511	285	85	55	110	Birmingham, Ala.	113	69	28	9	3	4	7
Albany, N.Y.	36	26	5	4		1		Chattanooga, Tenn.	49	28	13	3	3	2	1
Allentown, Pa.	11	6	3	2	-	-	-	Louisville, Kv.	100	66	21	ě	3	4	4
Buffalo, N.Y.	110	71	27	5	4	3	13	Memphis, Tenn.	169	104	30	16	10	ÿ	11
Lamgen, N.J. Elizabeth N.I	30	23	9	3	•	1	-	Mobile, Ala.	46	31	8	2	2	3	3
Erie, Pa.†	41	30	7	3	1	:	1	Montgomery, Ala.	59	37	16	4	1	1	2
Jersey City, N.J.S	59	38	11	7	1	2	ż		121	/4	31	4	•	4	
N.Y. City, N.Y.	1,400	881	254	196	50	19	49	W.S. CENTRAL	1,604	957	356	180	59	52	60
Newark, N.J. Peterson, N. I	30	13	9	5	2	1	2	Baton Rouge, La.	44	28	9	2 6	1	3	5
Philadelphia, Pa.	372	224	85	26	17	19	14	Corpus Christi, Tex.§	50	37	10	ž	i	-	i
Pittsburgh, Pa.†	72	46	17	5		4	3	Dallas, Tex.	183	108	35	26	9	5	5
Reading, Pa.	30	25	3	1	1	-	7	El Paso, Tex.	46	31	10	3	2	2	2
Rochester, N.Y.	102	72	20	7	2	1	9	Houston Tex 5	643	375	19	10	22	16	17
Screneciacy, N.T.	20	17	5	2	i	:	-	Little Rock, Ark.	70	42	16	5	5	2	3
Syracuse, N.Y.	110	74	27	3	4	2	6	New Orleans, La.	142	78	32	21	2	9	-
Trenton, N.J.	30	18	6	4	1	1	2	San Antonio, Tex.	132	73	31	16	5	7	13
Utica, N.Y.	24	21	3	:	-	•	-	Tulea Okla	111	15	20	5	2	2	3
TORKERS, IN. T.	44	39	- 4	1	-	-		MOLINITAIN	606	250	112	70	22	21	22
E.N. CENTRAL	2,349	1,517	509	1/6	68	/9	101	Albuquerque, N. Mex	K. 70	40	19	10	- 33	2	2
Canton, Ohio	20	15	5		-		3	Colo. Springs, Colo.	40	29	4	3	1	3	3
Chicago, III.§	564	362	125	45	10	22	16	Denver, Colo.	97	55	19	12	2	9	2
Cincinnati, Ohios	145	102	28	8	5	2	12	Las vegas, Nev.	100	5/	21	15	6	1	1
Cleveland, Ohio	153	96	40	12	5	11	4	Phoenix, Ariz.	116	64	20	14	11	7	3
Davton, Ohio	121	79	28	9	-	5	6	Pueblo, Colo.	28	19	2	6	1	-	2
Detroit, Mich.	316	191	61	41	15	8	11	Sait Lake City, Utah	52	27	12	3	3	7	:
Evansville, Ind.	56	44	.7	3	2	:	1	Lucson, Ariz.	85	55	15	6	7	2	3
Fort Wayne, Ind.	53	36	10	2	3	2	4	PACIFIC	1,849	1,174	344	207	64	53	118
Grand Rapids, Mich.	50	34	6	6	3	1	5	Berkeley, Calif.	75	3	11	1	5	5	18
Indianapolis, Ind.	144	83	34	11	4	12	2	Glendale, Calif.	29	25	2	ĭ	ĭ		
Madison, Wis.	26	15	6	3	1	1	1	Honolulu, Hawaii	77	54	16	3	2	2	10
Milwaukee, Wis.	154	117	25	8	2	2	9	Long Beach, Calif.	68	46	10	.8	2	2	5
Rockford III.	44	28	11	3	1	- 7	2	Los Angeles Calif.	502	311	13	13	23	5	23
South Bend, Ind.	44	30	10	ĩ	ż	1	2	Pasadena, Calif.	24	21	3		-		-
Toledo, Ohio	109	80	22	2	4	1	10	Portland, Oreg.	156	109	26	13	3	5	6
Youngstown, Ohio	50	32	12	3	-	3	-	Sacramento, Calif.	131	78	32	13	6	2	11
W.N. CENTRAL	777	546	150	37	19	25	33	San Diego, Calif.	163	108	2/	28	6	4	17
Des Moines, Iowa	53	38	12	1	1	1	3	San Jose, Calif.	157	94	36	20	2	5	13
Juluth, MINN. Kanese City, Kane	33	21	р Д	2	1	1		Seattle, Wash.	143	89	26	15	4	9	2
Kansas City, Kalis.	131	93	20	12	i	5	3	Spokane, Wash.	50	36	.7	4	1	2	4
Lincoln, Nebr.	36	26	8	-	1	1	4	Tacoma, Wash.	41	26	11	•	1	3	•
Minneapolis, Minn.	149	107	25	5	4	8	6	TOTAL 1	12,594**	7,969	2,561	1,209	432	412	601
Dmaha, Nebr.	105	75	18	5	3	4	8								
St. LOUIS, MO. St. Paul, Minn.	60	02 45	13	9		1	3								
Wichita, Kans.§	71	54	14	i	1	1	ž								

