

# MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

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## National Drunk and Drugged Driving Prevention Month — December 1993

Persons who drive while impaired by alcohol or other drugs are a public health hazard to themselves and to others. Each year, alcohol-related crashes result in approximately 17,700 deaths in the United States. In addition, impaired driving is a leading cause of death among teenagers and young adults: more than one third of such fatalities occur among persons aged <25 years.

The injuries, disabilities, and deaths associated with impaired driving are preventable. Accordingly, December has been designated National Drunk and Drugged Driving Prevention Month by the National Drunk and Drugged Driving Prevention Month Coalition, a nationwide public/private sector coalition devoted to preventing crashes related to impaired driving. The theme of this year's campaign is "Let's Take a Stand! Friends Don't Let Friends Drive Drunk." Additional information about National Drunk and Drugged Driving Prevention Month is available from Tarry Hess, Office of Alcohol and State Programs (NTS-22), National Highway Traffic Safety Administration, 400 7th Street, SW, Washington, DC 20590; telephone (202) 366-6976 or from Carrie Hartshorne, Office of the Director, National Center for Injury Prevention and Control, CDC, 4770 Buford Highway, NE, Atlanta, GA 30341; telephone (404) 488-4690.

### *Health Objectives for the Nation*

## Reduction in Alcohol-Related Traffic Fatalities — United States, 1990–1992

Alcohol-related traffic crashes are a leading cause of unintentional injury deaths and a substantial contributor to health-care costs in the United States (1). Approximately 40% of persons will be involved in an alcohol-related crash during their lifetime (2). In 1992, alcohol was involved in an estimated 17,700 traffic fatalities and 355,000 traffic injuries (2,3). In 1990, alcohol-related crashes cost \$46.1 billion, including \$5.1 billion in medical expenses (4,5). This report summarizes data regarding alcohol-related traffic fatalities (ARTFs) from the National Highway Traffic Safety Administration's (NHTSA) Fatal Accident Reporting System (FARS) during 1982–1992.

*Alcohol-Related Traffic Fatalities — Continued*

NHTSA defines ARTFs as traffic deaths in which a driver, pedestrian, or bicyclist had a blood alcohol concentration (BAC)  $\geq 0.01$  g/dL. Each year, approximately 80% of ARTFs involve at least one driver or pedestrian with a BAC  $\geq 0.10$  g/dL, the legal level of intoxication in most states. NHTSA uses statistical models to estimate BACs for drivers and pedestrians where BAC test results are not available (6). In 1992, BAC test results were available for 47% of all drivers, pedestrians, and bicyclists involved in fatal crashes.

Data from FARS indicate that ARTFs, as a proportion of all traffic fatalities, decreased since 1982, especially during 1991 and 1992 (Table 1). From 1982 through 1992, the number of ARTFs decreased 30%, from 25,165 to 17,699, while ARTFs, as a proportion of all traffic fatalities, decreased from 57% to 45%.

From 1990 through 1992, ARTFs decreased 20%, from 22,084 to 17,699; in comparison, during the same period, nonalcohol-related traffic fatalities decreased 4%, from 22,515 to 21,536. In addition, ARTFs, as a percentage of all traffic fatalities, decreased from 50% in 1990 to 45% in 1992, the largest 2-year reduction since 1982, when uniformly reported data on ARTFs first became available.

Compared with 1990, the number of alcohol-involved driver fatalities in 1992 decreased at all BACs (Table 2). Reductions in the number of alcohol-involved driver fatalities were greater for drivers aged 15–20 years than for drivers aged  $\geq 21$  years. In addition, evidence of alcohol use decreased 22% among male drivers who died in crashes and 16% among female drivers. Data from 15 states\* that tested more than 85% of drivers who died in 1990 and 1992 indicate a 22% decrease in the number of drivers with BACs  $\geq 0.20$  g/dL, a 24% decrease in those with BACs 0.10–0.19 g/dL, and a 24% decrease in those with BACs 0.01–0.09 g/dL. Overall, ARTFs decreased 20% or more in 26 states and 15% or more in 33 states during 1990–1992 (Figure 1).

The number of alcohol-involved driver fatalities decreased during 1990–1992 across all categories of drivers and among traditionally hard-to-reach populations

\*California, Colorado, Connecticut, Delaware, Hawaii, Illinois, Maine, Montana, New Mexico, Oregon, Rhode Island, South Dakota, Washington, West Virginia, and Wisconsin.

**TABLE 1. Estimated number and percentage of total traffic fatalities, by blood alcohol concentration (BAC)\* — United States, 1982–1992**

Year	Total	BAC=0.00 g/dL		BAC $\geq 0.01$ g/dL		BAC $\geq 0.10$ g/dL	
		No.	(%)	No.	(%)	No.	(%)
1982	43,945	18,780	(42.7)	25,165	(57.3)	20,356	(46.3)
1983	42,589	18,943	(44.5)	23,646	(55.5)	19,174	(45.0)
1984	44,257	20,499	(46.3)	23,758	(53.7)	18,992	(42.9)
1985	43,825	21,109	(48.2)	22,716	(51.8)	18,111	(41.3)
1986	46,087	22,042	(47.8)	24,045	(52.2)	18,936	(41.1)
1987	46,390	22,749	(49.0)	23,641	(51.0)	18,529	(39.9)
1988	47,087	23,461	(49.8)	23,626	(50.2)	18,731	(39.8)
1989	45,582	23,178	(50.8)	22,404	(49.2)	17,862	(39.2)
1990	44,599	22,515	(50.5)	22,084	(49.5)	17,650	(39.6)
1991	41,508	21,621	(52.1)	19,887	(47.9)	15,928	(38.4)
1992	39,235	21,536	(54.9)	17,699	(45.1)	14,123	(36.0)

\*BAC distributions are estimates for drivers and nonoccupants involved in fatal crashes. Numbers of fatalities and drivers involved are rounded to the nearest whole number.

*Alcohol-Related Traffic Fatalities — Continued*

(e.g., drivers with high BACs [22%], drivers with previous convictions for impaired driving [18%], motorcyclists [30%], pickup-truck drivers [17%], and teenagers [34%]).

The decline in the number of ARTFs was greater for drivers than for pedestrians during 1990–1992. The number of alcohol-involved driver fatalities decreased 21%, while nonalcohol-involved driver fatalities decreased 5%. In contrast, alcohol-involved pedestrian fatalities decreased 13%, and nonalcohol-involved pedestrian fatalities decreased 16%.

