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Serum Cross-Reactive Antibody Response to a Novel Influenza A (H1N1) Virus After Vaccination with Seasonal Influenza Vaccine

As of May 19, 2009, a total of 5,469 confirmed or probable cases* of human infection with a novel influenza A (H1N1) virus had been documented in 47 states and the District of Columbia (1,2). In addition, the virus had spread to 41 countries (3), with a total of 4,774 cases reported in countries outside the United States. Because producing a novel influenza A (H1N1) virus vaccine will take several months (4), determining whether receipt of seasonal influenza vaccine might offer any protection against the novel influenza A (H1N1) virus is important. Therefore, using stored serum specimens collected during previous vaccine studies, CDC assessed the level of cross-reactive antibody to the novel influenza A (H1N1) virus in cohorts of children and adults before and after they had been vaccinated with the 2005–06, 2006–07, 2007–08, or 2008–09 influenza season vaccines. The results indicated that before vaccination, no cross-reactive antibody to the novel influenza A (H1N1) virus existed among children. Among adults, before vaccination, cross-reactive antibody was detected in 6%–9% of those aged 18–64 years and in 33% of those aged >60 years. Previous vaccination of children with any of four seasonal trivalent, inactivated influenza vaccines (TIV) or with live, attenuated influenza vaccine (LAIV) did not elicit a cross-reactive antibody response to the novel influenza A (H1N1) virus. Among adults, vaccination with seasonal TIV resulted in a twofold increase in cross-reactive antibody response to the novel influenza A (H1N1) virus among those aged 18–64 years, compared with a twelvefold to nineteenfold increase in cross-reactive antibody response to the seasonal H1N1 strain; no increase in cross-reactive antibody response to the novel influenza A (H1N1) virus was observed among adults aged >60 years. These data suggest that receipt of recent (2005–2009)

seasonal influenza vaccines is unlikely to elicit a protective antibody response to the novel influenza A (H1N1) virus.

Serum specimens were provided to CDC from academic, government, and industry partners for use as part of the public health response to the emergence of the novel influenza A (H1N1) virus. The specimens had been collected from healthy human participants, with written, informed consent. All participants had been vaccinated either 1) intramuscularly with licensed TIV developed for the northern hemisphere 2005–06, 2006–07, 2007–08, or 2008–09 influenza seasons or 2) intranasally with licensed LAIV developed for the northern hemisphere 2005–06 or 2006–07 influenza seasons. The serum specimens were grouped for influenza serology testing by the age of participants and formulation of the vaccines.

Microneutralization (MN) and hemagglutination inhibition (HI) assays were performed at CDC, according to standard MN and HI procedures (5,6). As with vaccine production, the seasonal influenza A (H1N1) viruses used in this study (A/New Caledonia/20/1999 [2005–06 and

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2006–07], A/Solomon Islands/3/2006 [2007–08], and A/Brisbane/59/2007 [2008–09]) were propagated in embryonated chicken eggs. The novel influenza A (H1N1) virus used in the study was A/California/04/2009, which was grown in Madin-Darby canine kidney cells. All procedures were performed in a biosafety level 2 laboratory using biosafety level 3 practices.[†] The HI assay was performed using 0.5% turkey red blood cells. Serum specimens were treated with receptor-destroying enzymes. Sera containing nonspecific agglutinins were heme-adsorbed and tested at an initial dilution of 1:10. For the MN assay, serum specimens were heat inactivated (at 133°F [56°C], for 30 minutes) and tested at an initial dilution of 1:10. For calculation of geometric mean titer (GMT) estimates, a titer of <10 was assigned a value of 5, and a titer of ≥1280 was assigned a value of 1280. Statistical significance was determined using a paired t-test.

An initial comparison between the HI and MN assays was made for panels of sera from children aged 6 months to 9 years (n = 28), adults aged 18–59 years (n = 30), and adults aged >60 years (n = 42). Although the estimated correlation between HI and MN titers was high (r = 0.82) for the seasonal vaccine strains, the MN assay generally yielded higher titers and detected more seroconversions (i.e., fourfold or greater increases in antibody titers) to A/California/04/2009 than the HI assay. Therefore, the MN assay was used to assess the level of cross-reactive antibody to A/California/04/2009 in populations before and after vaccination with seasonal influenza vaccines. Although serum HI antibody titers of 40 are associated with at least a 50% reduction in risk for influenza infection or disease in populations (7), no such correlate of protection exists for MN antibody titers. Therefore, a linear regression model was used to predict the MN titer for seasonal influenza A (H1N1) viruses that corresponded to an HI titer of 40 and to measure titer achievement against the seasonal vaccine strain and the novel influenza A (H1N1) virus. In the pediatric population, an HI titer of 40 corresponded to an MN titer of 40, whereas in the adult population the corresponding MN titer was ≥160.

Among 79 children ranging in age from 6 months to 9 years, little evidence was found of prevaccination cross-reactive antibodies to A/California/04/2009 (Table 1). In addition, after vaccination with seasonal TIV, no seroconversions to A/California/04/2009 virus were detected, whereas seroconversions to the seasonal vaccine strains were detected in 67%–100% of children. Children vaccinated with LAIV also had no seroconversions to the A/California/04/2009 virus.

[†] Biosafety level information is available at <http://www.cdc.gov/od/ohs/biosfty/bmbl5/bmbl5toc.htm>.

TABLE 1. Cross-reactive microneutralization (MN) antibody response to novel influenza A (H1N1) virus* in pediatric recipients (aged 6 months–9 years) of seasonal influenza vaccines

Vaccine	Influenza season	Influenza virus	Age group	No.	% with fourfold or greater increase in antibody titer [†]	% with MN titer of ≥ 40 [§]		Geometric mean titer (GMT) [¶]		
						Prevac-cination	Postvac-cination	Prevaccination (95% CI ^{**})	Postvaccination (95% CI)	Postvac-cination to prevaccina-tion ratio
TIV ^{††}	2005–2007 ^{§§}	A/New Caledonia/20/1999	6 mos–9 yrs	33	67	42	94	31 (21–46)	255 (172–378)	8
		A/California/04/2009			0	0	5 (4–6)	6 (6–7)	1	
	2007–08	A/Solomon Is/3/2006	5–9 yrs	13	85	54	100	42 (22–80)	575 (303–1093)	14
		A/California/04/2009			0	8	8	10 (7–15)	12 (8–17)	1
	2008–09	A/Brisbane/59/2007	6 mos–3 yrs	9	100	0	100	5 (4–7)	285 (202–402)	57
		A/California/04/2009			0	0	0	5 (—)	5 (—)	1
LAIV ^{¶¶}	2005–2007 ^{§§}	A/New Caledonia/20/1999	6 mos–9 yrs	24	25	46	79	33 (17–63)	73 (38–139)	2
		A/California/04/2009			0	0	4	5 (4–6)	6 (5–7)	1

* A/California/04/2009.

[†] A fourfold or greater increase in antibody titer indicates seroconversion (a response to the vaccine).[§] A linear regression model was used to predict the MN titer for seasonal H1N1 viruses that corresponded to a hemagglutination inhibition (HI) antibody titer of 40. (Serum HI antibody titers of 40 are associated with at least a 50% decrease in risk for influenza infection or disease [7]). In pediatric populations, an HI titer of 40 corresponds with an MN titer of 40.[¶] A titer of 1280 was used for all samples with a titer of ≥ 1280 . The dilution of sera in the first well is based on the combination of a 1:10 serum dilution with an equal volume of diluted virus for a final serum dilution referred to as 1:10. In the statistical models, study participants were treated as random effects sampled from a larger population of study participants, and duplicate samples were treated as random effects nested within each study participant.^{**} Confidence interval.^{††} Trivalent, inactivated influenza vaccine.^{§§} 2005–06 and 2006–07 influenza seasons.^{¶¶} Live, attenuated influenza vaccine.

Consistent with previous reports (4), vaccination of adults with seasonal TIV resulted in seroconversion to the seasonal influenza A (H1N1) vaccine strain in 74% of adults aged 18–64 years, 78% of adults aged 18–40 years, and 54% of adults aged >60 years (Table 2). In contrast, seroconversion to the A/California/04/2009 virus was detected in 19% of adults aged 18–64 years and 3% of adults aged >60 years who received the 2007–08 vaccine and in 12% of adults aged 18–40 years who received the 2008–09 vaccine. Compared with responses to the seasonal influenza A (H1N1) vaccine virus, postvaccination to prevaccination GMT ratios for the response to A/California/04/2009 virus were fivefold to tenfold lower among all adults. However, 6% of adults aged 18–40 years, 9% of adults 18–64 years, and 33% of adults aged >60 years had prevaccination MN titers of ≥ 160 . After vaccination with seasonal vaccine, 7% of adults aged 18–40 years, 25% of adults aged 18–64 years, and 43% of adults aged >60 years had postvaccination titers of ≥ 160 to A/California/04/2009. The prevaccination GMT of adults aged >60 years against the novel 2009 H1N1 strain was significantly higher than against the seasonal 2007–08 H1N1 vaccine component ($p < 0.001$).

Reported by: J Katz, PhD, K Hancock, PhD, V Veguilla, MPH, W Zhong, PhD, XH Lu, MD, H Sun, MD, E Butler, MPH, L Dong, MD, PhD, F Liu, MD, PhD, ZN Li, MD, PhD, J DeVos, MPH, P Gargiullo, PhD, N Cox, PhD, Influenza Div, National Center for Immunization and Respiratory Diseases, Coordinating Center for Infectious Diseases, CDC.

Editorial Note: The results in this report suggest that vaccination with recent (2005–2009) seasonal influenza vaccines is unlikely to provide protection against the novel influenza A (H1N1) virus. Although vaccination of adults with seasonal TIV generally resulted in a small increase in antibodies against the novel influenza A (H1N1) virus, whether such levels of cross-reactive antibody provide any protection against infection with novel influenza A (H1N1) virus is unknown. These results are consistent with the substantial degree of genetic divergence of the novel influenza A (H1N1) virus of swine origin from recent seasonal human H1N1 viruses; A/California/04/09 shares only 72%–73% amino acid identity in the HA1 portion of the hemagglutinin molecule with the seasonal viruses used in this study. For comparison, the amino acid sequence identity in the HA1 portion among seasonal vaccine strains used in this study is 97%–98%.

Although the number of sera from children tested in this analysis was small, results indicate that U.S. children are largely serologically naïve to the novel influenza A (H1N1) virus and that vaccination with seasonal TIV or LAIV does not elicit any measurable level of cross-reactive antibody to the novel virus. Results among adults suggest that some degree of preexisting immunity to the novel H1N1 strains exists, especially among adults aged >60 years. One possible explanation is that some adults in this age group have had previous exposure, either through infection or vaccination, to an influenza A (H1N1) virus that is genetically and antigenically more closely related

TABLE 2. Cross-reactive microneutralization (MN) antibody response to novel influenza A (H1N1) virus* in adult recipients of seasonal influenza vaccines

Vaccine	Influenza season	Influenza virus	Age group (yrs)	No.	% with fourfold or greater increase in antibody titer†	% with MN titer of ≥160§		Geometric mean titer (GMT)¶		
						Prevaccination	Postvaccination	Prevaccination (95% CI)**	Postvaccination (95% CI)	Postvaccination to prevaccination ratio
TIV††	2007–08	A/Solomon Is/3/2006	18–64	134	74	28	92	48 (40–59)	561 (462–682)	12
		A/California/04/2009			19	9	25	28 (23–34)	53 (43–66)	2
	2008–09	A/Brisbane/59/2007	18–40	83	78	20	88	29 (22–38)	546 (418–713)	19
		A/California/04/2009			12	6	7	11 (9–14)	21 (16–26)	2
	2007–08	A/Solomon Is/3/2006	>60	63	54	14	54	31 (22–42)	143 (105–194)	5
		A/California/04/2009			3	33	43	92 (71–121)	97 (74–127)	1

* A/California/04/2009.

† A fourfold or greater increase in antibody titer indicates seroconversion (a response to the vaccine).

§ A linear regression model was used to predict the MN titer for seasonal H1N1 viruses that corresponded to a hemagglutination inhibition (HI) antibody titer of 40. (Serum HI antibody titers of 40 are associated with at least a 50% decrease in risk for influenza infection or disease [7]). In adult populations, an HI titer of 40 corresponds with an MN titer of ≥160.

¶ A titer of 1280 was used for all samples with a titer of ≥1280. The dilution of sera in the first well is based on the combination of a 1:10 serum dilution with an equal volume of diluted virus for a final serum dilution referred to as 1:10. In the statistical models, study participants were treated as random effects sampled from a larger population of study participants, and duplicate samples were treated as random effects nested within each study participant.

** Confidence interval.

†† Trivalent, inactivated influenza vaccine.

to the novel influenza A (H1N1) virus than are contemporary seasonal H1N1 strains. Ongoing assessment of the cross-reactive antibody response among persons in different age groups might identify a particular age group that would allow further clarification of the cross-reactive serologic response. Development of a strain-specific vaccine against the novel influenza A (H1N1) virus is needed for optimal protection against the virus among persons of all ages.