TABLE IV. Deaths in 121 U.S. cities,* week ending August 13, 1988 (32nd Week)

*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.

included. *Pneumonia and influenza. †Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks. †Trotal includes unknown ages. \$Data not available. Figures are estimates based on average of past available 4 weeks.

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Salmonella enteritidis – Continued

New Jersey. Forty-seven (60%) of 78 trainees interviewed reported having had onset of gastrointestinal illness from June 13 to June 16, 1988. Two were hospitalized and recovered; seven stool cultures were taken, and all yielded SE. Epidemiologic data implicated scrambled eggs served on June 11 and 12. In addition, culture of a pooled egg mixture obtained at the hotel yielded SE. Neither the clinical isolates nor the isolate from the eggs were lysine-positive. Since most SE isolates are lysine-positive, a relationship between the SE strains found in the patients and in the eggs seems probable. The implicated Grade A eggs were traced to a farm in Pennsylvania.

Livonia, New York. In late May 1988, an outbreak of gastrointestinal illness occurred among patrons of a restaurant in Livonia, New York. Twelve (38%) of 32 persons who attended a brunch on May 22 reported diarrhea, nausea, vomiting, or abdominal cramps. Stool cultures from four patients yielded SE. Egg omelets made from pooled Grade A eggs were the only food statistically associated with illness. Investigation did not identify improper food handling practices, such as cross-contamination or inadequate storage, which could have played a role in this outbreak. None of the food handlers were ill, and none had stool cultures that yielded *Salmonella*. The implicated Grade A eggs were traced to a Maryland farm.

Reported by: GC Taylor, MPH, Fort Monmouth; MA Meadows, LW Jargowsky, MPH, Monmouth County Dept of Health; K Pilot, MJ Teter, DO, J Brook, MD, ME Petrone, MD, KC Spitalny, MD, State Epidemiologist, New Jersey State Dept of Health. SB Spitz, MS, Monroe County Dept of Health; RJ Davin, J Ellison, Livingston County Dept of Health; SF Kondracki, JJ Guzewich, MPH, JK Fudala, MS, JG Debbie, DVM, DL Morse, MD, State Epidemiologist, New York State Dept of Health: Enteric Diseases Br, Div of Bacterial Diseases, Center for Infectious Diseases, CDC.

Editorial Note: A total of 6390 SE isolates were reported for 1987 (16% of total reported *Salmonella* isolates). SE is the second most common *Salmonella* serotype reported. National surveillance data for 1987 indicate continued high isolation rates of SE in the northeast, mid-Atlantic, and south Atlantic regions (Figure 1). Recent isolation rates of SE have also increased in the east north central, mountain, and Pacific regions of the country. The outbreaks described in this report confirm the





Salmonella enteritidis - Continued

continuing association between eggs and outbreaks of SE infections (1). Of the 19 outbreaks caused by SE with a known vehicle reported to CDC in 1987, 15 (79%) were associated with Grade A shell eggs. No vehicle of transmission was known for 11 other reported outbreaks of SE infections in 1987. An examination of data from 1973 to 1987 reveals that most outbreaks caused by SE occur during the summer months (Figure 2). Warm temperatures may provide opportunities for SE to multiply and survive in the eggs during production, transport, storage, or use.