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**Editorial Note:** The findings in this report indicate that the national health objectives for the year 2000 regarding ARTFs have been surpassed (7). Based on preliminary census data, the overall incidence of ARTFs has declined from the baseline of 9.8 per 100,000 persons in 1987 to 6.9 in 1992, below the goal of 8.5 (objective 4.1). The rate for persons aged 15–24 years declined from the baseline of 21.5 in 1987 to 14.1, also below the goal of 18.0 (objective 4.1b).

These findings suggest that current measures to reduce alcohol-impaired driving are successful and should continue. The number and percentage of ARTFs have declined since 1982, despite a 40% increase in vehicle miles traveled during that time. Effective measures have included prompt license suspension for persons who drive while intoxicated; lowering legally permissible BACs to 0.08 g/dL for adults and 0.02 g/dL for drivers aged <21 years; sobriety checkpoints; and public education, community awareness, and media campaigns about the dangers of alcohol-impaired driving. To further reduce ARTFs and nonfatal injuries, additional strategies should be considered, such as those outlined during the Surgeon General's Workshop on Drunk Driving (8) and in the national plan for injury prevention and control (9). Examples of these strategies include altering social norms to make alcohol-impaired driving

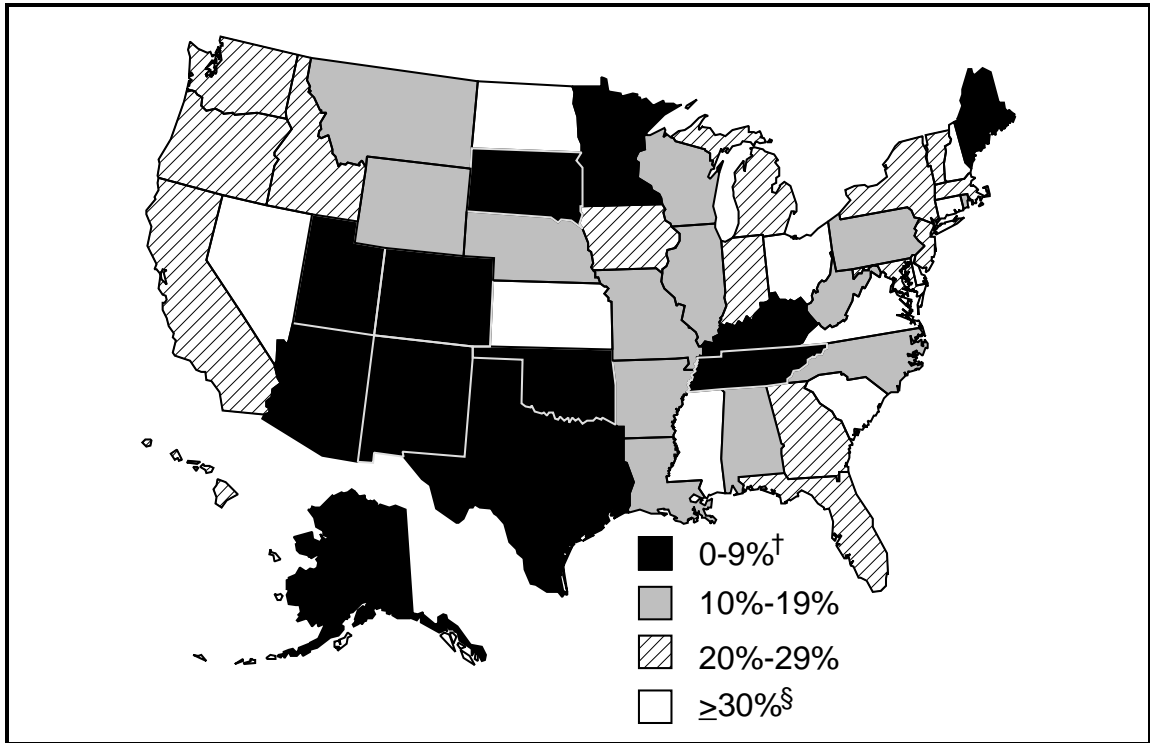
**TABLE 2. Estimated number of driver fatalities and percentage change from 1990 to 1992, by blood alcohol concentration (BAC)\* and driver age — United States**

Driver age (yrs)	BAC=0.00 g/dL			BAC ≥0.01 g/dL			BAC ≥0.10 g/dL		
	1990	1992	(% change)	1990	1992	(% change)	1990	1992	(% change)
0–14†	97	69	(–28.9)	9	10	(+11.1)	4	4	( 0 )
15–20	2,400	2,058	(–14.3)	1,653	1,095	(–33.8)	1,227	788	(–35.8)
21–24	1,310	1,254	(– 4.3)	1,957	1,544	(–21.1)	1,643	1,256	(–23.6)
25–34	2,518	2,275	(– 9.7)	4,137	3,140	(–24.1)	3,543	2,704	(–23.7)
35–44	1,863	1,829	(– 1.8)	2,195	1,892	(–13.8)	1,877	1,632	(–13.1)
45–54	1,458	1,495	(+ 2.5)	937	828	(–11.6)	777	674	(–13.3)
55–64	1,398	1,256	(–10.2)	509	432	(–15.1)	411	328	(–20.2)
≥65	2,877	2,999	(+ 4.2)	421	396	(– 5.9)	287	274	(– 4.5)
Unknown	3	5	(+40.0)	8	6	(–25.0)	8	6	(–25.0)
<b>Total</b>	<b>13,924</b>	<b>13,240</b>	<b>(– 4.9)</b>	<b>11,826</b>	<b>9,343</b>	<b>(–21.0)</b>	<b>9,777</b>	<b>7,666</b>	<b>(–21.6)</b>

\* BAC distributions are estimates for drivers and nonoccupants involved in fatal crashes. Numbers of fatalities and drivers involved are rounded to the nearest whole number.

† Although usually too young to drive legally, persons in this age group are included for completeness of the data set.

Source: Fatal Accident Reporting System, National Highway Traffic Safety Administration.

*Alcohol-Related Traffic Fatalities — Continued***FIGURE 1. Percentage decrease in alcohol-related traffic fatalities\*, by state — United States, 1990–1992**

\*Traffic deaths in which a driver, pedestrian, or bicyclist had a blood alcohol concentration  $\geq 0.01$  g/dL.