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Federal and State Cigarette Excise Taxes – United States, 1995–2009

On April 1, 2009, the largest federal cigarette excise tax increase in history went into effect, bringing the combined federal and average state excise tax for cigarettes to \$2.21 per pack and achieving the *Healthy People 2010* (HP2010) objective (27-21a) to increase the combined federal and average state cigarette excise tax to at least \$2 per pack (1). This report summarizes changes in the federal excise tax, as well as state excise taxes for all 50 states and the District of Columbia (DC) from December 31, 1995 to April 1, 2009.* The findings indicate that the federal excise tax increased from 24 cents per pack in 1995 to \$1.01 per pack in 2009, and the average state excise tax increased from 32.7 cents per pack to \$1.20 per pack during the same period.† These increases represent a 321% increase in the federal excise tax and a 267% increase in the average state excise tax since 1995. Price increases should be combined with other evidence-based policy and clinical

* For this report, DC is included among results for states.

† The federal tax of \$50.33 for cigarettes is levied per 1,000 cigarettes. When calculated per pack of 20 cigarettes, this is \$1.0066 per pack. For this study, this fractional tax is referred to as \$1.01 per pack.

interventions to meet HP2010 objectives to decrease smoking prevalence and reduce the burden from smoking-attributable death and disease.

Cigarettes and other tobacco products are taxed by federal, state, and local governments in various ways, including excise taxes, which are levied per unit, such as per pack of 20 cigarettes (2). Federal and state excise tax rates are set by legislation, are contained in federal and state statutes, and typically are collected before the point of sale (i.e., from manufacturers, wholesalers, or distributors), as denoted by a tax stamp.

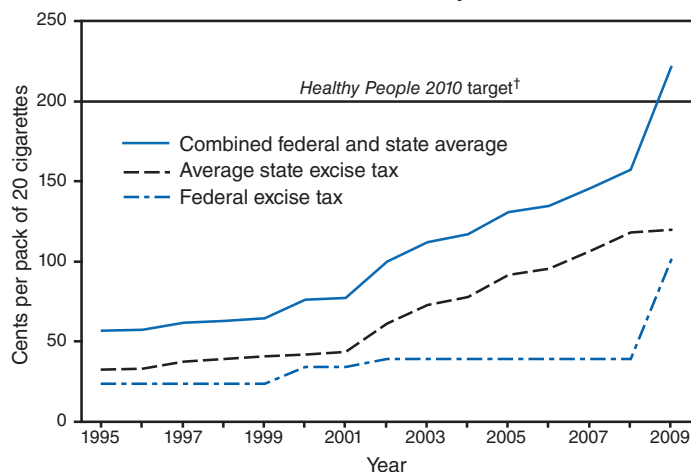
Federal excise tax data were obtained from an online database that contains statutes and other legislation. From December 31, 2005, to April 1, 2009, the federal excise tax on cigarettes increased three times. As a result of the 1998 budget agreement, the federal cigarette excise tax made a graduated increase from 24 cents per pack to 34 cents per pack on January 1, 2000, and then to 39 cents per pack on January 1, 2002 (2). As a funding mechanism for the State Children's Health Insurance Program (SCHIP), the federal excise tax on cigarettes increased from 39 cents per pack to \$1.01 cents per pack effective April 1, 2009.[§] These increases resulted in a 321% overall increase in the federal cigarette excise tax since December 31, 1995 (Figure 1).

State excise tax data were collected from the CDC's State Tobacco Activities Tracking and Evaluation (STATE) system database, an electronic data warehouse that contains tobacco-related epidemiologic and economic data and information on state tobacco-related legislation.[¶] The STATE system tracks state laws on excise taxes for cigarettes with excise tax data in effect since the fourth quarter of 1995. This study did not include excise taxes that became effective after April 1, 2009. Consistent with the measure used for the HP2010 objective, average state excise taxes were calculated for this report.

From December 31, 1995, to April 1, 2009, a total of 107 separate cigarette excise tax increases and one decrease occurred in 45 states and DC. The state cigarette excise tax did not change from December 31, 1995, to April 1, 2009, in five states (Florida, Mississippi, Missouri, North Dakota, and South Carolina). As of April 1, 2009, South Carolina had the lowest state cigarette excise tax, at 7 cents per pack, whereas New York had the highest state cigarette excise tax, at \$2.75 per pack (Table). The average state cigarette excise tax on April 1, 2009, was \$1.20 per pack, a 267% increase from the December 31, 1995, average state cigarette excise tax of 32.7 cents per pack.

The average state cigarette excise tax among major tobacco-growing states (Kentucky, Virginia, North Carolina, South Carolina, Georgia, and Tennessee) was 38.5 cents per pack on

FIGURE 1. State and federal cigarette excise taxes, by year — United States,* December 31, 1995, to April 1, 2009



* District of Columbia is included among results for states.

† Objective 27-21a: to increase the combined federal and average state cigarette excise tax to at least \$2 per pack.

April 1, 2009, compared with 7 cents on December 31, 1995 (a 444% increase). Among all other states (including DC) the average cigarette excise tax was \$1.31 per pack on April 1, 2009, compared with 36 cents on December 31, 1995 (a 263% increase).

In 2003, New Jersey increased its cigarette excise tax to \$2.05 per pack, and Rhode Island increased its state cigarette excise tax to \$1.71 per pack; when combined with the federal cigarette excise tax in 2003 of 39 cents per pack, these two states became the first to achieve the HP2010 objective. As of April 1, 2009, 28 states had achieved the HP2010 objective of \$2.00 per pack when the state cigarette excise tax was combined with the federal excise tax (Figure 2).

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Editorial Note: Cigarette smoking and exposure to secondhand smoke result in approximately 443,000 premature deaths, 5.1 million years of potential life lost, and \$97 billion in productivity losses in the United States each year (3). Comprehensive tobacco control program and policy recommendations have been provided to the public health community with the goal of reducing tobacco use and secondhand smoke exposure so that they are no longer a significant public health problem in the United States (4,5). CDC and the Institute of Medicine (IOM) recommend that comprehensive tobacco control programs be implemented fully in every state and territory to accelerate the reduction in smoking prevalence among all U.S. citizens and decrease the public health burden of smoking-related disease (4,5). Although tax increases are an evidence-based policy intervention that will reduce smoking

[§] Children's Health Insurance Program Reauthorization Act of 2009; public law no: 111-3 (2009).

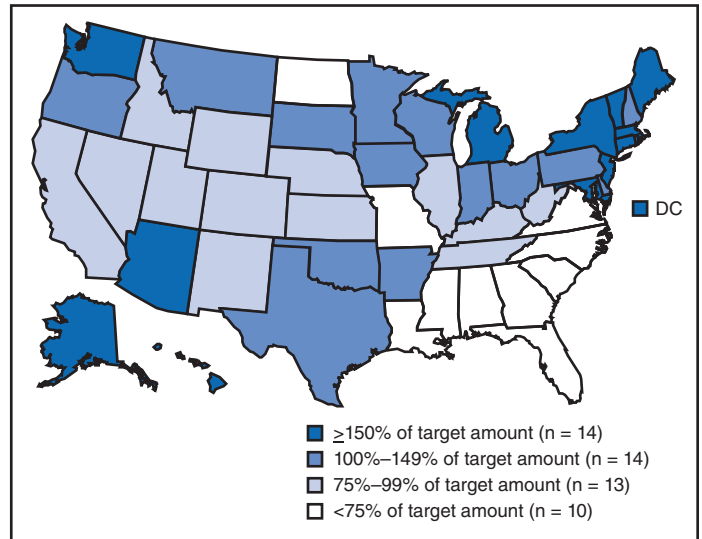
[¶] Available at <http://www.cdc.gov/tobacco/statesystem>.

TABLE. State and federal cigarette excise taxes — United States,* December 31, 1995, and April 1, 2009

State	Cents per pack of 20 cigarettes		
	December 31, 1995	April 1, 2009	% increase 1995 to 2009
Alabama	16.5	42.5	158
Alaska	29.0	200.0	590
Arizona	58.0	200.0	245
Arkansas	31.5	115.0	265
California	37.0	87.0	135
Colorado	20.0	84.0	320
Connecticut	50.0	200.0	300
Delaware	24.0	115.0	379
District of Columbia	65.0	200.0	208
Florida	33.9	33.9	0
Georgia	12.0	37.0	208
Hawaii	60.0	200.0	233
Idaho	28.0	57.0	104
Illinois	44.0	98.0	123
Indiana	15.5	99.5	542
Iowa	36.0	136.0	278
Kansas	24.0	79.0	229
Kentucky	3.0	60.0	1,900
Louisiana	20.0	36.0	80
Maine	37.0	200.0	441
Maryland	36.0	200.0	456
Massachusetts	51.0	251.0	392
Michigan	75.0	200.0	167
Minnesota	48.0	123.0	156
Mississippi	18.0	18.0	0
Missouri	17.0	17.0	0
Montana	18.0	170.0	844
Nebraska	34.0	64.0	88
Nevada	35.0	80.0	129
New Hampshire	25.0	133.0	432
New Jersey	40.0	257.5	544
New Mexico	21.0	91.0	333
New York	56.0	275.0	391
North Carolina	5.0	35.0	600
North Dakota	44.0	44.0	0
Ohio	24.0	125.0	421
Oklahoma	23.0	103.0	348
Oregon	38.0	118.0	211
Pennsylvania	31.0	135.0	335
Rhode Island	61.0	246.0	303
South Carolina	7.0	7.0	0
South Dakota	33.0	153.0	364
Tennessee	13.0	62.0	377
Texas	41.0	141.0	244
Utah	26.5	69.5	162
Vermont	44.0	199.0	352
Virginia	2.5	30.0	1,100
Washington	81.5	202.5	148
West Virginia	17.0	55.0	224
Wisconsin	44.0	177.0	302
Wyoming	12.0	60.0	400
State average	32.7	120.0	267
Federal excise tax	24.0	101.0†	321
Combined federal and state average	56.7	221.0	290

* District of Columbia is included among results for states.
 † The federal tax of \$50.33 is levied per 1,000 cigarettes. When calculated per pack of 20 cigarettes, this is \$1.0066 per pack. This fractional tax was rounded to \$1.01 per pack.

FIGURE 2. Progress toward the *Healthy People 2010* target* for combined state and federal cigarette excise taxes — United States,† April 1, 2009



* Objective 27-21a: to increase the combined federal and average state cigarette excise tax to at least \$2 per pack. Data were calculated by combining the cigarette excise tax rate in each state with the federal cigarette excise tax.
 † District of Columbia is included among results for states.

prevalence independently, excise tax increases are more effective and have greater public health impact when combined with other evidence-based components of comprehensive tobacco control programs (5).

A 10% increase in the real price of cigarettes is estimated to reduce consumption by nearly 4% (6). The Task Force on Community Preventive Services recommends price increases through excise taxes as an effective policy intervention to prevent smoking initiation by adolescents and young adults, reduce cigarette consumption, and increase the number of smokers who quit (6). The 2000 report of the U.S. Surgeon General, *Reducing Tobacco Use*, concluded that raising tobacco excise taxes is one of the most effective tobacco prevention and control strategies (2). Specifically, it found that increasing the price of tobacco products would decrease the prevalence of tobacco use, particularly among youths and young adults, and that tobacco excise tax increases would lead to substantial long-term improvements in health (2). Tax revenues also might support the prevention and treatment components of comprehensive state tobacco control programs (2).

Although the average cigarette excise tax among tobacco-growing states has increased by a greater percentage (444% since December 31, 1995) compared with non-tobacco-growing states (264% since December 31, 1995), the individual cigarette excise tax rates in these states and other bordering

southeastern states remain substantially lower than the rest of the country (Figure 2). In addition to having lower excise taxes, these states typically do not have strong statewide tobacco control policies, such as laws that would protect the public from secondhand smoke exposure in worksites, restaurants, and bars.

Persons in lower-income groups usually smoke more, meaning they expend a greater share of their income to cigarette excise taxes than other socioeconomic groups (2,7,8). Cigarette excise taxes increase the purchase price of cigarettes and can pose a disproportionate economic burden on lower socioeconomic populations (7–9). However, because low-income groups are more responsive to price increases, increasing the real price of cigarettes can reduce cigarette consumption among low-income smokers by a greater percentage than among higher-income smokers, and thereby diminish socioeconomic smoking disparities (7–9). As excise tax increases diminish these smoking disparities, they potentially reduce disparities in morbidity and life expectancy (9). In addition to gaining health benefits attributable to quitting, groups with lower incomes will spend less on cigarettes and more resources will be available to spend on food, housing, and other goods (7).