Although food handling errors can contribute to outbreaks of *Salmonella* infections, the outbreaks in Fort Monmouth, New Jersey (ice cream), and Livonia, New York (egg omelet), demonstrate that SE infections can occur even when acceptable food preparation techniques have been used.

An SE control program is being developed by state health departments, poultry scientists, the egg industry, the U.S. Department of Agriculture, the Food and Drug Administration, and CDC. Long-term control of SE may depend on the elimination of infected flocks or use of pasteurized egg products. Proper handling and cooking of eggs can minimize the risk of salmonellosis (2); thorough cooking kills Salmonella.*

Clinicians are encouraged to report cases of salmonellosis to local and state health departments. *Salmonella* isolates can be serotyped by most state public health laboratories to aid in epidemiologic investigations.

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^{*}Further information about proper food preparation with eggs can be obtained through local county extension home economists or by calling the USDA Meat and Poultry Hotline (800) 535-4555.





Current Trends

Influenza - United States, 1987-88 Season

Influenza A(H3N2), the predominant type of influenza virus isolated in the United States during the 1987–88 season, exhibited antigenic drift from previous epidemic strains (1). Many of the isolates resembled two strains first recognized in China during 1987, A/Sichuan/2/87 and A/Shanghai/11/87. Outbreaks reported during 1987–88 in the United States that were associated with these viruses occurred in all age groups, including residents of nursing homes. Antigenic variants of influenza B also circulated during the 1987–88 season, with most isolates resembling B/Victoria/2/87 (2). The number of influenza B virus isolates increased late in the season when the first outbreaks associated with this virus were reported; at the same time, influenza A(H3N2) declined. Influenza A(H1N1) viruses similar to A/Taiwan/1/86, the predominant influenza virus during the 1986–87 season (3), were the least frequently isolated viruses during the 1987–88 season and were associated with only one possible outbreak, which occurred among college students. The number of influenza A(H1N1) virus isolates also increased late in the season.

Sources for surveillance of influenza were the same as for the 1986–87 season (3) with these exceptions:

- Sentinel physician surveillance network. The number of reporting physicians increased to 141. A subgroup of 40 physicians collected nasopharyngeal specimens from selected cases and immediately submitted those specimens for virus processing. Rapid culture confirmation techniques were used to identify and report positive results to the physicians within 24 hours of test results or 5–6 days of specimen collection. The culture confirmation technique identified the type of influenza virus but not the subtype of influenza A.
- 2. World Health Organization (WHO) collaborating laboratories. Fifty-three (instead of the previous 64) laboratories, based in state or local health departments, universities, or hospitals, reported by postcard the number of specimens tested and the number and type of influenza viruses isolated for each week from early October through mid-May. Data from the other WHO collaborating laboratories were reported through the Epidemiologic Surveillance Project (4).
- 3. Epidemiologic Surveillance Project (ESP). In this project, case reports of cultureconfirmed influenza were submitted electronically to CDC from state health departments in Georgia, Kentucky, North Carolina, South Carolina, Texas, and Vermont. All cases identified by WHO collaborating laboratories and other participating laboratories in these states were reported. Information reported for each case included patient age, county of residence, date of specimen collection, date of report to state, type of influenza virus identified, and, if known, the subtype for type A influenza viruses.

The first suspected outbreak of influenza A(H3N2) occurred in cruise ship passengers who were touring Alaska during August (5). In October, a probable outbreak of influenza A(H3N2) occurred among American tourists traveling in the Orient aboard a cruise ship (6). The first reported domestic outbreak of influenza-like illness occurred in November in preschool children in Colorado; influenza A(H3N2) was isolated from a specimen obtained from the index patient (7). Sporadic isolates of influenza B were also reported early in the season from Arizona, Hawaii, and

Influenza - Continued

Wisconsin (8). However, the first reported outbreak of influenza B occurred in February in a Connecticut nursing home. Most reported outbreaks of influenza A(H3N2) and influenza B occurred in nursing homes or other long-term-care settings.