<sup>†</sup>Alaska, Colorado, and Utah reported increases in alcohol-related fatalities.

<sup>§</sup>Data from Mississippi are provisional.

Source: Fatal Accident Reporting System, National Highway Traffic Safety Administration.

socially unacceptable, limiting alcohol availability among underaged youth, implementing responsible alcohol service training for those who sell and serve alcohol, implementing early alcohol treatment and rehabilitation programs, offering alternative transportation programs for those of legal drinking age, and increasing the perception of risk for arrest for alcohol-impaired driving.

The public health impact of alcohol-impaired driving underscores the need for intensified preventive efforts by public health and traffic-safety agencies (10). Accordingly, the U.S. Department of Transportation has established for 1997 two major traffic-safety goals: 1) to reduce the proportion of ARTFs to 43% and 2) to increase safety-belt use to 75%. In response, NHTSA is developing a combined campaign to address both impaired driving and safety-belt nonuse through education, enforcement, and prevention activities. Attainment of these goals may save an estimated 2900 lives and \$5.8 billion annually, including nearly \$1 billion in health-care costs (5).

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*Alcohol-Related Traffic Fatalities — Continued*

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Current Trends**Update: Influenza Activity —  
United States and Europe, 1993–94 Season**

In collaboration with the World Health Organization (WHO) international collaborating laboratories and with state and local health departments in the United States, CDC conducts surveillance to monitor influenza activity and to detect antigenic changes in the circulating strains of influenza viruses. Laboratory surveillance indicates the predominance of influenza type A so far this season. This report summarizes influenza activity in the United States and Europe from mid-September through mid-November 1993.

**United States**

From September 19 through November 6, nearly all state and territorial epidemiologists reported either sporadic\* levels of influenza-like illness (ILI) or no activity. The first reports of regional activity associated with laboratory-confirmed outbreaks of influenza were from Wyoming and Montana for the week ending November 13 and from Idaho for the week ending November 20. From October 3 through November 13,

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\*Levels of activity are: 1) *sporadic*—sporadically occurring influenza-like illness (ILI) or culture-confirmed influenza, with no outbreaks detected; 2) *regional*—outbreaks of ILI or culture-confirmed influenza in counties with a combined population of less than 50% of the state's total population; and 3) *widespread*—outbreaks of ILI or culture-confirmed influenza in counties having a combined population of 50% or more of the state's total population.

*Influenza — Continued*

an average of less than 3% of all patient visits to family practitioners participating in the CDC sentinel physician surveillance system was for ILI.

From September 23 through November 23, 14 states (Alaska, California, Colorado, Connecticut, Hawaii, Louisiana, New Mexico, New York, North Carolina, Ohio, Oregon, South Dakota, Texas, and West Virginia) reported sporadic isolates of influenza type A. The outbreaks of culture-confirmed influenza A in Montana and Wyoming were the first since August–September, when three outbreaks of influenza A(H3N2) were reported in Louisiana (1). The outbreaks in Montana and Wyoming were associated with high absentee rates in two neighboring schools in those states. The first outbreak was recognized on November 4, when 18 (45%) of 40 students in an elementary school in Wyoming were absent. During November 4–11, the neighboring school in Montana (302 students in grades kindergarten through 12) reported daily absentee rates of 8%–14%. Seven of 10 nasopharyngeal specimens collected from students, household contacts, and others living in the community were positive for influenza type A by antigen detection as of November 30; two of these were confirmed by viral culture. In mid-November, Idaho reported outbreaks of ILI in schools in two southern counties; daily absentee rates were high (10%–44%), and influenza type A viruses were isolated from four students aged 9–15 years.

Of the influenza A viruses reported since late September, eight were subtyped; seven were identified as type A(H3N2) and one as type A(H1N1). Four of these viruses were further characterized at CDC and are antigenically related to the A/Beijing/32/92(H3N2) strain, the type A(H3N2) strain included in the 1993–94 influenza vaccine.

As of November 19, WHO collaborating laboratories in the United States have not reported influenza type B viruses.

**Europe**

In all European countries except the United Kingdom, influenza activity occurred at low levels from October 1 through mid-November. Influenza type A(H3N2) has been the predominant virus isolated. In addition, sporadic cases of influenza type B have been diagnosed by antigen detection. One isolate of influenza type A(H1N1) was reported from France.

During the week ending November 13, influenza activity in the United Kingdom became widespread. Influenza activity began early in October with outbreaks of ILI in Scotland and England. In Scotland, an outbreak that began among university students and staff extended into the surrounding community, and an outbreak in a residential home for the elderly affected both residents and staff. In England, outbreaks occurred in a residential home and in a boarding school. Influenza type A(H3N2) was isolated from ill persons in all four of these outbreaks. Outbreaks of ILI were reported in additional institutions, and general practitioners have reported increased levels of ILI in communities. The Central Public Health Laboratory in London analyzed 136 influenza isolates from sporadic cases and from outbreak-related cases; all were antigenically related to the A/Beijing/32/92(H3N2) virus.

In Finland, beginning the week ending October 30, outbreaks of ILI among children were associated with absentee rates of 50% in some elementary schools; influenza type A was diagnosed in two patients by antigen detection. In Sweden, the incidence of ILI was increasing among all age groups by the end of October; influenza type A(H3N2) was isolated from two patients, and influenza A was diagnosed by

*Influenza — Continued*

antigen detection in three. France, Czechoslovakia, and the Netherlands have reported either antigen detection of influenza type A or isolation of influenza type A(H3N2) from sporadic cases.

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**Editorial Note:** The findings in this report indicate that levels of influenza activity during November were higher than usually seen during this time of year. Reports of confirmed influenza outbreaks in early November illustrate the importance of prompt vaccination of unvaccinated high-risk persons before widespread activity occurs. Because protective levels of antibody develop within 2 weeks after vaccination, vaccine ideally should be administered at least 2 weeks before influenza outbreaks are expected. However, influenza vaccine should continue to be offered to high-risk persons after influenza activity is documented in a community. Because early viral surveillance has indicated the predominance of influenza type A, the antiviral drugs amantadine and rimantadine, which are effective against influenza type A viruses, can be used for prevention and treatment. When vaccine is administered after influenza type A has begun to circulate in a community, amantadine or rimantadine can be administered for 2 weeks after vaccination to provide protection until vaccine-induced antibody has developed (2,3).