The findings in this report are subject to at least three limitations. First, the STATE system only collects state-level excise tax data; it does not reflect city, county, or other local excise tax or any state or local sales tax that might be in place in some jurisdictions. Although not included in this study, at least 460 local communities impose a local tax on cigarettes, including New York City (\$1.50 per pack) and Chicago-Cook County (\$2.68 per pack) (10). Second, HP2010 objective 27-21a measures the simple mean of the legislated excise tax in states, not a weighted average that reflects relevant factors such as smoking rate, population size, and demographics. Finally, the excise tax amounts presented in this report, including the HP2010 target of \$2, are not adjusted for inflation. Had the HP2010 goal been required to have the buying power of \$2.00 when the objectives were published in 2000, the inflation-adjusted goal on April 1, 2009, would be approximately \$2.47.**

Increases in state and federal cigarette excise taxes per pack since 1995 have provided an important contribution to preventing tobacco use and promoting cessation. IOM concluded that because excise taxes place a disproportionate burden on lower-income smokers, revenue from excise tax increases should be coupled with existing governmental financing to support cessation programs and services, especially for lower-income smokers. Telephone-based tobacco-use quitlines are an example of existing cessation services that might be expanded using

excise tax revenue. Quitlines are a free, evidence-based cessation service currently available to all populations in every state and DC through a toll-free access number (800-QUIT-NOW [800-784-8669]). IOM further recommends that states dedicate a portion of their tobacco excise tax revenue by statute, if constitutionally permissible, to fund state tobacco control programs at levels recommended by CDC (5). If every state were to fund their tobacco control programs at the level of investment recommended by CDC in its *Best Practices for Comprehensive Tobacco Control Programs — 2007*, in 5 years an estimated 5 million fewer persons in the United States would smoke, and hundreds of thousands of premature tobacco-related deaths would be prevented each year (4).

Acknowledgment

This report is based, in part, on contributions by R Patrick, JD, C Holmes, SS Eidson, JD, and L Lineberger, MayaTech Corporation, Silver Spring, Maryland; and T Pechacek, PhD, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.

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**Based on calculation using the Bureau of Labor Statistics Consumer Price Index inflation calculator, available at <http://data.bls.gov/cgi-bin/cpicalc.pl>.

Health Warnings on Tobacco Products – Worldwide, 2007

Many countries require that tobacco product* packaging includes health warnings about the risks associated with tobacco use (1–3). Health warnings on tobacco product packages are effective in highlighting the perception of health risk (4), supporting the intention to quit tobacco use (5), discouraging the intention to begin tobacco use, and increasing cessation rates (6). Prominent displays of health warnings increase their effectiveness; larger warnings, with pictures, are more likely to be noticed, better communicate health risks, provoke greater emotional response, and further motivate tobacco users to quit (7–9). This report assesses the current status of tobacco packaging health warning requirements worldwide. Governments could further discourage tobacco use by requiring prominent health warnings on tobacco packaging.

Placing health warnings on tobacco product packages was one of the key evidence-based interventions included in the World Health Organization Framework Convention on Tobacco Control (WHO-FCTC) (2), the first public health treaty negotiated under WHO auspices, which was adopted in 2005. Within 3 years, participating countries agreed to implement health warnings describing the harmful effects of all tobacco products. Article 11 (Packaging and labeling of tobacco products) of WHO-FCTC requires government bodies such as ministries of health to approve and ensure the display of large, clear, visible, and legible warnings on at least 30%, and preferably 50% or more, of the principal display area of tobacco packages.

In early 2007, WHO's Tobacco Free Initiative collected information about legally mandated use of tobacco health warnings through a questionnaire distributed to all 193 WHO member states and one territory. Data specific to health warnings were collected for seven criteria: 1) mandate of specific tobacco use health warnings; 2) inclusion of health warnings on tobacco packs and outside packaging; 3) use of large, clear, and visible health warnings; 4) rotation of health warnings; 5) use of the principal languages of the country; 6) inclusion of pictorial warnings; and 7) descriptions of specific harmful effects of tobacco use in health.

National data collectors were appointed by ministries of health and local WHO offices in each country to complete the questionnaire; regional data collectors, appointed for each of the six WHO regional offices, verified the accuracy and completeness of the data. The regional data collectors in turn submitted the data for further processing and analysis to the

Tobacco Free Initiative. The results were validated by each of the member states and then published in 2008 (1).

Data reported from 176 member states indicated that 77 (44%) did not require any warnings on cigarette packs, and 71 (40%) required warnings covering less than 30% of the principal display area. Among the member states, 23 (13%) had warnings that covered at least 30% of the main package display area and included one of the seven warning criteria. Five countries (Australia, Brazil, Canada, Thailand, and Uruguay) (3%) had warnings that covered 50% or more of the principal display areas and included all seven criteria. Among the 176 countries, 15 (9%) required pictorial warnings, and 66 (38%) countries had laws that ban the use of deceptive marketing terms (such as "light" and "mild") that falsely convey that a particular product is less harmful than other tobacco products.

The percentage of member states that had no warnings or warnings that covered less than 30% of the principal package display area was high across all WHO regions: African Region (88%), American Region (74%), Eastern Mediterranean Region (82%), European Region (92%), South East Asia Region (82%), and the Western Pacific Region (71%). The level of implementation of health warnings was associated with a nation's economic status.[†] Approximately 58% of low-income countries, 45% of middle-income countries, and 24% of high-income countries had not implemented any health warnings (Figure).

Reported by: Tobacco Free Initiative, World Health Organization, Geneva, Switzerland.

Editorial Note: Guidelines for implementation of Article 11 were adopted in November 2008 to assist countries in meeting their WHO-FCTC obligations. These guidelines propose that national authorities approve regulations that require warnings on display areas of tobacco packaging that are of size and characteristics that will enhance the effectiveness of health warnings (3).

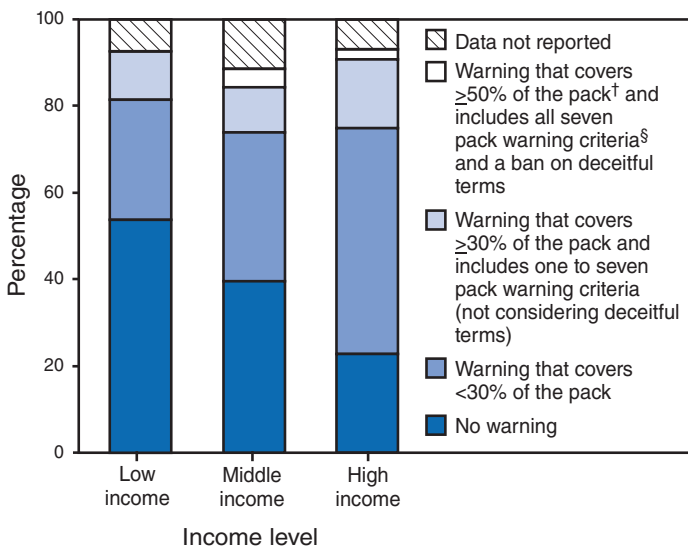
Governments can use cigarette packaging to raise awareness among smokers and nonsmokers about the health risks of tobacco use. Health warnings provide countries with a relatively inexpensive method of informing consumers about the risks of smoking (7). However, findings from WHO's Tobacco Free Initiative indicate that the strategy of placing health warnings on tobacco packaging has been implemented comprehensively in only a few countries.

Countries can require that packaging include larger health warnings, effective text, and pictures. Pictorial warnings

*Products entirely or partly made of leaf tobacco and intended for smoking, sucking, chewing, or snuffing.

[†] For this report, countries are classified according to their 2007 gross national income per capita, calculated using the World Bank Atlas method, as low income (\leq \$935), middle income (\$936–\$11,455) and high income (\geq \$11,456). Additional information is available at <http://www.worldbank.org> under Data & Research.

FIGURE. Percentage of countries that require health warnings on tobacco packaging, by extent of warning required and country income level* — World Health Organization, 2008



* Countries are classified according to their 2007 gross national income per capita, calculated using the World Bank Atlas method, as low income ($\leq \$935$), middle income ($\936 – $\$11,455$) and high income ($\geq \$11,456$). Additional information is available at <http://www.worldbank.org> under Data & Research.

† Available at http://www.who.int/tobacco/mpower/mpower_report_full_2008.pdf.

§ Data specific to health warnings were collected for seven criteria: 1) mandate of specific tobacco use health warnings; 2) inclusion of health warnings on tobacco packs and outside packaging; 3) use of large, clear, and visible health warnings; 4) rotation of health warnings; 5) use of the principal languages of the country; 6) inclusion of pictorial warnings; and 7) descriptions of specific harmful effects of tobacco use in health.

combined with text warnings can increase the effectiveness of health messages.[§] Pictorial warnings also convey health messages to persons who might not be able to read or understand the written information. To help increase the use of pictorial health warnings, the Tobacco Free Initiative will host a repository of pictorial health warnings and provide training in their use.

Health warnings on tobacco products should not be an isolated tobacco control measure. Instead, countries should implement comprehensive tobacco control programs. To help countries fulfil the requirements of WHO-FCTC, WHO has established MPOWER, a package of technical assistance for six tobacco control policies: 1) monitoring tobacco use and prevention policies; 2) protecting persons from tobacco smoke; 3) offering help to quit tobacco smoking; 4) warning about dangers of tobacco; 5) enforcing bans on tobacco advertising, promotion, and sponsorship; and 6) raising taxes on tobacco. These policies are proven to reduce tobacco use (1,2) and can be effectively supported by legislation requiring prominent health warnings on tobacco packaging.

[§] Additional information and examples of pictorial health warnings are available at <http://www.who.int/tobacco/resources/publications/wntd/2009/materials/brochure/en/index.html>.

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Alcohol Use Among Pregnant and Nonpregnant Women of Childbearing Age — United States, 1991–2005

Alcohol consumption during pregnancy is a risk factor for poor birth outcomes, including fetal alcohol syndrome, birth defects, and low birth weight (1). In the United States, the prevalence of fetal alcohol syndrome is estimated at 0.5–2.0 cases per 1,000 births, but other fetal alcohol spectrum disorders (FASDs)* are believed to occur approximately three times as often as fetal alcohol syndrome (2). The 2005 U.S.

*FASD is an umbrella term that includes fetal alcohol syndrome (a lifelong condition that causes physical and mental disabilities, characterized by abnormal facial features, growth deficiencies, and central nervous system problems) and other harmful effects on persons whose mothers use alcohol during pregnancy. These effects include physical, mental, behavioral, or learning disabilities with possible lifelong implications. The term FASD is not intended for use as a clinical diagnosis.

Surgeon General's advisory on alcohol use in pregnancy, advises women who are pregnant or considering becoming pregnant to abstain from using alcohol (2). Binge drinking is particularly harmful to fetal brain development (2,3). *Healthy People 2010* objectives include increasing the percentage of pregnant women who report abstinence from alcohol use to 95% and increasing the percentage who report abstinence from binge drinking to 100% (4). To examine the prevalence of any alcohol use and binge drinking among pregnant women and nonpregnant women of childbearing age in the United States and to characterize the women with these alcohol use behaviors, CDC analyzed 1991–2005 data from Behavioral Risk Factor Surveillance System (BRFSS) surveys. The findings indicated that the prevalence of any alcohol use and binge drinking among pregnant and nonpregnant women of childbearing age did not change substantially from 1991 to 2005. During 2001–2005, the highest percentages of pregnant women reporting any alcohol use were aged 35–44 years (17.7%), college graduates (14.4%), employed (13.7%), and unmarried (13.4%). Health-care providers should ask women of childbearing age about alcohol use routinely, inform them of the risks from drinking alcohol while pregnant, and advise them not to drink alcohol while pregnant or if they might become pregnant (2,5).

BRFSS conducts state-based, random-digit-dialed telephone surveys of the noninstitutionalized U.S. civilian population aged ≥ 18 years, collecting data on health conditions and health risk behaviors. For this report, CDC analyzed BRFSS data from 1991 to 2005 from all 50 states and the District of Columbia for women aged 18–44 years. The median response rate among states, based on Council of American Survey and Research Organizations (CASRO) guidelines, ranged from 71.4% in 1993 to 51.1% in 2005. This report focuses on two drinking behaviors: any use, defined as having at least one drink of any alcoholic beverage in the past 30 days, and binge drinking, defined as having five or more drinks on at least one occasion in the past 30 days.[†] The wording of the question regarding any alcohol use was changed in 1993, 2001, and 2005,[§] the wording of the question regarding binge drinking was changed in

1993 and 2001.[¶] BRFSS questionnaires are available at <http://www.cdc.gov/brfss/questionnaires/questionnaires.htm>.

Percentage estimates and 95% confidence intervals were calculated each year for the two drinking behaviors among pregnant and nonpregnant women. Logistic regression was used to examine the association of age, race/ethnicity, education, employment, and marital status with the two drinking behaviors for pregnant and nonpregnant women with the behaviors as the dependent variables and sociodemographic characteristics as the independent variables in the models. Adjusted odds ratios (AORs) were calculated to describe significant differences by characteristic category. Data from 2001–2005 were aggregated to provide stable estimates to assess the association of these characteristics with the drinking behaviors. Data were weighted to state population estimates and aggregated to represent a nationwide estimate.

Of the 533,506 women aged 18–44 years surveyed during 1991–2005, 22,027 (4.1%) reported being pregnant at the time of the interview. The prevalence of any alcohol use and binge drinking among pregnant and nonpregnant women from 1991 to 2005 did not change substantially over time (Figure). The average annual percentage of any alcohol use among pregnant women was 12.2% (range: 10.2%–16.2%), of binge drinking among pregnant women was 1.9% (range: 0.7%–2.9%), of any alcohol use among nonpregnant women was 53.7% (range: 51.6%–56.3%), and of binge drinking among nonpregnant women was 12.1% (range: 10.8%–13.7%).