According to reports by sentinel physicians, the mean percentage of total weekly patient visits associated with influenza-like illness was 4.8% (Figure 1). Sentinel physicians also reported each week whether an outbreak of influenza is occurring among their patients. Outbreaks were reported primarily during January and February by physicians in the western and central regions of the country and during February and March by physicians from the eastern regions.

Morbidity reports from state epidemiologists indicated that peak influenza activity occurred during February and early March (Figure 1). Widespread or regional outbreaks were reported in 44 states and the District of Columbia (Figure 2). Outbreaks in the western and central regions of the country were reported earlier than those in the eastern regions.

WHO collaborating laboratories tested 26,732 specimens for influenza viruses. Isolates were recovered from 2,532 (9.5%) of these specimens. Nineteen hundred (75%) of the isolates were influenza A(H3N2), 430 (17.0%) were influenza B, and 202 (8.0%) were influenza A(H1N1) (Figure 3). Isolation of influenza A(H3N2) peaked during February, while influenza B and influenza A(H1N1) peaked during late March (Figure 1). Sentinel physicians submitted an additional 420 specimens for testing; 119 (28.3%) of these were positive for influenza viruses. Of the positive specimens, 110 (92.4%) were type A, and nine (7.6%) were type B influenza.

Combining all laboratory reports, influenza A(H3N2) viruses were reported from 49 states and the District of Columbia; influenza B, from 26 states in all regions of the country and the District of Columbia; and influenza A(H1N1), from 19 states primarily in the eastern, central, and southern regions of the country.

The proportion of deaths associated with pneumonia and influenza (P&I) reported from 121 cities exceeded the epidemic threshold for 9 weeks, from the week ending February 20 through the week ending April 16 (Figure 4). Eighty-six percent of the P&I deaths reported occurred in persons \geq 65 years of age. The 1987–88 season was the fifth year in the last decade that influenza A(H3N2) predominated. In each of the 5 years, excess mortality associated with P&I has occurred.

Preliminary analysis of the data received through ESP indicates the relative proportions of influenza virus types reported through this system were similar to those reported on postcards by the other WHO collaborating laboratories. Of the 661 isolates reported through ESP, 508 (76.9%) were type A(H3N2), 94 (14.2%) were type B, 20 (3.0%) were type A(H1N1), and 39 (5.9%) were type A viruses, not subtyped. Of the ESP isolate reports, 354 (53.6%) were reported from Harris County, Texas, where special influenza studies are conducted by the Influenza Research Center at the Baylor College of Medicine. The mean age of patients from whom isolates were recovered was 27 years for influenza A(H3N2), 20 years for influenza A(H1N1), and 19 years for influenza B. The median number of days between specimen collection and the date the results of virus testing were reported to the state epidemiologist was 27 days. Most reports were then transmitted to CDC within 1 week.

Reported by: Participating state and territorial epidemiologists and state laboratory directors. WHO Collaborating Laboratories. Sentinel Physicians of the American Academy of Family Physicians. Participating Veterans Administration Hospitals. Letterman Army Medical Center, San Francisco, California. Hackensack Hospital, Hackensack, New Jersey. Strong Memorial Hospi-

Influenza – Continued

FIGURE 1. Indicators of influenza activity, by week – United States, October 1987–May 1988



*Reported to CDC by 141 physician members of the American Academy of Family Physicians. A patient with a temperature ≥38.7 C (100 F) and at least cough or sore throat was considered to have influenza-like illness.

[†]Reported to CDC by state and territorial epidemiologists who used the following categories: no cases, sporadically occurring cases, regional outbreaks (occurring in counties collectively constituting <50% of the state's population), or widespread outbreaks (occurring in counties collectively constituting ≥50% of the state's population).

^{\$}Reported to CDC by 53 WHO collaborating laboratories (not including military laboratories).