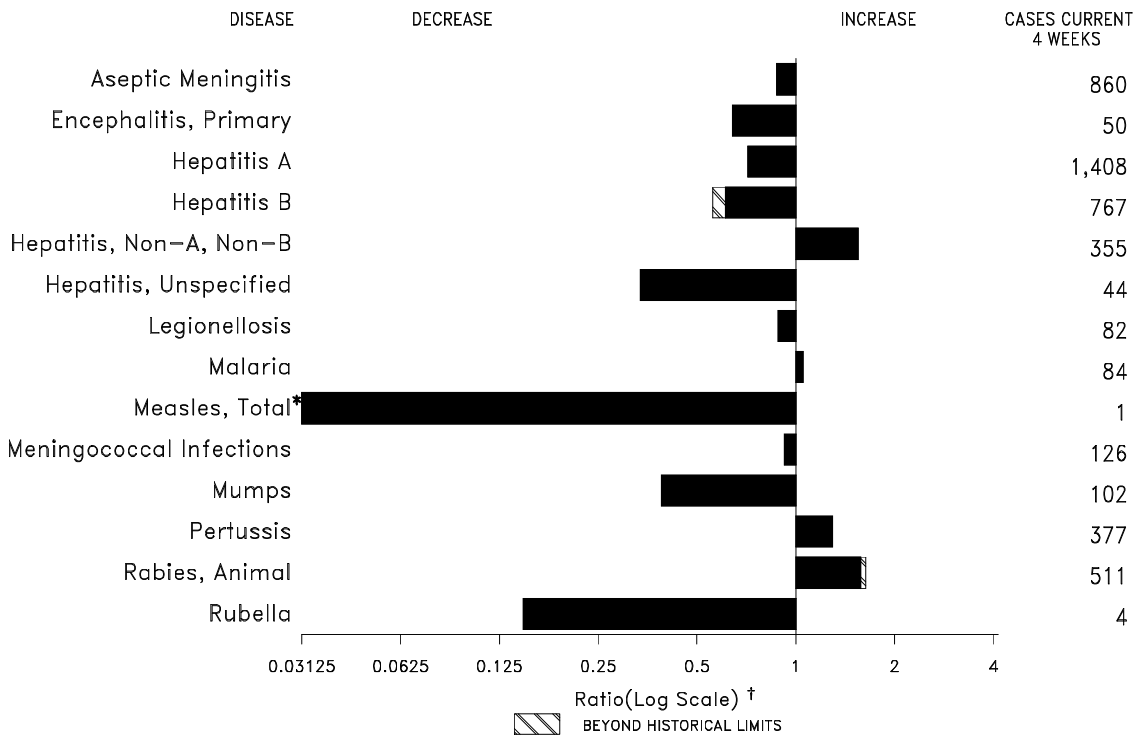
The increased circulation of influenza type A(H3N2) virus may increase the risk for outbreaks in nursing homes and facilities housing elderly persons; such outbreaks were reported during the latter half of the 1992–93 season and during August–September in Louisiana (1). Therefore, such facilities should now ensure that their residents have received influenza vaccine and also should develop contingency plans for rapid administration of amantadine or rimantadine in the event of suspected or confirmed influenza type A outbreaks.

Influenza surveillance findings are updated at least every other week throughout the influenza season, and summaries are available by computer to subscribers of the Public Health Network and to health-care providers and the public through the CDC Voice Information System, telephone (404) 332-4555.

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**FIGURE I. Notifiable disease reports, comparison of 4-week totals ending November 27, 1993, with historical data — United States**



\*The large apparent decrease in reported cases of measles (total) reflects dramatic fluctuations in the historical baseline. (Ratio (log scale) for week forty-seven is 0.00306).  
 † Ratio of current 4-week total to mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is based on the mean and two standard deviations of these 4-week totals.

**TABLE I. Summary — cases of specified notifiable diseases, United States, cumulative, week ending November 27, 1993 (47th Week)**

	Cum. 1993		Cum. 1993
AIDS*	92,056	Measles: imported	55
Anthrax	-	indigenous	222
Botulism: Foodborne	19	Plague	10
Infant	60	Poliomyelitis, Paralytic <sup>§</sup>	-
Other	2	Psittacosis	49
Brucellosis	82	Rabies, human	1
Cholera	17	Syphilis, primary & secondary	24,425
Congenital rubella syndrome	6	Syphilis, congenital, age < 1 year <sup>¶</sup>	1,493
Diphtheria	-	Tetanus	40
Encephalitis, post-infectious	148	Toxic shock syndrome	207
Gonorrhea	351,250	Trichinosis	13
<i>Haemophilus influenzae</i> (invasive disease) <sup>†</sup>	1,108	Tuberculosis	19,632
Hansen Disease	164	Tularemia	118
Leptospirosis	40	Typhoid fever	313
Lyme Disease	6,808	Typhus fever, tickborne (RMSF)	422

\*Updated monthly; last update November 28, 1993.  
 †Of 1058 cases of known age, 347 (33%) were reported among children less than 5 years of age.  
 §Two (2) cases of suspected poliomyelitis have been reported in 1993; 4 of the 5 suspected cases with onset in 1992 were confirmed; the confirmed cases were vaccine associated.  
 ¶Reports through second quarter of 1993.







**TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending November 27, 1993, and November 21, 1992 (47th Week)**

Reporting Area	Syphilis (Primary & Secondary)		Toxic-Shock Syndrome	Tuberculosis		Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1993	Cum. 1992	Cum. 1993	Cum. 1993	Cum. 1992	Cum. 1993	Cum. 1993	Cum. 1993	Cum. 1993
UNITED STATES	24,425	30,595	207	19,632	20,830	118	313	422	7,906
NEW ENGLAND	363	599	15	475	463	-	29	4	1,511
Maine	7	8	3	35	19	-	-	-	-
N.H.	29	37	5	9	17	-	2	-	128
Vt.	1	1	1	5	6	-	-	-	34
Mass.	115	295	5	263	258	-	21	4	637
R.I.	16	36	1	50	35	-	-	-	-
Conn.	195	222	-	113	128	-	6	-	712
MID. ATLANTIC	2,135	4,154	32	4,262	4,872	1	65	27	2,812
Upstate N.Y.	189	315	16	508	638	1	18	7	2,060
N.Y. City	1,067	2,319	1	2,442	2,807	-	26	-	-
N.J.	288	512	-	738	840	-	15	10	410
Pa.	591	1,008	15	574	587	-	6	10	342
E.N. CENTRAL	3,775	4,706	42	2,107	2,029	4	38	13	108
Ohio	1,050	763	11	288	291	-	8	8	6
Ind.	315	256	2	208	178	1	1	1	11
Ill.	1,456	2,168	8	1,107	1,052	2	21	2	23
Mich.	531	842	21	417	425	1	7	2	18
Wis.	423	677	-	87	83	-	1	-	50
W.N. CENTRAL	1,386	1,375	13	450	493	38	2	23	321
Minn.	62	90	2	62	138	-	-	1	40
Iowa	61	52	6	53	39	-	-	7	71
Mo.	1,140	1,047	2	227	216	15	2	11	22
N. Dak.	2	1	-	6	9	-	-	-	59
S. Dak.	1	-	-	12	20	17	-	3	41
Nebr.	10	24	-	18	22	3	-	-	11
Kans.	110	161	3	72	49	3	-	1	77
S. ATLANTIC	7,066	8,223	24	3,762	3,856	4	46	207	1,903
Del.	90	188	1	43	48	-	1	1	129
Md.	349	568	1	351	357	-	8	11	571
D.C.	298	353	-	147	100	-	-	-	16
Va.	606	670	7	386	312	-	6	11	365
W. Va.	13	17	-	68	83	-	-	6	85
N.C.	1,687	2,271	4	494	516	2	3	125	98
S.C.	868	1,097	-	354	374	-	-	10	150
Ga.	995	1,594	2	691	798	-	3	36	440
Fla.	2,160	1,465	9	1,228	1,268	2	25	7	49
E.S. CENTRAL	3,619	3,874	11	1,431	1,336	4	7	57	195
Ky.	317	157	3	343	353	1	2	11	19
Tenn.	973	1,089	4	424	386	2	2	32	72
Ala.	770	1,305	2	447	363	1	3	4	104
Miss.	1,559	1,323	2	217	234	-	-	10	-
W.S. CENTRAL	5,282	5,683	2	2,149	2,502	47	7	76	566
Ark.	682	819	-	166	194	27	-	7	40
La.	2,350	2,377	-	-	198	-	1	1	6
Okla.	339	407	2	145	149	16	1	64	62
Tex.	1,911	2,080	-	1,838	1,961	4	5	4	458
MOUNTAIN	215	315	14	482	531	14	10	15	165
Mont.	1	7	-	23	-	5	-	2	24
Idaho	-	1	2	12	22	-	-	-	6
Wyo.	8	5	-	6	-	3	-	10	22
Colo.	67	60	2	54	60	1	5	3	26
N. Mex.	24	39	1	59	71	2	2	-	9
Ariz.	93	154	1	212	235	-	2	-	59
Utah	10	8	6	28	65	2	1	-	4
Nev.	12	41	2	88	78	1	-	-	15
PACIFIC	584	1,666	54	4,514	4,748	6	109	-	325
Wash.	55	74	7	240	275	1	7	-	-
Oreg.	37	46	-	89	119	2	1	-	-
Calif.	478	1,534	47	3,905	4,052	3	98	-	304
Alaska	8	4	-	49	55	-	-	-	21
Hawaii	6	8	-	231	247	-	3	-	-
Guam	2	3	-	31	59	-	1	-	-
P.R.	459	302	-	185	200	-	-	-	43
V.I.	39	62	-	2	3	-	-	-	-
Amer. Samoa	-	-	-	2	-	-	1	-	-
C.N.M.I.	7	6	-	38	52	-	-	-	-

U: Unavailable



Epidemiologic Notes and Reports**Hypothermia-Related Deaths —  
Cook County, Illinois, November 1992–March 1993**

Hypothermia results from the inability of the body to maintain a sufficiently high body temperature and is defined clinically as the lowering of core body temperature to  $\leq 95$  F ( $\leq 35$  C) (1). Environmental hypothermia results from a combination of heat loss by convection (degree of wind exposure), conduction, and radiation to the surrounding air (2). Although hypothermia-related deaths are preventable, during 1979–1990, 9362 deaths in the United States were attributed to environmental hypothermia or excessive cold (*International Classification of Diseases, Ninth Revision* [ICD-9], codes E901.0, E901.8, and E901.9; excludes manmade cold [E901.1]).\* From November 1992 through March 1993, 22 hypothermia-related deaths were identified by the Cook County (Chicago), Illinois (1990 population: 5,105,067), medical examiner. This report summarizes information on those deaths and describes specific findings in four of the deaths.

**Summary findings.** Nineteen (86%) of the 22 persons who died were male, 12 (54%) were black, and six (27%) were aged  $\geq 65$  years. Eight (36%) of the decedents were homeless. Twelve (54%) had evidence of substance abuse at autopsy examination: nine (41%), ethanol intoxication; two (9%), neuroleptic intoxication; and one (5%), narcotics.

**Case 1.** In November 1992, a deceased 70-year-old man was found outside in a snow-covered area at the bottom of steps leading to his basement. The decedent had a history of alcohol abuse. An autopsy revealed a blood ethanol level of 0.19 g/dL. The decedent had not been seen for 36 hours before the discovery of his body, during which time outside temperatures ranged from 20 F to 35 F ( $-6.7$  C to  $1.7$  C). The underlying cause of death was hypertensive cardiovascular disease, with exposure to environmental cold secondary to alcohol intoxication being a contributing factor.

**Case 2.** In December 1992, a deceased 45-year-old, fully clothed, homeless man was found lying on a mattress in an abandoned building. The temperature at the time he was found was 30 F ( $-1.1$  C) with a wind chill factor of  $-20$  F ( $-29.0$  C). An autopsy revealed a blood ethanol level of 0.06 g/dL. Autopsy findings strongly suggested acute hemorrhagic pancreatitis, and death was attributed to acute hemorrhagic pancreatitis, with hypothermia due to cold exposure being a contributing factor.