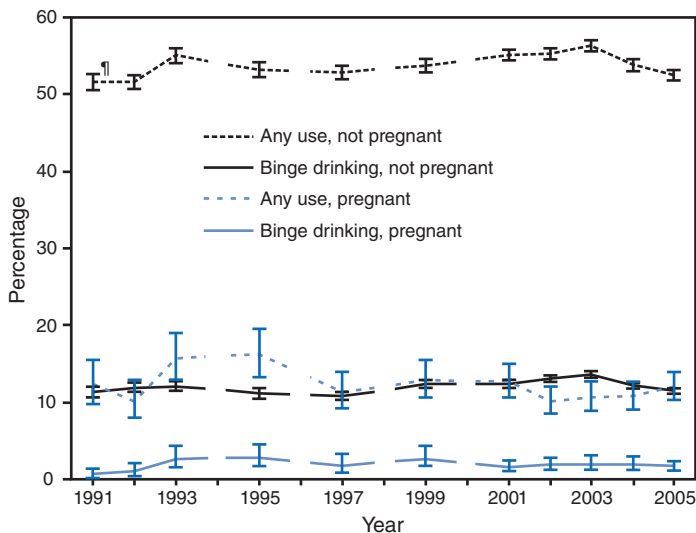
Of the 329,975 women aged 18–44 years surveyed during 2001–2005, 13,820 (4.2%) reported being pregnant at the time of the interview. Among pregnant women, 17.7% of those aged 35–44 years reported any alcohol use, compared with 8.6% of pregnant women aged 18–24 years (AOR = 2.3). Greater percentages of pregnant women with at least some college education (11.2%), or a college degree or more (14.4%), reported alcohol use than pregnant women with a high school diploma or less (8.5%) (AORs = 1.4 and 1.9, respectively). A greater percentage of employed pregnant women (13.7%) reported alcohol use than unemployed pregnant women (8.3%, AOR = 1.5), and a greater percentage of unmarried pregnant women (13.4%) reported alcohol use than married pregnant women (10.2%, AOR = 2.2) (Table). In addition, a greater percentage of employed pregnant women (2.3%) reported binge drinking, compared with unemployed pregnant women

[†] Beginning in 2006, the definition of binge drinking by women changed to four drinks on at least one occasion. Because of this change, data collected after 2005 are not included.

[§] Any alcohol use: "Have you had any beer, wine, wine coolers, cocktails, or liquor during the past month, that is, since (date from one month before interview)" (1991–1992). "During the past month, have you had at least one drink of any alcoholic beverage such as beer, wine, wine coolers, or liquor?" (1993–2000). "A drink of alcohol is one can or bottle of beer, one glass of wine, one can or bottle of wine cooler, one cocktail, or one shot of liquor. During the past 30 days, how often have you had at least one drink of any alcoholic beverage?" (2001–2004). "During the past 30 days, have you had at least one drink of any alcoholic beverage such as beer, wine, a malt beverage or liquor?" (2005).

[¶] Binge drinking: "Considering all types of alcoholic beverages, that is beer, wine, wine coolers, cocktails, and liquor, as drinks, how many times during the past month did you have five or more drinks on an occasion?" (1991–1992). "Considering all types of alcoholic beverages, how many times during the past month did you have five or more drinks on an occasion?" (1993–2000). "Considering all types of alcoholic beverages, how many times during the past 30 days did you have five or more drinks on an occasion?" (2001–2005).

FIGURE. Percentage of women aged 18–44 years who reported any alcohol use or binge drinking,* by pregnancy status — Behavioral Risk Factor Surveillance System (BRFSS) surveys, United States,† 1991–2005‡



* Defined as five or more drinks on at least one occasion.

† BRFSS survey data were not available for 1994, 1996, 1998, and 2000. Data also were not available from Kansas, Nevada, and Wyoming for 1991; from Arkansas and Wyoming for 1992; from Rhode Island for 1993 and 1994; from the District of Columbia for 1995; and from Hawaii for 2004.

‡ Beginning in 2006, the definition of binge drinking by women changed to four drinks on at least one occasion. Because of this change, data collected after 2005 are not included.

§ 95% confidence interval.

(1.3%, AOR = 1.8), and a greater percentage of unmarried pregnant women (3.6%) reported binge drinking than married pregnant women (1.1%, AOR = 4.4).

Reported by: CH Denny, PhD, J Tsai, MD, RL Floyd, DSN, PP Green, MSPH, Div of Birth Defects and Developmental Disabilities, National Center on Birth Defects and Developmental Disabilities, CDC.

Editorial Note: A 2002 report using 1991–1999 BRFSS data determined that, from 1995 to 1999, the percentage of pregnant women reporting any alcohol use decreased, whereas the prevalence of binge drinking during pregnancy and the prevalence of both drinking behaviors among nonpregnant women did not change (6). This report expands on the 2002 report, examining data collected during 1991–2005; this broader perspective indicates that alcohol use and binge drinking among pregnant women and nonpregnant women of childbearing age did not change substantially over time. The prevalence of both types of drinking behavior among pregnant women remain greater than the *Healthy People 2010* targets, and greater progress will be needed to reach them (4,6).

Alcohol use levels before pregnancy are a strong predictor of alcohol use during pregnancy (7). A proportion of women who use alcohol continue that use during the early weeks of gestation because they do not realize they are pregnant.

Approximately 40% of women realize they are pregnant at 4 weeks of gestation, a critical period for fetal organ development (e.g., central nervous system, heart, and eyes) (7). Because approximately half of all births are unplanned, clinicians should screen and advise women of childbearing age of the potential consequences of using alcohol during pregnancy (2).

The findings that, among pregnant women, those who were older, more educated, employed, and unmarried were more likely to use alcohol support results from previous studies, but the reasons for these patterns are not well understood (6,8). Further research is needed; however, some possible reasons include that 1) older women might be more likely to be alcohol dependent and have more difficulty abstaining from alcohol while pregnant, 2) more educated women and employed women might have more discretionary money for the purchase of alcohol, and 3) unmarried women might attend more social occasions where alcohol is served.

The findings in this report are subject to at least five limitations. First, BRFSS data are self-reported and subject to recall and social desirability biases; underreporting of negative health behaviors such as binge drinking and any alcohol use while pregnant is likely. Second, BRFSS survey questions have changed over time, which can affect prevalence estimates. Third, declining response rates, likely attributable in large part to changes in telephone technology (e.g., increases in caller identification and dedicated fax and computer lines) and greater reluctance among the public to respond to telephone surveys, might affect prevalence estimates. Fourth, BRFSS excludes households without landline telephones, including households with only cellular telephones; therefore, the results might not be representative of certain segments of the U.S. population. Finally, this report likely underestimates the current prevalence of binge drinking because it used the 2005 definition (five or more drinks on at least one occasion) rather than the current definition for women of four or more drinks on at least one occasion.

Alcohol use during pregnancy continues to be an important public health concern. Effective screening and counseling are available for women of childbearing age in the preconception and prenatal periods (9). CDC's efforts to reduce the prevalence of alcohol use during pregnancy include funding Fetal Alcohol Spectrum Disorders Regional Training Centers to improve health-care provider skills at screening women for alcohol use and providing brief interventions (10). CDC also is engaged in ongoing work with the Substance Abuse and Mental Health Services Administration and the Indian Health Service to train health-care workers in systems of care funded by these agencies (i.e., alcohol and drug treatment programs and women's health clinics in American Indian communities) (9). Finally, seven CDC-funded state-based fetal alcohol syndrome prevention

TABLE. Estimated percentage* of women aged 18–44 years who reported any alcohol use or binge drinking,† by pregnancy status and selected characteristics — Behavioral Risk Factor Surveillance System (BRFSS) surveys, United States, 2001–2005‡

Characteristic	Pregnant						Nonpregnant					
	Any use			Binge drinking			Any use			Binge drinking		
	%	AOR¶	95% CI**	%	AOR	95% CI	%	AOR	95% CI	%	AOR	95% CI
Total	11.2			1.8			54.6			12.6		
Age group (yrs)												
18–24	8.6	1.0	Referent	2.5	1.0	Referent	55.5	1.0	Referent	19.6	1.0	Referent
25–34	11.2	1.4	(1.1–1.7)	1.4	0.7	(0.4–1.2)	55.1	0.9	(0.9–0.9)	12.2	0.7	(0.7–0.8)
35–44	17.7	2.3	(1.7–3.0)	1.8	0.9	(0.5–1.6)	53.6	0.8	(0.8–0.9)	8.9	0.5	(0.5–0.5)
Race/Ethnicity												
White, non-Hispanic	11.6	1.0	(0.8–1.4)	1.8	1.1	(0.6–2.0)	60.9	1.8	(1.7–1.9)	14.9	1.9	(1.7–2.0)
Black, non-Hispanic	10.3	0.8	(0.5–1.1)	2.1	0.8	(0.4–1.6)	43.3	0.9	(0.8–0.9)	6.8	0.6	(0.6–0.7)
Hispanic (any race)	10.2	1.0	Referent	1.7	1.0	Referent	41.1	1.0	Referent	8.9	1.0	Referent
Other race, non-Hispanic	12.1	1.1	(0.7–1.7)	2.5	1.6	(0.7–3.6)	46.0	0.9	(0.9–1.0)	9.7	1.0	(0.9–1.2)
Education												
High school diploma or less	8.5	1.0	Referent	1.8	1.0	Referent	43.1	1.0	Referent	11.6	1.0	Referent
Some college	11.2	1.4	(1.1–1.8)	2.0	1.4	(0.8–2.3)	57.2	1.6	(1.6–1.7)	14.4	1.2	(1.1–1.2)
College degree or more	14.4	1.9	(1.4–2.4)	1.8	1.8	(0.9–3.4)	66.3	2.4	(2.3–2.5)	12.0	1.1	(1.0–1.1)
Employed												
Yes	13.7	1.5	(1.3–1.9)	2.3	1.8	(1.2–2.8)	59.1	1.5	(1.5–1.6)	13.5	1.4	(1.3–1.4)
No	8.3	1.0	Referent	1.3	1.0	Referent	46.1	1.0	Referent	10.9	1.0	Referent
Married												
Yes	10.2	1.0	Referent	1.1	1.0	Referent	52.6	1.0	Referent	8.4	1.0	Referent
No	13.4	2.2	(1.7–2.7)	3.6	4.4	(2.4–8.0)	56.9	1.4	(1.3–1.4)	17.6	2.2	(2.1–2.2)

* Percentages weighted to represent the U.S. population.

† Defined as five or more drinks on at least one occasion.

‡ Beginning in 2006, the definition of binge drinking by women changed to four drinks on at least one occasion. Because of this change, data collected after 2005 are not included.

¶ Adjusted odds ratio; model includes age, race/ethnicity, education, employment, and marital status.

** Confidence interval.

programs for women in high-risk geographic areas or high-risk subpopulations are completing work in developing, implementing, and evaluating population-based and targeted programs. These programs will provide valuable insights for CDC's continuing efforts to reduce the prevalence of alcohol use during pregnancy.

Acknowledgment

This report is based, in part, on data contributed by BRFSS state coordinators and contributions by O Devine, PhD, National Center on Birth Defects and Developmental Disabilities, CDC.

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Progressive Vaccinia in a Military Smallpox Vaccinee — United States, 2009

On May 19, this report was posted as an MMWR Early Release on the MMWR website (<http://www.cdc.gov/mmwr>).

Progressive vaccinia (PV), previously known as vaccinia necrosum, vaccinia gangrenosum, or disseminated vaccinia, is a rare, often fatal adverse event after vaccination with smallpox vaccine, which is made from live vaccinia virus (*V.*). During

recent vaccination programs potential cases of PV were investigated, but none met standard case definitions (2). PV has not been confirmed to have occurred in the United States since 1987 (3). On March 2, 2009, a U.S. Navy Hospital contacted the Poxvirus Program at CDC to report a possible case of PV in a male military smallpox vaccinee. The service member had been newly diagnosed with acute myelogenous leukemia M0 (AML M0). During evaluation for a chemotherapy-induced neutropenic fever, he was found to have an expanding and nonhealing painless vaccination site 6.5 weeks after receipt of smallpox vaccine. Clinical and laboratory investigation confirmed that the vaccinee met the Brighton Collaboration and CDC adverse event surveillance guideline case definition for PV (4,5). This report summarizes the patient's protracted clinical course and the military and civilian interagency governmental, academic, and industry public health contributions to his complex medical management. The quantities of investigational and licensed therapeutics and diagnostics used were greater than anticipated based on existing smallpox preparedness plans. To support future public health needs adequately, the estimated national supply of therapeutics and diagnostic resources required to care for smallpox vaccine adverse events should be reevaluated.