Influenza - Continued

tal, Rochester, New York. Vanderbilt Univ, Nashville, Tennessee. Influenza Research Center, Baylor College of Medicine, Houston; 5th Army Medical Laboratory, Fort Sam Houston; USAF School of Aerospace Medicine, Epidemiology Div. Brooks AFB, Texas. Div of Surveillance and Epidemiologic Studies, Epidemiology Program Office; WHO Collaborating Center for Influenza, Influenza Br, Div of Viral Diseases, Center for Infectious Diseases, CDC.

Editorial Note: During the 1986–87 season, influenza A(H1N1) was the most frequently isolated influenza virus. Since its reappearance in 1977, A(H1N1) has primarily been associated with morbidity in younger persons. In contrast, influenza A(H3N2) – the predominant strain during the 1987–88 season (3) – causes morbidity in all age groups and mortality in the elderly. In 1986–87, only 2.3% of all influenza isolates were from persons \geq 65 years of age, while in 1987–88, 20.7% of the influenza A(H3N2) isolates reported by WHO collaborating laboratories reporting through the postcard system were from persons in this age group (Table 1) (3). The excess mortality associated with P&I is consistent with an increased occurrence of influenza in the elderly (Figure 4).

The 1987–88 influenza epidemic was associated with strains that exhibited antigenic drift from the strain that had been included in the vaccine. However, because these variations were not recognized until the fall of 1987, the trivalent influenza vaccine could not be modified to include the new variant. As a result, the efficacy of the vaccine, at least in certain high-risk persons, may have been reduced.

Efforts to improve influenza control are emphasizing rapid detection and reporting of influenza viruses—including those circulating in the Far East—in time to consider incorporating these viruses into the influenza vaccine. In addition, surveillance in the United States augmented by laboratory support enhances the monitoring of influenza, often before outbreaks occur, and can contribute to influenza control by enabling the use of antiviral agents in locations where influenza A is circulating.

The ESP and Sentinel Physician Surveillance have expanded options for epidemiologic surveillance of influenza. The ESP for influenza surveillance was first operated during the 1987–88 influenza season and provided data not reported by the

FIGURE 2. Maximum level of influenza outbreak activity reported by state – United States, October 10, 1987–April 30, 1988



Influenza – Continued





Influenza – Continued

postcard system. Specimen collection dates and additional case-specific information permit more detailed epidemiologic analysis than the postcard reporting system, thereby enhancing surveillance of both morbidity and viral isolation. The results of the Sentinel Physician Surveillance Network, a pilot study in progress for several years, have demonstrated the feasibility of a relatively inexpensive method for rapid confirmation of influenza in specimens collected by family physicians and have provided prompt feedback to these physicians (9).





*Reported to CDC from 121 U.S. cities. P&I deaths include all deaths for which pneumonia is listed as a primary or underlying cause or for which influenza is listed on the death certificate. The predominant virus strain is shown above the peak of mortality for each epidemic season. The epidemic threshold for the 1987–88 influenza season was estimated at 1.645 standard deviations above the values projected on the basis of a periodic regression model applied to observed P&I deaths for the previous 5-year period but excluding the observations during influenza outbreaks.

TABLE	1. Specim	ens tes	ted an	d influ	ienza ty	pe A(H3N	2) viruses	isolated,	by age
group,	as reporte	d to C	DC by \	NHO (collabora	ting labo	ratories*	- United	States,
Octobe	r 1987–Ap	ril 1988				-			

Age group [†]	Spec tes	imens sted	Туре	Percent specimens	
(yrs)	No.	(%)	No.	(%)	positive
<20	12,475	(56.2)	764	(49.8)	6.1
20-64	6,999	(31.5)	451	(29.4)	6.4
>64	2,739	(12.3)	318	(20.7)	11.6
Total	22,213	(100.0)	1533	(100.0)	6.9

*These numbers do not include reports of isolates through the ESP.

[†]Age group was not specified for an additional 4,519 specimens tested. Influenza virus was isolated from 321 (7.1%) of these specimens; 248 (77.3%) of the isolates were influenza A(H3N2).

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Influenza – Continued

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FIGURE I. Reported measles cases - United States, Weeks 28-31, 1988

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The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on 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 public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: Editor, *Morbidity and Mortality Weekly Report*, Centers for Disease Control, Atlanta, Georgia 30333.

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