**Case 3.** In January 1993, police discovered a deceased 64-year-old woman who had been lying on her bed inside her unheated residence. The body was decomposed and frozen. The decedent had a history of a leg injury and limited mobility. The average low temperature for the 5 days preceding her discovery was 29 F ( $-1.7$  C). A toxicology screen was negative; at autopsy, death was attributed to atherosclerotic cardiovascular disease, with hypothermia due to cold exposure being a contributing factor.

**Case 4.** In March 1993, a deceased 21-year-old man was found lying on the shore of Lake Michigan; the body was lightly clothed. Autopsy findings were unremarkable,

\*These data were obtained from the Compressed Mortality File (CMF), provided by CDC's National Center for Health Statistics, and have been prepared in accordance with the external cause-of-death codes from the ICD-9. The CMF contains information from death certificates filed in the 50 states and the District of Columbia.

*Hypothermia-Related Deaths — Continued*

except for a blood ethanol level of 0.19 g/dL. The underlying cause of death was hypothermia, and alcohol intoxication was considered a contributing factor.

*Reported by: R Dames, Office of the Medical Examiner, Cook County, Chicago. Health Studies Br, and Surveillance and Programs Br, Div of Environmental Hazards and Health Effects, National Center for Environmental Health, CDC.*

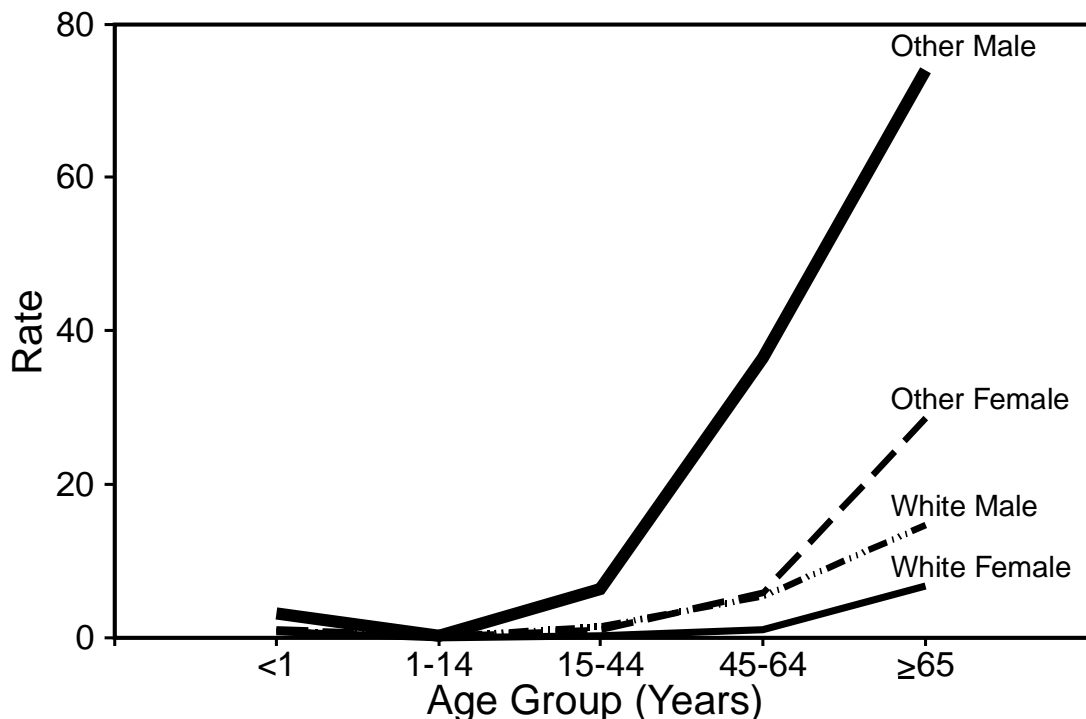
**Editorial Note:** Each year, approximately 780 persons in the United States die from exposure to cold<sup>†</sup>. Although these deaths are preventable, the number of deaths attributed to environmental hypothermia remained stable from 1979–1990 (annual age-adjusted death rates for hypothermia ranged from 2.2 to 4.3 per million population<sup>§</sup>).

National death rates for hypothermia during 1979–1990 varied by sex, age, and race (Figure 1). Most hypothermia-related deaths during that period occurred among men (6730 [72]%; average annual death rate: 4.9 per million population); the rate for men was 2.7 times that for women (1.8 per million population). Nearly half (4568 [49%]) of all hypothermia-related deaths occurred among persons aged  $\geq 65$  years. Differences in hypothermia mortality by race most likely reflect differences in factors

<sup>†</sup>Hypothermia-related deaths that were listed with an underlying cause of death as exposure to excessive cold (ICD-9 codes E901.0, E901.8, and E901.9 [excluding E901.1]).

<sup>§</sup>Age-adjusted death rates were standardized using the 1980 population census and are presented as deaths per million population.

**FIGURE 1. Average annual age-specific hypothermia death rates,\* by sex, and race<sup>†</sup> of decedent — United States, 1979–1990**



\* Underlying cause of death listed on death certificates as ICD-9 codes E901.0, E901.8, and E901.9 per million population.

<sup>†</sup>Data on race/ethnicity were collected only for "white," "black," and "other" races. In this analysis, blacks composed 76% of persons of all other races; rates for "other" races were too small for stable estimates.

*Hypothermia-Related Deaths — Continued*

related to socioeconomic status, such as nutrition or access to adequate shelter. The age-adjusted death rate for 1979–1990 for whites (2.4 per million population) was 3.6 times lower than the rate for persons of all other races.<sup>¶</sup> In addition, during this period the rate for white females (1.4 per million population) was 2.9 times lower than the rate for females of all other races (4.0 per million population), 2.4 times lower than the rate for white males (3.4 per million population), and 9.6 times lower than the rate for males of all other races (13.5 per million population).

Persons at excess risk for hypothermia include the elderly, the very young, and the homeless, and risk factors associated with hypothermia are alcohol use, neuroleptic medications, hypothyroidism, cerebrovascular disease, some forms of mental illness, and poverty (3–5). The most common cause of hypothermia is chronic, indoor cold stress that affects the immobile, elderly, chronically ill, or poor populations (3). The elderly are particularly vulnerable because of an impaired shivering mechanism, lower levels of protective fat, lower metabolic rates, limited mobility, chronic illnesses, and lack of perception of cold (3). The onset of hypothermia is often insidious with early manifestations of exposure including shivering, numbness, fatigue, poor coordination, slurred speech, impaired mentation, blueness or puffiness of the skin, and irrationality (2,6).