Case Description

On January 13, 2009, a healthy service member aged 20 years received a primary smallpox vaccination (ACAM2000 [Acambis, Inc., Cambridge, Massachusetts]) in accordance with the U.S. Department of Defense smallpox vaccination policy*; no other vaccinations were administered that day. Twelve days later, the patient visited a local hospital with fever and headache of 1 day's duration and was admitted for workup of leukopenia after his white blood cell count was found to be 1,400 cells/mm³. On January 28, after transfer to a U.S. Navy tertiary-care facility, he was diagnosed with AML M0. On January 30 and February 13, the patient underwent two successive rounds of induction chemotherapy with cytarabine, idarubicin, and dexamethasone. Before initial chemotherapy, the vaccination site pustule had a central crust and measured approximately 1 cm in diameter with minimal surrounding erythema. During the patient's hospital stay from the end of January to the beginning of March, his vaccination site dressing was changed daily.

On March 2, during the evaluation of neutropenic fever, the failure of the patient's vaccination site to heal was described. An annular lesion with a deep bulla, raised violaceous leading

edge, and a central crust that bled with pressure was noted. The size of the lesion had progressed to approximately 4 x 4 cm with minimal surrounding erythema or induration (Figure). The patient described no pain at the site, although he reported occasional pruritus. A swab of the lesion and serum were sent to CDC for viral and serologic analysis. Viral analysis of the swab by multiple real-time polymerase chain reaction (PCR) assays for orthopoxvirus and vaccinia yielded evidence of viral DNA; viral culture was positive for orthopoxvirus. Serum showed equivocal to absent levels of anti-orthopoxvirus immunoglobulin G (IgG) and immunoglobulin M (IgM) by enzyme-linked immunosorbent assay. The results of the diagnostic testing combined with the patient's medical history met the PV level 1 case definition as defined by the Brighton Collaboration and the confirmed case definition as described by CDC surveillance guidelines (4,5). The criteria met by both case definitions were 1) a documented clinical diagnosis of a disease that is known to be associated with cell-mediated immunodeficiency (in this case AML M0), 2) the primary vaccination site's failure to resolve (in this case >6 weeks post vaccination), and 3) the laboratory confirmation of vaccinia virus as the causative agent.

On March 3, imiquimod was applied directly to the lesion. Within 24 hours of confirmation of PV on March 4, the patient received licensed Vaccinia Immune Globulin Intravenous (Human) (VIGIV) (Cangene Corporation, Winnipeg, Canada). On March 5 and March 6, oral and topical ST-246 (SIGA Technologies, Corvallis, Oregon) were administered under an Emergency Investigational New Drug (E-IND) application. The patient remained stable until the evening of

FIGURE. Photographs of a progressive vaccinia lesion in a military smallpox vaccinee, taken on A) March 5,* B) March 27† and C) April 27, 2009§¶



Photo/U.S. Navy

* 51 days post vaccination.

† 73 days post vaccination and 9 days post development of satellite lesions.

§ 104 days post vaccination and 40 days post development of satellite lesions.

¶ Images have been altered to remove features that might identify the patient.

* Information about U.S. Department of Defense policies regarding smallpox vaccination and screening before smallpox vaccination is available at <http://www.smallpox.army.mil>.

March 7, when he became septic with *Pseudomonas aeruginosa*, likely from a perirectal abscess. He required intubation, maximal vasopressor support, multiple antibiotics, and stress dose corticosteroids. He then developed multiorgan failure and began continuous venovenous hemodialysis. During the next 12 days, the patient slowly stabilized. As a consequence of the duration and amount of vasopressor support, the patient required a bilateral trans-tibial amputation because of dry gangrene of his feet.

During March 6–19, the patient received additional oral and topical ST-246 and VIGIV; his ST-246 levels were noted to be lower than those achieved both in healthy subjects in phase I clinical trials and in successful treatment of nonhuman primates with systemic orthopoxvirus disease. The lesion size remained unchanged, but the central crust of the vaccination site sloughed off, followed by most of the outer “ring” flattening, leaving a shallow ulcer with healthy-appearing granulation tissue. During his steroid taper, additional satellite lesions surrounding the vaccination site appeared on March 18, and viral DNA was detected again in the blood. These lesions became vesicular in nature, and on March 26, after a second E-IND was issued, CMX001 (Chimerix, Inc., Research Triangle Park, North Carolina), a lipid conjugate of cidofovir, was administered.

From March 24 onward, the satellite and main vaccination site lesions continued to crust, the scabs separated, and underlying tissue epithelialized (Figure). Blood viral DNA levels cleared on March 29. On April 10, the borders of lesions again appeared raised; a shave biopsy grew methicillin-resistant *Staphylococcus aureus*, which responded to antibiotic therapy. The patient received intermittent granulocyte colony-stimulating factor, and his absolute neutrophil and lymphocyte

count increased over time. By May 1, significant portions of the scabs/eschars had fallen off or were removed manually, revealing healthy epidermis. Numerous therapeutics with different biologic mechanisms were used to treat PV in this patient (Table).

From February 21 onward, the patient had remained in contact isolation, first for a *Clostridium difficile* infection and then for his progressive vaccinia infection. On May 5, contact precautions were discontinued because of the lack of viable virus in lesion specimens from the previous 4 weeks. No cases of contact vaccinia were identified among this patient’s health-care workers or close contacts.

During March 3–May 18, nearly 200 clinical specimens (lesion and satellite swabs/crusts, ethylenediaminetetraacetic acid [EDTA] blood, bone marrow, and serum) were collected and submitted to CDC to evaluate disease progression and guide therapeutic interventions. After April 23, swabs from satellite lesions or the main vaccination site showed significantly reduced or absent levels of viral DNA, and no viable virus was detected after April 2. Oropharyngeal sampling and bone marrow biopsies from early and late March, respectively, were negative for vaccinia virus. Orthopoxvirus DNA was detected in EDTA blood at intermittent times during the course of the patient’s infection; however, no viable virus was cultured from blood. As of May 12, the patient had no demonstrable IgM response to orthopoxvirus; IgG levels appeared fully reliant on VIGIV infusion.

During March 3–May 18, a total of 20 conference calls to discuss patient status and treatment options were held between the Vaccine Healthcare Centers Network, Military Vaccine Agency (MILVAX), Bureau of Medicine and Surgery of the Navy, CDC, Food and Drug Administration (FDA),

TABLE. Administration dates and dosages of therapeutics used in treatment of progressive vaccinia in a military smallpox vaccinee — United States, March 5–May 18, 2009

Treatment*	Formulation	Dosage	Application	Administration dates
ST-246	Oral	400 mg	Once daily	March 5–19
		800 mg	Once daily	March 20–24
		1200 mg	Once daily	March 25 to present†
ST-246	Topical	1%, 0.5 mL	Once daily	March 6; April 21–May 12
		1%, 0.5 mL	Twice daily	March 7–April 20
CMX001	Oral	200 mg	Once per date	March 26
		100 mg	Once per date	April 1, 7, 13, 20, 27
Imiquimod	Topical	5%, 12.5 mg	Once daily	March 24–May 12
VIGIV§¶**	Intravenous	6,000 U/kg	Once per date	March 4, 11, 20; April 1, 3, 6, 8, 18
		18,000 U/kg	Once per date	April 9
		24,000 U/kg	Once per date	March 24; April 14, 23, 28; May 8

* In summary, the patient’s dose of oral ST-246 was increased twice to obtain more optimal drug levels, CMX001 was begun, topical ST-246 and imiquimod continued, as well as periodic infusions of VIGIV at varying doses.

† As of May 18, 2009.

§ Vaccinia Immune Globulin Intravenous (Human).

¶ VIGIV is supplied as a 15 mL single dose vial containing >50,000 U/vial.

** Patient received a total of 16,740,000 U of VIGIV during March 4–May 8.

National Institutes of Health (NIH), SIGA Technologies, Chimerix, Inc., and academic and health-care professionals. As of May 18, MILVAX provided 22 and the Strategic National Stockpile (SNS) provided 254 vials of VIGIV used in treatment of this case.

Reported by: *E Lederman, MD, H Groff, MD, T Warkentien, MD, A Reese, MD, US Naval Medical Center. D Hruby, PhD, T Bolken, D Grosenbach, PhD, S Yan, PhD, SIGA Technologies, Corvallis, Oregon. W Painter, MD, L Trost, MD, B Lampert, MD, Chimerix, Inc., Research Triangle Park, North Carolina. J Cohen, MD, National Institutes of Health; R Engler, MD, Walter Reed Vaccine Healthcare Center; W Davidson, MPH, S Smith, MS, K Wilkins, Z Braden, Y Li, PhD, I Damon, MD, Div of Viral and Rickettsial Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases, CDC.*

Editorial Note: Although PV is a rare adverse event (one case per million during routine vaccination during 1963–1968), its case fatality rate in primary U.S. vaccinees was 15% despite treatment with massive amounts of VIG (intramuscular) (6). Extensive surgical debridement was sometimes required, even necessitating disarticulation of the arm to “debulk” the amount of infectious material (7). Before smallpox vaccination, patients are screened for numerous contraindications (8). At the time of his vaccination, the patient described in this report did not have any obvious signs or symptoms that would meet any exclusion criteria for vaccination. Training in use of, and careful adherence to, screening tools can identify vaccine candidates at risk for PV and other adverse events (2). Despite this, vaccinees with occult immunodeficiencies might not be recognized, and therefore appropriately deferring vaccination in these persons is not always possible.

Lack of inflammation at the expanding vaccination site is the hallmark of PV. Any smallpox vaccinee who has an expanding, nonhealing, painless vaccination site without inflammation for more than 2 weeks should be evaluated for an underlying immunodeficiency, and diagnosis of and treatment for PV should be considered. Health-care providers should report suspected cases of PV or other adverse events to the Vaccine Adverse Event Reporting System (VAERS).[†] Suspected cases of PV also should be reported to state health officials and CDC for clinical consultation and to obtain select therapeutics available only through the SNS. State health departments should call the CDC Emergency Operations Center at 770-488-7100.

This patient's protracted clinical course is consistent with previously published cases reports and surveillance summaries. The development of progressive vaccinia, historically observed in patients with cellular immunodeficiencies, often leads to superinfection and subsequent sepsis (i.e., fungal, parasitic, and bacterial infections resulting in toxic or septicemic shock, then ultimately death). Past treatment typically included massive

doses of VIG, administration of thiosemicarbazone, blood products, and supportive care for accompanying infections (7,9). The improvement of progressive vaccinia in this patient was associated with receipt of VIGIV (the only licensed product for treatment of vaccinia adverse events stockpiled by the SNS), ST-246, and CMX001, and an increase in lymphocyte count. The use of two antiviral agents with different mechanisms of action[§] was enabled by the research and development of medical countermeasures for smallpox preparedness activities, as well as the use of the emergency IND process. As of May 18, the patient had shed nearly all of the scab material on and around the vaccination site.

The rapid mobilization of military, CDC, FDA, NIH, drug manufacturer, and academic and health-care human resources to review the case's status and to provide daily, then biweekly laboratory findings that guided treatment recommendations, was enabled by smallpox public health preparedness research and training efforts. Future cases of PV likely will require similar intensive and multidisciplinary clinical consultation. Experts with background in vaccine safety, PV treatment, clinical virology, infectious disease, and immunodeficiencies should be engaged.

Continuing medical education and reinforcement of training related to the prevention, early recognition, and treatment of smallpox vaccine-related adverse events should be part of smallpox vaccination programs.[¶] The patient described in this report received VIGIV in the amount originally estimated to treat 30 persons. The extraordinary amounts of VIGIV used to treat this single case of PV underscore the need to reevaluate the adequacy of the national stockpiled supply of this or other medical countermeasures (treatment or prophylactic). Such reevaluation, with additional focus on immunocompromised hosts, will aid in the smallpox vaccination program planning and overall smallpox preparedness efforts.

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[§] ST-246 prevents viral egress, whereas CMX001 inhibits viral replication, and some data suggest they are synergistic in vitro (10).

[¶] CDC's clinical evaluation tools for smallpox vaccine adverse reactions are available at <http://emergency.cdc.gov/agent/smallpox/vaccination/clineval>.

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with novel influenza A (H1N1) infection had been reported in California. This report summarizes the 30 hospitalized cases as of May 17, including a detailed description of four cases that illustrate the spectrum of illness severity and underlying risk factors. This preliminary overview indicates that, although the majority of hospitalized persons infected with novel influenza A (H1N1) recovered without complications, certain patients had severe and prolonged disease. All hospitalized patients with novel influenza A (H1N1) infection should be monitored carefully and treated with antiviral therapy, including patients who seek care >48 hours after illness onset (4,5).

Summary of Hospitalized Cases

Beginning on April 20, 2009, the California Department of Public Health (CDPH) and local health departments in Imperial and San Diego counties worked with hospital infection-control practitioners to initiate enhanced surveillance for hospitalized cases of laboratory-confirmed or probable novel influenza A (H1N1) infection at all 25 hospitals in the two counties. Three days later, on April 23, 2009, CDPH extended this surveillance statewide. Cases are reported as either probable (defined as detection of influenza A by real-time reverse transcription–polymerase chain reaction [rRT-PCR] that is unsubtypable for human influenza virus subtypes H1 or H3) or confirmed (defined as positive by CDC protocol for rRT-PCR for novel influenza A H1N1).^{*} Approximately 96% of unsubtypable California specimens subsequently have been confirmed as novel influenza A (H1N1) at CDC or at the CDPH Viral and Rickettsial Disease Laboratory (VRDL).[†]

For this report, a hospitalized case was defined as a confirmed or probable case of novel influenza A (H1N1) infection in a patient who was hospitalized for ≥ 24 hours. Of the 30 hospitalized patients, 26 were confirmed and four were probable (confirmatory testing is in progress); symptom onset ranged from April 3 to May 9. The cases were reported from 11 counties, most of which are located in southern or central California. The largest number of patients, (15 [50%]) resided in San Diego and Imperial counties. Of the 26 patients for whom information on ethnicity was available, 17 (65%) were Hispanic. Ages of the 30 patients ranged from 27 days to 89 years, with a median age of 27.5 years; 21 (70%) were female. Four (13%) patients had traveled to Mexico in the 7 days before onset of illness. None of the 30 patients reported exposure to swine or a known confirmed case of novel influenza A (H1N1) infection.

^{*} Additional information available at <http://www.who.int/csr/resources/publications/swineflu/realtimeptcr/en/index.html>.

[†] Additional information available at [http://ww2.cdph.ca.gov/programs/vrld/pages/enhancedsurveillanceforinfluenzaa\(h1\).aspx](http://ww2.cdph.ca.gov/programs/vrld/pages/enhancedsurveillanceforinfluenzaa(h1).aspx).

Hospitalized Patients with Novel Influenza A (H1N1) Virus Infection – California, April–May, 2009

On May 18, this report was posted as an MMWR Early Release on the MMWR website (<http://www.cdc.gov/mmwr>).

Since April 15 and 17, 2009, when the first two cases of novel influenza A (H1N1) infection were identified from two southern California counties, novel influenza A (H1N1) cases have been documented throughout the world, with most cases occurring in the United States and Mexico (1–3). In the United States, early reports of illnesses associated with novel influenza A (H1N1) infection indicated the disease might be similar in severity to seasonal influenza, with the majority of patients not requiring hospitalization and only rare deaths reported, generally in persons with underlying medical conditions (2,3). As of May 17, 2009, 553 novel influenza A (H1N1) cases, including 333 confirmed and 220 probable cases, had been reported in 32 of 61 local health jurisdictions in California. Of the 553 patients, 30 have been hospitalized. No fatal cases associated

The most common admission diagnoses were pneumonia and dehydration. Nineteen patients (64%) had underlying medical conditions; the most common were chronic lung disease (e.g., asthma and chronic obstructive pulmonary disease), conditions associated with immunosuppression, chronic cardiac disease (e.g., congenital heart disease and coronary artery disease), diabetes, and obesity. The most common symptoms were fever, cough, vomiting, and shortness of breath; diarrhea was uncommon. Of the 25 patients who had chest radiographs, 15 (60%) had abnormalities suggestive of pneumonia, including 10 with multilobar infiltrates and five with unilobar infiltrates. Six patients were admitted to the intensive care unit (ICU), and four required mechanical ventilation. Five patients were pregnant. Two of these developed complications, including spontaneous abortion and premature rupture of the membranes; the fetuses were at 13 and 35 weeks gestation, respectively.

Of the 24 patients tested for influenza A in the hospital, the rapid antigen test was positive in 16 and negative in five; three patients tested positive by other methods (direct immunofluorescent antibody [two patients] and culture [one patient]). None of the 30 patients had microbiologic evidence of secondary bacterial infection by blood, urine, or sputum cultures (or endotracheal aspirate or bronchoalveolar lavage cultures in the case of intubated patients). Fifteen (50%) received antiviral treatment with oseltamivir; for five patients, treatment was initiated within 48 hours of onset of symptoms. Among the 15 not treated with antivirals, six sought care >48 hours after illness onset. Of the 22 patients with available history, six (27%) had received seasonal influenza vaccination. As of May 17, 23 patients had been discharged to home, with a median length of hospital stay of 4 days (range: 1–10 days). Seven patients remained in the hospital, with median lengths of stay of 15 days (range: 4–167 days) (Tables 1 and 2).

Case Reports

Patient 3. An infant girl aged 5 months was born prematurely at 27 weeks in early December 2008 with intrauterine growth retardation and congenital heart disease with patent ductus arteriosus and ventricular septal defect. The infant had a complicated hospital course in the neonatal ICU after birth, including development of bronchopulmonary dysplasia and respiratory distress syndrome requiring prolonged mechanical ventilation and multiple courses of steroids, several episodes of clinical sepsis and pneumonia, and chronic anemia and thrombocytopenia. By the fifth month, the infant had been weaned from the ventilator and was doing well on high-flow nasal cannula oxygen. However, on hospital day number 150, she developed a new nonproductive cough and fever, with a new

TABLE 1. Case characteristics for 30 hospitalized patients with novel influenza A (H1N1) — California, April 15, 2009–May 17, 2009

Case characteristic	No.	(%)
Age group (yrs)		
<5	6	(20)
5-19	7	(23)
20-39	8	(27)
40-59	4	(13)
>60	5	(17)
Chronic comorbid illness*		
Chronic lung disease [†]	11	(37)
Other immunosuppression [§]	6	(20)
Chronic cardiac disease [¶]	5	(17)
Diabetes mellitus	4	(13)
Obesity	4	(13)
Seizure disorder	3	(10)
Pregnancy	5	(17)
Symptoms and signs		
Fever	29	(97)
Cough	23	(77)
Vomiting	14	(46)
Shortness of breath	13	(43)
Chills	11	(37)
Sore throat	10	(33)
Body aches	10	(33)
Rhinorrhea	9	(30)
Headache	5	(17)
Conjunctivitis	3	(10)
Diarrhea	3	(10)
Altered mental status	2	(7)
Generalized weakness	2	(7)
Clinical findings and course		
Infiltrates on chest radiograph**	15	(60)
Intensive-care unit admission	6	(20)
Mechanical ventilation	4	(13)
Antiviral treatment	15	(50)

* Conditions listed are not mutually exclusive; certain patients had multiple underlying chronic diseases.

[†] Includes asthma, chronic obstructive pulmonary disorder, bronchopulmonary dysplasia/respiratory distress syndrome, bronchiolitis obliterans organizing pneumonia, Sjogren syndrome, and obstructive sleep apnea.

[¶] Includes congenital heart disease, atrial fibrillation, status-post aortic valve replacement, and coronary artery disease.

[§] Includes immunosuppressive drugs, cancer, and congenital immunodeficiency.

** Out of 25 cases with chest radiographs.

infiltrate of the right lung on chest radiograph that progressed to complete opacification of both lung fields. Multiple blood, urine, and sputum cultures were unrevealing; rapid antigen test was positive for influenza A, with subsequent confirmation at the CDPH VRDL for novel influenza A (H1N1). The source of the infant's infection is still under investigation. The infant was reintubated and started on broad spectrum antibiotics and oseltamivir at a dose of 2 mg/kg every 12 hours, 3 days after fever. As of May 14, the patient remained hospitalized in critical condition.

Patient 16. A previously healthy woman aged 29 years, who was 28 weeks pregnant, sought care at an emergency

TABLE 2. Detailed clinical characteristics for 30 hospitalized patients with novel influenza A (H1N1) — California, April 15, 2009–May 17, 2009 (Continued)

Patient no.	Age	Sex	Underlying conditions*	Admission diagnosis	Abnormal complete blood count values	Chest radiographic findings	Intensive-care unit admission	Mechanical ventilation	Antiviral treatment	Length of stay
1	27 days	F	None	Rule out sepsis	None	Normal	No	No	None	3 days
2	6 wks	M	None	Pneumonia	Anemia [†]	Bilateral infiltrates	No	No	Oseltamivir	6 days
3	5 mos	F	Prematurity, intrauterine growth retardation, bronchopulmonary dysplasia, congenital heart disease, chronic corticosteroid administration	Respiratory Distress Syndrome, prematurity	Leukocytosis, [§] anemia [†]	Bilateral infiltrates	Yes	Yes	None	Still hospitalized: day 167
4	17 mos	M	None	Pneumonia respiratory failure	Leukocytosis, [§] lymphopenia [¶]	Bilateral infiltrates	No	No	None	2 days
5	3 yrs	F	None	Dehydration	None	Not done	No	No	Oseltamivir	1 day
6	3 yrs	M	T-cell immunodeficiency	Pneumonia	Leukocytosis, [§] anemia [†]	Bilateral infiltrates	Yes	No	Oseltamivir	5 days
7	7 yrs	F	Asthma, obesity	Asthma exacerbation	Leukopenia ^{**}	Unilobar infiltrate	No	No	None	4 days
8	9 yrs	M	Asthma	Dehydration	Leukopenia ^{**} Lymphopenia [¶]	Hyperinflation, perivascular cuffing	No	No	None	5 days
9	15 yrs	M	Seizure disorder	Dehydration	Lymphopenia [¶]	Normal	No	No	Oseltamivir	1 day
10	15 yrs	M	Cerebral palsy, asthma, seizure disorder	Fever, seizure	Thrombocytopenia ^{††}	Multilobar infiltrates	No	No	Oseltamivir	Still hospitalized: day 11
11	17 yrs	F	Pregnancy	Not available	Not available	Not available	No	No	Oseltamivir	5 days
12	19 yrs	F	None	Acute pharyngitis	Lymphopenia [¶]	Normal	No	No	Oseltamivir	1 day
13	19 yrs	F	Pregnancy	Rule out sepsis	Lymphopenia [¶]	Not done	No	No	None	2 days
14	21 yrs	F	None	Dehydration	None	Normal	No	No	Oseltamivir	2 days
15	26 yrs	F	None	Pneumonia, respiratory failure	None	Unilobar infiltrate	No	No	Oseltamivir	2 days
16	29 yrs	F	Pregnancy	Pneumonia	Leukocytosis [§]	Bilateral infiltrates	Yes	No	None	9 days
17	30 yrs	F	Diabetes melitus, obesity	Viral syndrome, vomiting	None	None	No	No	Oseltamivir	1 day
18	32 yrs	M	Obstructive sleep apnea	Respiratory failure	Leukocytosis, [§] lymphopenia [¶]	Bilateral infiltrates	Yes	Yes	Oseltamivir	8 days

department on April 26 with complaints of subjective fever, productive cough, and increasing shortness of breath during the preceding 10 days. Upon initial evaluation, the patient's vital signs were notable for low grade fever (99.6°F [37.6°C]), a respiratory rate of 38 breaths per minute, blood pressure of 112/57 mmHg, heart rate of 104 beats per minute, and oxygen saturation of 87% on room air. A chest radiograph revealed bilateral perihilar interstitial infiltrates with mediastinal lymphadenopathy. Her complete blood count and chemistries were normal except for an elevated white blood cell count of 11.4 cells/mm³ with a differential of 42% segmented neutrophils, 45% bands, and 9% lymphocytes. The patient was admitted

to the ICU and started on broad spectrum antibiotics (azithromycin and ceftriaxone). Serial fetal ultrasounds were normal. Multiple blood, urine, and sputum cultures were unrevealing; rapid antigen test was positive for influenza A, with subsequent confirmation of novel influenza A (H1N1) at the CDPH VRDL. She was not treated with antiviral medications. She gradually improved and was discharged on amoxicillin after 9 days.

Patient 18. A man aged 32 years with a history of obstructive sleep apnea sought care at an emergency department on May 5 with a 3-day history of fever, chills, and productive cough. The patient reported he had been taking amoxicillin for a diagnosis

TABLE 2. (Continued) Detailed clinical characteristics for 30 hospitalized patients with novel influenza A (H1N1) — California, April 15, 2009–May 17, 2009

Patient no.	Age	Sex	Underlying conditions*	Admission diagnosis	Abnormal complete blood count values	Chest radiographic findings	Intensive-care unit admission	Mechanical ventilation	Antiviral treatment	Length of stay
19	34 yrs	F	Asthma, pregnancy	Dehydration	Leukopenia,** thrombocytopenia ^{††}	None	No	No	None	7 days
20	35 yrs	F	None	Pneumonia	Leukocytosis, [§] anemia [†]	Not done	No	No	None	7 days
21	35 yrs	F	Down syndrome, congenital heart defect, congenital T-cell deficiency	Pneumonia, respiratory failure	Lymphopenia, [¶] thrombocytopenia ^{††}	Bilateral infiltrates	No	No	None	4 days
22	40 yrs	F	Asthma, HTN, obesity	Pneumonia, respiratory failure	Leukocytosis, [§] anemia [†]	Bilateral infiltrates	Yes	Yes	None	Still hospitalized: day 18
23	41 yrs	F	Autoimmune hepatitis/biliary cirrhosis s/p liver transplant, HTN, obesity	Viral syndrome	Leukopenia, [¶] anemia, [†] thrombocytopenia ^{††}	Unilobar infiltrate	No	No	Oseltamivir	6 days
24	42 yrs	F	Asthma, gastrointestinal reflux, pregnancy	Premature rupture of membranes, pre-eclampsia	None	Not done	No	No	Oseltamivir	4 days
25	49 yrs	M	Aortic valve replacement, HTN, lupus nephritis, seizure disorder	Fever	Lymphopenia, [¶] anemia, [*] thrombocytopenia ^{††}	Not available	No	No	None	Still hospitalized: day 15
26	69 yrs	M	COPD, HTN, atrial fibrillation	Respiratory distress	Leukopenia [¶]	Normal	No	No	None	Still hospitalized: day 13
27	72 yrs	F	COPD, BOOP, DM, atrial fibrillation, HTN, chronic corticosteroid administration	Respiratory distress	Leukocytosis [§]	Unilobar infiltrate	No	No	None	10 days
28	73 yrs	F	COPD, HTN	Respiratory distress	Lymphopenia [¶]	Normal	No	No	Oseltamivir	3 days
29	87 yrs	F	CAD, COPD, HTN, breast cancer	Pneumonia, respiratory failure	Leukocytosis, [§] anemia [†]	Bilateral infiltrates and pleural effusions	Yes	Yes	None	Still hospitalized: day 27
30	89 yrs	F	Sjogren syndrome, pulmonary fibrosis, chronic corticosteroid administration, HTN	Not available	Leukocytosis, [§] positive D-dimer	Unilobar infiltrate	No	No	Oseltamivir	Still hospitalized: day 4

* HTN: hypertension. COPD: chronic obstructive pulmonary disease. CAD: coronary artery disease. BOOP: bronchiolitis obliterans organizing pneumonia.