Measures to prevent hypothermia-related deaths include education of the public and health-care providers about heat preservation strategies and provision of outreach programs for identifying and sheltering persons at risk. During cold weather, particular attention should be given to increasing caloric intake, using insulated or layered clothing and headgear, and providing heated shelter with suitable relative humidity (3,7). In addition, persons who are active outdoors during cold weather should avoid fatigue, remain dry, prepare to take emergency shelter, carry fire-starting materials (waterproof matches and firestarters), and abstain from drinking alcoholic beverages (2,7).

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<sup>¶</sup>Data on race/ethnicity were collected only for “white,” “black,” and “other” races. In this analysis, blacks composed 76% of persons of all other races; rates for “other” races were too small for stable estimates.

## Epidemiologic Notes and Reports

### **Jin Bu Huan Toxicity in Adults — Los Angeles, 1993**

Jin Bu Huan (JBH) is a traditional Chinese herbal product used as a sedative and analgesic. During 1993, public health and health-care providers in Colorado reported three children with unintentional overdoses of JBH that caused central nervous system and respiratory depression with rapid onset of life-threatening bradycardia (1). Subsequently, the first cases of acute hepatitis attributed to use of JBH were diagnosed in three women in Los Angeles during July and August 1993. Patients 1 and 2 were referred to a Los Angeles hepatology clinic by their physicians; patient 3 was identified by patient 2. All three patients had purchased JBH at the same health-food store. This report summarizes the investigation of these cases.

#### **Patient 1**

In July, a 66-year-old woman sought care from her physician for fever, nausea, and fatigue of 2–3 days' duration. She was anicteric and had a palpable, nontender liver. Liver test results were elevated. Serologic tests for hepatitis A and hepatitis B indicated complete resolution of past infection; the test for hepatitis C infection was negative.

During May–July, she had used two JBH tablets at night, two to three times per week, for back pain and insomnia. In mid-July, she reportedly discontinued use of JBH because of her illness, and her alanine aminotransferase level was 786 U/L (normal: <40 U/L); aspartate aminotransferase (AST), 463 U/L (normal: <35 U/L); alkaline phosphatase, 176 U/L (normal: <108 U/L); and total bilirubin, 0.7 mg/dL (normal: <1.2 mg/dL). By mid-August, her liver test results had improved to slightly above normal; however, 4 weeks later, her alanine aminotransferase level was 961 U/L; AST, 595 U/L; alkaline phosphatase, 169 U/L; and total bilirubin, 0.4 mg/dL. Three weeks before these tests, the patient had resumed use of JBH (two tablets each night) for 1 week. She discontinued use of JBH a second time in early September. Three weeks later, she was asymptomatic, and liver enzymes had returned to normal.

#### **Patient 2**

In August, a 24-year-old woman sought care from her physician for fever, nausea, vomiting, fatigue, and pruritus of 2–3 days' duration; she had jaundice, excoriations of her limbs, and tender hepatomegaly. She was hospitalized for 5 days. Serologic tests were negative for hepatitis A, hepatitis B, and hepatitis C; cytomegalovirus; Epstein-Barr virus; and human immunodeficiency virus. Serum ceruloplasmin was normal. The peripheral white blood cell count was 10,800 per mm<sup>3</sup> with 7% eosinophils.

During June and July, she had used four JBH tablets at night, four times per week, for insomnia. She noted onset of jaundice in mid-July but continued use of JBH for 2 additional weeks. Two weeks after she reportedly discontinued use of JBH because of her illness, her alanine aminotransferase level was 1468 U/L; AST, 895 U/L; alkaline phosphatase, 133 U/L; and total bilirubin, 28.0 mg/dL (indirect bilirubin, 17 mg/dL [normal: <0.3 mg/dL]). Liver biopsy revealed acute hepatitis with many eosinophils in the portal tracts and cholestasis consistent with a drug reaction. To treat pruritus, cholestyramine therapy was initiated during her hospital stay; pruritus improved



*Jin Bu Huan Toxicity — Continued*

slightly. Nine weeks after discontinuing use of JBH, the patient was asymptomatic, and liver enzymes had returned to normal.

**Patient 3**

In August, a 45-year-old woman sought care from her physician for nausea, anorexia, fatigue, pruritus, and abdominal (right upper-quadrant) pain of 2–3 days' duration. Physical examination revealed tender hepatomegaly. Liver test results were elevated. Serologic tests for hepatitis A, hepatitis B, and hepatitis C were negative.

During May–August, she had used four JBH tablets at night, three to four times per week, for insomnia. During January–August, she had intermittently used another Chinese herbal product, Ma Huang (active ingredients include ephedrine and pseudoephedrine). She noticed onset of jaundice in mid-September, and her alanine aminotransferase level was 1308 U/L; AST, 1002 U/L; alkaline phosphatase, 225 U/L; and total bilirubin, 3.4 mg/dL. In late September, she reportedly discontinued use of both herbal products because of her illness. During the next 2 weeks, her symptoms resolved, except for mild pruritus. Nine weeks after discontinuing use of JBH, the patient was asymptomatic, and her liver test results had improved.

**Summary Findings**

No patient reported alcohol abuse. Ultrasound examination of the liver and biliary tract was normal for all patients; tests for antinuclear, antismooth muscle, and anti-mitochondrial antibodies were negative. All patients had normal prothrombin times throughout the course of their illnesses.

**Follow-Up Investigation**

The package insert for JBH tablets recommends use to treat pain and insomnia and indicates a composition of 70% starch and 30% levo-alkaloid from the plant *Polygala chinensis*. Tablets from patients 1 and 2 were analyzed at Colorado State University, using nuclear magnetic resonance and gas chromatography/mass spectroscopy; the tablets contained 36% (28.8 mg) levo-tetrahydropalmatine (L-THP), a chemical present in plants of the genera *Stephania* and *Corydalis* but not present in the genus *Polygala* (the plant of origin indicated on the package insert) (2,3). The remaining constituents were inert and no other plant materials or chemicals were identified. The Food and Drug Administration (FDA) is investigating this product.