[†] Hematocrit <35%.

[§] Total leukocyte count >10 cells/mm³.

[¶] Total lymphocyte count <800 cells/mm³.

** White blood cell count <5,000 cells/mm³.

^{††} Platelet count <150,000 cells/mm³.

of sinusitis, following complaints of vertigo and dizziness, for the past 2 weeks. His vital signs showed a temperature of 99.1°F (37.3°C), blood pressure of 89/58 mmHg, and heart rate of 84 beats per minute. Physical exam of the chest showed good air movement bilaterally, although chest radiograph revealed bilateral infiltrates. His complete blood count and chemistries were normal except for an elevated white blood cell count of 13.8 cells/mm³ with a differential of 94% segmented neutrophils and 4% lymphocytes. An arterial blood gas showed respiratory acidosis and hypoxemia with pO₂ of 80 mm Hg on room air. The patient was admitted to the ICU on empiric broad spectrum antibiotics and required intubation on the second

hospital day for worsening hypoxemia. Initial microbiologic workup and influenza rapid antigen tests were negative; the patient was started on oseltamivir on hospital day 2. A repeat rapid antigen test and bronchoalveolar lavage viral culture were positive for influenza A, with subsequent confirmation of novel influenza A (H1N1). The patient improved, was extubated on hospital day 5, and was discharged on hospital day 10.

Patient 29. A woman aged 87 years with multiple medical problems, including recently diagnosed breast cancer with possible abdominal metastasis, hypertension, diabetes mellitus, coronary artery disease, cerebrovascular disease, chronic renal insufficiency, and obesity, was brought for care at an emergency

department on April 21 after being found unconscious by her daughter. The patient had reported onset of fever, cough, and weakness 2 days before admission and also new onset of orthopnea and bilateral leg swelling. She was wheelchair bound and had no recent history of travel or known contact with ill persons. In the emergency room the patient was afebrile, with a blood pressure of 57/39 mmHg, pulse 57, respiratory rate of 14 breaths per minute, and oxygen saturation of 87% on room air. Electrocardiogram was suggestive of non Q-wave myocardial infarction. Chest radiograph showed bilateral pneumonia and congestive heart failure with marked cardiomegaly. Her laboratory abnormalities included an elevated white blood cell count of 13.4 cells/mm³, mild anemia with a hematocrit of 34%, a mildly elevated creatinine at 1.8 mg/dL, alanine aminotransferase of 36 units/L and aspartate aminotransferase of 160 units/L, and markedly elevated troponin and creatinine kinase levels of 29.43 ng/mL and 653 IU/L, respectively. The patient went into respiratory arrest and was subsequently intubated and started on low dose dopamine, and admitted to the ICU with a diagnosis of myocardial infarction, congestive heart failure, pneumonia and presumed sepsis. A chest computed tomography (CT) scan showed complete atelectasis of the right middle lobe, bilateral ground glass opacities of the upper lobes, and bilateral pleural effusions. A subsequent bronchoscopy identified a large cauliflower-shaped mass in the right lower lobe airway. Multiple blood, urine, and sputum cultures were unrevealing; rapid antigen test was positive for influenza A, with subsequent confirmation of novel influenza A (H1N1) at the CDPH VRDL. The patient remains hospitalized in critical condition under intensive care.

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Editorial Note: Initial surveillance for hospitalized cases of novel influenza A (H1N1) infection in California indicates that the majority of patients were discharged after short hospital stays. Previously healthy patients without underlying chronic medical conditions recovered with an uncomplicated hospital course and a median length of stay of 2.5 days (range: 1–7 days). Although one third of hospitalized patients had abnormal chest radiographs with multilobar infiltrates, only 9%

were treated with oseltamivir; nonetheless, most had favorable outcomes. Of five pregnant women, two developed serious sequelae; however, the role that preceding infection with novel influenza A (H1N1) played in these outcomes is unclear.

Certain hospitalized patients in California experienced severe disease and prolonged hospital courses. Of note, three of the six California patients admitted to an ICU continue to require prolonged intensive care. Extremes in age and multiple and debilitating underlying medical conditions might be contributing to the severity of illness in these patients. Although chronic underlying medical conditions and pregnancy classically are associated with a greater risk for complications for seasonal influenza (6), one patient (patient 18) who was relatively healthy with only mild chronic pulmonary disease required intensive care and mechanical ventilation. More data are needed regarding which populations are at greatest risk for hospitalization and severe sequelae after infection with novel influenza A (H1N1).

As of May 15, 2009, 9% of approximately 11,600 clinical specimens submitted for testing to California public health laboratories since April 27, 2009, were positive by rRT-PCR for influenza A; of those, 23% and 28% were subtyped as seasonal influenza A/H1 and A/H3, respectively. These results indicate that seasonal influenza viruses continue to circulate throughout California and might be a cause of influenza-like illness and positive results from rapid antigen tests. Although rapid antigen test results were positive in 67% of tested cases in this series, anecdotal reports from other cases confirmed at CDPH VRDL, tested mostly in the outpatient setting, suggest that false positive and negative results are common. Accordingly, CDPH has emphasized the importance of testing influenza viruses in the state with rRT-PCR. CDPH also has advised clinicians in California to collect respiratory specimens for rRT-PCR testing, subtyping, and further characterization at public health laboratories from patients who are hospitalized or who die with febrile respiratory illness.

Additional information regarding California testing guidelines is available at [http://ww2.cdph.ca.gov/programs/vrdl/pages/diagnostictestingforswineinfluenzaA\(H1\).aspx](http://ww2.cdph.ca.gov/programs/vrdl/pages/diagnostictestingforswineinfluenzaA(H1).aspx). Additional information regarding novel influenza A (H1N1) treatment guidance and other CDC recommendations is available at <http://www.cdc.gov/h1n1flu/guidance>.

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Notice to Readers

World No Tobacco Day — May 31, 2009

Tobacco use is one of the major preventable causes of premature death and disease in the world (1). The World Health Organization (WHO) attributes approximately 5.4 million deaths per year to tobacco use (or one in 10 deaths among adults worldwide), a number expected to exceed 8 million per year by 2030 (2).

Evidence-based tobacco control programs that are comprehensive, sustained, and support nonsmoking behaviors have been shown to prevent and reduce tobacco use (3). Such programs combine educational, clinical, regulatory, economic, and social strategies to establish smoke-free policies and social norms, to promote and assist tobacco users to quit, and to prevent initiation of tobacco use. Strategies include increasing the unit price of tobacco products and implementing smoking bans through policies, regulations, and laws; providing insurance coverage of tobacco use treatment; and limiting minors' access to tobacco products.

As part of a comprehensive tobacco control program, prominent tobacco health warnings that appear on packs of cigarettes have been proven to motivate users to quit and to

reduce the appeal of tobacco for those who are not yet addicted (4). WHO's theme for World No Tobacco Day, which will take place on May 31, 2009, is Tobacco Health Warnings. Additional information on World No Tobacco Day 2009 is available at <http://www.who.int/tobacco/wntd/2009>.

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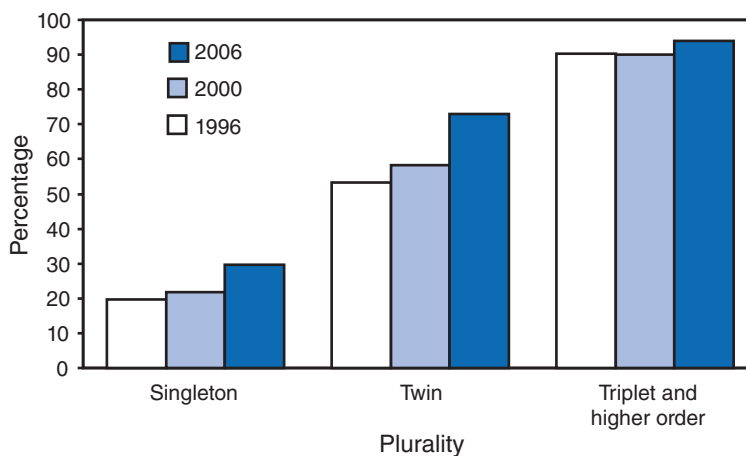
Errata: Vol. 58, No. 18

In the report, “Novel Influenza A (H1N1) Virus Infections in Three Pregnant Women—United States, April–May 2009,” on page 498, the second and third sentences in the first complete paragraph should read as follows: “The specimen was forwarded to the Virus Surveillance and Diagnostic Branch Laboratory, Influenza Division, CDC, where it **could not be confirmed** as novel influenza A (H1N1) virus. On April 30, a repeat nasopharyngeal specimen **and sputum specimen were collected that were both** positive by rRT-PCR for novel influenza A (H1N1) virus at CDC.”

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage of Live Births by Cesarean Delivery, by Plurality — United States, 1996, 2000, and 2006



The percentage of live births by cesarean delivery varies substantially by plurality. In 2006, the percentage was 72.9% for births in twin deliveries and 93.9% for births in triplet and higher order deliveries, compared with 29.6% for singleton births. From 1996 to 2006, the percentage of cesarean deliveries increased 50% for singletons and 37% for twins. The percentage of cesarean deliveries for triplet and higher order deliveries remained high throughout 1996–2006, increasing slightly from 2000 to 2006.

SOURCE: National Vital Statistics System, 1996, 2000, and 2006. Available at <http://www.cdc.gov/nchs/births.htm>.

TABLE I. Provisional cases of infrequently reported notifiable diseases (<1,000 cases reported during the preceding year) — United States, week ending May 16, 2009 (19th week)*