*Reported by: GM Woolf, MD, SE Rojter, MD, FG Villamil, MD, JM Vierling, MD, Section of Hepatology, Cedars-Sinai Medical Center, Los Angeles; W Katkov, MD, St. Johns Hospital, Santa Monica. FR Stermitz, MD, JJ Beck, Dept of Chemistry and Agricultural Experiment Station, Colorado State Univ, Fort Collins. Center for Food Safety and Applied Nutrition, Food and Drug Administration. Health Studies Br, Div of Environmental Hazards and Health Effects, National Center for Environmental Health, CDC.*

**Editorial Note:** The ingredients identified in the JBH tablets from two of the three case-patients in this report were identical to those extracted from the JBH tablets retrieved from the previously reported cases of overdose in children (1). The findings in this report suggest that JBH (or one of its components) may be hepatotoxic; therefore, JBH should be avoided by persons with known liver disease.

The severity of the adverse health effects in these three cases underscores the potential health risks associated with use of herbal products. Based on surveys in Australia, England, the Netherlands, and New Zealand, the reported proportion of adult patients who seek consultations with and treatment by providers of

*Jin Bu Huan Toxicity — Continued*

unconventional therapy\* varies from 4% to 50% (4). In a recent survey of U.S. adults, 34% of respondents reported using unconventional therapy, and 3% reported using herbal products (5). In addition, during 1990, U.S. residents made an estimated 425 million office visits to providers of unconventional therapy; expenditures associated with such therapies totaled approximately \$13.7 billion (5). In North America, Chinese herbal products are sold as over-the-counter remedies for a variety of ailments. Although package inserts claim the safety of herbal products, previous reports have documented the risk of adverse health effects associated with use of these products (1,4-7).

Because the marketers of herbal products maintain that these products are dietary supplements rather than drugs, herbal products generally are not subject to standard testing for safety and efficacy. For the three cases in this report, the plant source and percentage of the active ingredient indicated on the JBH labels were incorrect. Such inaccuracies mislead consumers and health-care providers and can impede prompt and proper medical treatment. In addition, consumers should be warned that the term "natural" on a label does not ensure product safety (8).

Reporting systems have not been available to collect data about adverse reactions to dietary supplements, including botanical products. However, FDA's newly implemented MEDWATCH program, which monitors reports of adverse reactions to FDA-regulated products, may enhance surveillance because it specifically requests reports of adverse reactions to dietary supplements (9). National surveillance programs such as this can assist in identifying and monitoring the adverse health effects of JBH and other herbal products and dietary supplements. To report cases of serious adverse reactions to dietary supplements, health-care providers should contact MEDWATCH (telephone [800] 332-1088) to request a reporting form.

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\*Interventions neither taught widely in medical schools nor generally available in hospitals.

## Quarterly Table Reporting Alcohol Involvement in Fatal Motor-Vehicle Crashes

The following table reports alcohol involvement in fatal motor-vehicle crashes in the United States for October–December 1992. This table, published quarterly in *MMWR*, focuses attention on the impact of alcohol use on highway safety.

A fatal crash is considered alcohol-related by the National Highway Traffic Safety Administration (NHTSA) if either a driver or nonoccupant (e.g., pedestrian) had a blood alcohol concentration (BAC) of  $\geq 0.01$  g/dL in a police-reported traffic crash. Those with a BAC  $\geq 0.10$  g/dL (the legal level of intoxication in most states) are considered intoxicated. Because BACs are not available for all persons in fatal crashes, NHTSA estimates the number of alcohol-related traffic fatalities based on a discriminant analysis of information from all cases for which driver or nonoccupant BAC data are available. There may be seasonal trends associated with these data.

### Estimated number and percentage of total traffic fatalities\* and drivers involved in fatal crashes, by age and blood alcohol concentration (BAC) level — United States, October–December, 1992

Age group (yrs)	No. fatalities <sup>§</sup>	Fatalities, by BAC <sup>†</sup>					
		BAC=0.00 g/dL		BAC=0.01–0.09 g/dL		BAC $\geq 0.10$ g/dL	
		No.	(%)	No.	(%)	No.	(%)
0–14	579	438	(75.7)	44	( 7.6)	97	(16.7)
15–20	1,512	902	(59.7)	192	(12.7)	418	(27.7)
21–24	1,119	451	(40.3)	136	(12.2)	532	(47.5)
25–34	1,958	781	(39.9)	175	( 8.9)	1,002	(51.2)
35–64	3,299	1,733	(52.5)	248	( 7.5)	1,318	(40.0)
$\geq 65$	1,861	1,537	(82.6)	106	( 5.7)	218	(11.7)
<b>Total</b>	<b>10,328</b>	<b>5,842</b>	<b>(56.6)</b>	<b>901</b>	<b>( 8.7)</b>	<b>3,584</b>	<b>(34.7)</b>

Age group (yrs)	No. drivers <sup>§</sup>	Drivers, <sup>¶</sup> by BAC <sup>**</sup>					
		BAC=0.00 g/dL		BAC=0.01–0.09 g/dL		BAC $\geq 0.10$ g/dL	
		No.	(%)	No.	(%)	No.	(%)
0–14 <sup>††</sup>	34	28	(81.5)	4	(10.5)	3	( 8.0)
15–20	1,907	1,452	(76.1)	162	( 8.5)	293	(15.4)
21–24	1,710	1,043	(61.0)	163	( 9.5)	504	(29.4)
25–34	3,396	2,206	(64.9)	230	( 6.8)	960	(28.3)
35–64	5,161	3,993	(77.4)	242	( 4.7)	926	(17.9)
$\geq 65$	1,489	1,371	(92.0)	34	( 2.3)	84	( 5.7)
<b>Total</b>	<b>13,697</b>	<b>10,093</b>	<b>(73.7)</b>	<b>835</b>	<b>( 6.1)</b>	<b>2,770</b>	<b>(20.2)</b>

\*Fatalities include all occupants and nonoccupants who died within 30 days of a motor-vehicle crash on a public roadway.

<sup>†</sup>BAC distributions are estimates for drivers and nonoccupants involved in fatal crashes. Numbers of fatalities are rounded to the nearest whole number.

<sup>§</sup>Includes only those for whom age is known.

<sup>¶</sup>Driver may or may not have been killed.

\*\*BAC distributions are estimates for drivers involved in fatal crashes. Numbers of drivers are rounded to the nearest whole number.

<sup>††</sup>Although usually too young to drive legally, persons in this age group are included for completeness of the data set.

Source: Fatal Accident Reporting System, National Highway Traffic Safety Administration.

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