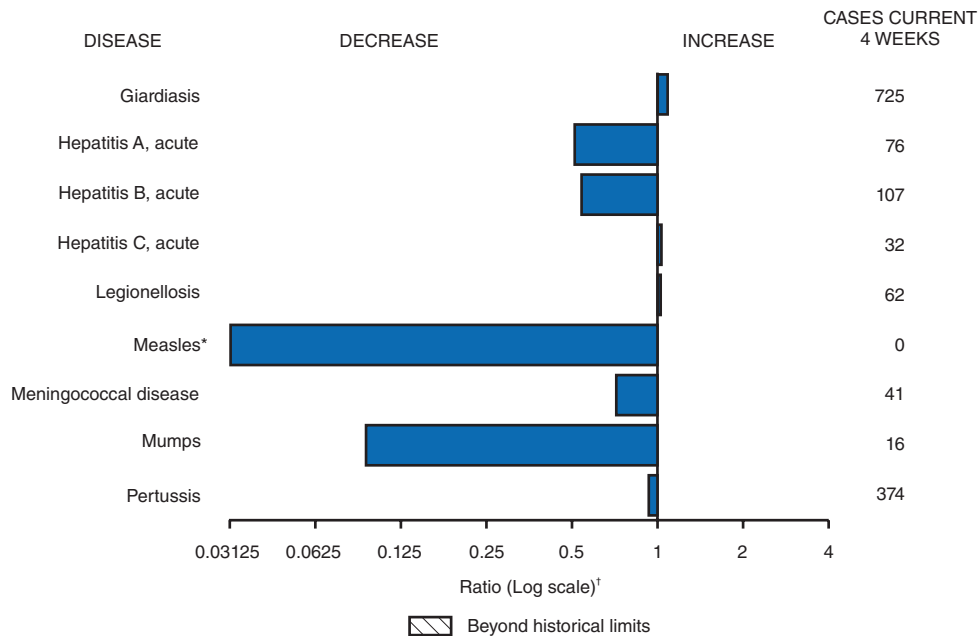
Disease	Current week	Cum 2009	5-year weekly average†	Total cases reported for previous years					States reporting cases during current week (No.)
				2008	2007	2006	2005	2004	
Anthrax	—	—	—	—	1	1	—	—	
Botulism:									
foodborne	—	6	0	17	32	20	19	16	
infant	—	19	1	108	85	97	85	87	
other (wound and unspecified)	—	11	1	19	27	48	31	30	
Brucellosis	2	33	3	77	131	121	120	114	NY (1), OH (1)
Chancroid	1	16	1	40	23	33	17	30	KS (1)
Cholera	—	2	0	3	7	9	8	6	
Cyclosporiasis§	1	30	16	138	93	137	543	160	SC (1)
Diphtheria	—	—	—	—	—	—	—	—	
Domestic arboviral diseases§,¶:									
California serogroup	—	—	0	62	55	67	80	112	
eastern equine	—	—	—	4	4	8	21	6	
Powassan	—	—	0	2	7	1	1	1	
St. Louis	—	—	0	13	9	10	13	12	
western equine	—	—	—	—	—	—	—	—	
Ehrlichiosis/Anaplasmosis§,**:									
<i>Ehrlichia chaffeensis</i>	2	54	7	1,098	828	578	506	338	SC (1), TN (1)
<i>Ehrlichia ewingii</i>	—	—	—	9	—	—	—	—	
<i>Anaplasma phagocytophilum</i>	1	23	6	739	834	646	786	537	NY (1)
undetermined	—	8	3	158	337	231	112	59	
<i>Haemophilus influenzae</i> ,††									
invasive disease (age <5 yrs):									
serotype b	—	11	0	28	22	29	9	19	
nonserotype b	—	77	3	234	199	175	135	135	
unknown serotype	—	67	4	166	180	179	217	177	
Hansen disease§	—	17	2	80	101	66	87	105	
Hantavirus pulmonary syndrome§	—	1	1	18	32	40	26	24	
Hemolytic uremic syndrome, postdiarrheal§	5	40	4	289	292	288	221	200	CT (1), NY (1), MO (1), NC (1), TN (1)
Hepatitis C viral, acute	10	282	15	868	845	766	652	720	OH (3), MI (2), MO (1), GA (1), FL (2), CA (1)
HIV infection, pediatric (age <13 years)§§	—	—	5	—	—	—	380	436	
Influenza-associated pediatric mortality§,¶¶	2	62	2	88	77	43	45	—	AZ (2)
Listeriosis	3	162	10	760	808	884	896	753	NY (1), NC (1), GA (1)
Measles***	—	16	2	140	43	55	66	37	
Meningococcal disease, invasive†††:									
A, C, Y, and W-135	4	115	6	341	325	318	297	—	MN (1), GA (1), FL (1), NM (1)
serogroup B	1	51	3	186	167	193	156	—	MN (1)
other serogroup	—	8	1	33	35	32	27	—	
unknown serogroup	10	201	13	603	550	651	765	—	NY (1), PA (1), KS (1), WA (1), CA (6)
Mumps	1	119	103	449	800	6,584	314	258	OH (1)
Novel influenza A virus infections§§§	—	5,710	—	2	4	N	N	N	
Plague	—	—	0	1	7	17	8	3	
Poliomyelitis, paralytic	—	—	—	—	—	—	1	—	
Polio virus infection, nonparalytic§	—	—	—	—	—	N	N	N	
Psittacosis§	—	6	0	9	12	21	16	12	
Q fever total§,¶¶¶:	1	21	3	120	171	169	136	70	
acute	1	18	1	108	—	—	—	—	CA (1)
chronic	—	3	0	12	—	—	—	—	
Rabies, human	—	—	—	1	1	3	2	7	
Rubella****	—	1	0	17	12	11	11	10	
Rubella, congenital syndrome	—	1	—	—	—	1	1	—	
SARS-CoV§,††††	—	—	—	—	—	—	—	—	
Smallpox§	—	—	—	—	—	—	—	—	
Streptococcal toxic-shock syndrome§	1	63	3	158	132	125	129	132	NY (1)
Syphilis, congenital (age <1 yr)	—	55	7	414	430	349	329	353	
Tetanus	—	4	0	19	28	41	27	34	
Toxic-shock syndrome (staphylococcal)§	1	31	2	73	92	101	90	95	CA (1)
Trichinellosis	—	9	0	38	5	15	16	5	
Tularemia	—	8	2	122	137	95	154	134	
Typhoid fever	1	115	7	444	434	353	324	322	MN (1)
Vancomycin-intermediate <i>Staphylococcus aureus</i> §	—	21	0	59	37	6	2	—	
Vancomycin-resistant <i>Staphylococcus aureus</i> §	—	—	0	—	2	1	3	1	
Vibriosis (noncholera <i>Vibrio</i> species infections)§	8	61	3	490	549	N	N	N	NC (2), FL (4), TN (1), CA (1)
Yellow fever	—	—	—	—	—	—	—	—	

See Table I footnotes on next page.

TABLE I. (Continued) Provisional cases of infrequently reported notifiable diseases (<1,000 cases reported during the preceding year) — United States, week ending May 16, 2009 (19th week)*

—: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts.
 * Incidence data for reporting year 2008 and 2009 are provisional, whereas data for 2004, 2005, 2006, and 2007 are finalized.
 † Calculated by summing the incidence counts for the current week, the 2 weeks preceding the current week, and the 2 weeks following the current week, for a total of 5 preceding years. Additional information is available at <http://www.cdc.gov/epo/dphsi/phs/files/5yearweeklyaverage.pdf>.
 § Not notifiable in all states. Data from states where the condition is not notifiable are excluded from this table, except starting in 2007 for the domestic arboviral diseases and influenza-associated pediatric mortality, and in 2003 for SARS-CoV. Reporting exceptions are available at <http://www.cdc.gov/epo/dphsi/phs/infdis.htm>.
 ¶ Includes both neuroinvasive and nonneuroinvasive. Updated weekly from reports to the Division of Vector-Borne Infectious Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases (ArboNET Surveillance). Data for West Nile virus are available in Table II.
 ** The names of the reporting categories changed in 2008 as a result of revisions to the case definitions. Cases reported prior to 2008 were reported in the categories: Ehrlichiosis, human monocytic (analogous to *E. chaffeensis*); Ehrlichiosis, human granulocytic (analogous to *Anaplasma phagocytophilum*), and Ehrlichiosis, unspecified, or other agent (which included cases unable to be clearly placed in other categories, as well as possible cases of *E. ewingii*).
 †† Data for *H. influenzae* (all ages, all serotypes) are available in Table II.
 §§ Updated monthly from reports to the Division of HIV/AIDS Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention. Implementation of HIV reporting influences the number of cases reported. Updates of pediatric HIV data have been temporarily suspended until upgrading of the national HIV/AIDS surveillance data management system is completed. Data for HIV/AIDS, when available, are displayed in Table IV, which appears quarterly.
 ¶¶ Updated weekly from reports to the Influenza Division, National Center for Immunization and Respiratory Diseases. Sixty-one influenza-associated pediatric deaths occurring during the 2008-09 influenza season have been reported.
 *** No measles cases were reported for the current week.
 ††† Data for meningococcal disease (all serogroups) are available in Table II.
 §§§ These cases were obtained from state and territorial health departments in response to novel Influenza A (H1N1) infections and include cases in addition to those reported to the National Notifiable Diseases Surveillance System (NNDSS). Because of the volume of cases and the method by which they are being collected, a 5-year weekly average for this disease is not calculated.
 ¶¶¶ In 2008, Q fever acute and chronic reporting categories were recognized as a result of revisions to the Q fever case definition. Prior to that time, case counts were not differentiated with respect to acute and chronic Q fever cases.
 **** No rubella cases were reported for the current week.
 †††† Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases.

FIGURE I. Selected notifiable disease reports, United States, comparison of provisional 4-week totals May 16, 2009, with historical data



* No measles cases were reported for the current 4-week period yielding a ratio for week 19 of zero (0).
 † 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.

Notifiable Disease Data Team and 122 Cities Mortality Data Team
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TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending May 16, 2009, and May 10, 2008 (19th week)*

Reporting area	Hepatitis (viral, acute), by type†										Legionellosis				
	A					B									
	Current week	Previous 52 weeks		Cum 2009	Cum 2008	Current week	Previous 52 weeks		Cum 2009	Cum 2008	Current week	Previous 52 weeks		Cum 2009	Cum 2008
		Med	Max				Med	Max				Med	Max		
United States	21	40	89	598	993	33	73	193	1,133	1,334	22	51	152	502	642
New England	—	2	8	31	51	1	1	4	11	28	—	2	18	14	34
Connecticut	—	0	4	9	10	—	0	3	4	12	—	0	5	6	7
Maine§	—	0	5	1	3	—	0	2	5	4	—	0	2	—	1
Massachusetts	—	1	3	14	26	—	0	2	1	8	—	1	7	6	12
New Hampshire	—	0	2	3	4	—	0	2	1	1	—	0	5	—	4
Rhode Island§	—	0	2	3	8	—	0	1	—	2	—	0	14	1	6
Vermont§	—	0	1	1	—	—	0	1	—	1	—	0	1	1	4
Mid. Atlantic	1	5	13	63	117	1	7	17	96	180	8	15	60	128	141
New Jersey	—	1	5	5	27	—	1	5	8	56	—	2	14	6	14
New York (Upstate)	1	1	4	17	25	1	1	11	24	23	6	5	24	51	37
New York City	—	2	6	17	33	—	2	4	22	35	—	2	12	12	20
Pennsylvania	—	1	4	24	32	—	2	8	42	66	2	6	35	59	70
E.N. Central	—	5	11	72	148	1	9	20	145	167	4	8	41	92	148
Illinois	—	1	5	16	54	—	2	7	17	50	—	2	13	8	22
Indiana	—	0	3	5	8	—	1	18	20	10	—	1	6	7	10
Michigan	—	2	5	26	58	—	2	8	45	61	—	2	16	18	43
Ohio	—	1	4	20	14	1	2	13	48	40	4	3	18	54	67
Wisconsin	—	0	3	5	14	—	0	3	15	6	—	0	3	5	6
W.N. Central	5	2	16	43	127	4	2	16	63	24	1	2	8	16	31
Iowa	—	1	6	5	60	—	0	3	8	7	—	0	2	7	7
Kansas	—	0	1	3	9	—	0	3	2	3	—	0	1	1	1
Minnesota	4	0	12	11	10	4	0	11	11	1	—	0	4	—	3
Missouri	—	0	3	15	14	—	1	5	32	12	1	1	7	5	10
Nebraska§	1	0	4	8	32	—	0	3	9	1	—	0	3	2	9
North Dakota	—	0	2	—	—	—	0	1	—	—	—	0	3	1	—
South Dakota	—	0	1	1	2	—	0	1	1	—	—	0	1	—	1
S. Atlantic	7	7	15	151	130	10	20	34	378	341	6	9	22	123	128
Delaware	—	0	1	1	2	—	0	2	10	9	—	0	2	1	2
District of Columbia	U	0	0	U	U	U	0	0	U	U	—	0	2	—	4
Florida	5	3	8	79	56	7	6	11	120	125	1	3	7	50	50
Georgia	—	1	4	20	22	1	3	9	52	55	—	1	5	18	11
Maryland§	—	1	4	16	16	—	2	5	35	32	—	2	9	22	27
North Carolina	1	1	9	16	9	—	1	19	107	25	5	0	7	22	7
South Carolina§	—	0	3	10	6	2	1	4	11	28	—	0	2	1	2
Virginia§	1	1	6	9	16	—	2	10	23	34	—	1	5	9	17
West Virginia	—	0	1	—	3	—	1	6	20	33	—	0	3	—	8
E.S. Central	—	1	9	10	17	—	8	13	106	139	—	2	10	22	29
Alabama§	—	0	2	1	4	—	2	7	30	38	—	0	2	2	4
Kentucky	—	0	3	1	7	—	2	7	31	40	—	1	4	11	16
Mississippi	—	0	2	5	—	—	1	3	5	13	—	0	1	—	—
Tennessee§	—	0	6	3	6	—	3	8	40	48	—	0	5	9	9
W.S. Central	—	4	43	47	90	5	12	96	176	272	—	2	21	20	16
Arkansas§	—	0	1	4	2	—	1	5	12	17	—	0	2	1	1
Louisiana	—	0	2	2	5	—	1	4	16	33	—	0	2	1	2
Oklahoma	—	0	6	1	3	2	2	16	40	26	—	0	6	1	1
Texas§	—	3	37	40	80	3	7	74	108	196	—	1	19	17	12
Mountain	1	3	31	52	79	1	3	10	45	61	2	2	8	30	30
Arizona	1	2	28	29	28	—	1	5	22	23	2	0	3	15	7
Colorado	—	0	2	7	17	—	0	3	8	9	—	0	2	1	3
Idaho§	—	0	1	—	12	1	0	2	2	3	—	0	1	—	1
Montana§	—	0	1	2	—	—	0	1	—	—	—	0	2	4	3
Nevada§	—	0	3	6	2	—	0	3	6	18	—	0	2	5	5
New Mexico§	—	0	1	5	14	—	0	2	4	6	—	0	2	—	3
Utah	—	0	2	3	3	—	0	3	3	1	—	0	2	5	8
Wyoming§	—	0	0	—	3	—	0	1	—	1	—	0	0	—	—
Pacific	7	8	25	129	234	10	6	36	113	122	1	4	9	57	85
Alaska	—	0	1	3	2	—	0	1	2	4	—	0	1	2	1
California	4	6	25	99	189	9	5	28	86	83	1	3	9	48	68
Hawaii	—	0	2	3	4	—	0	1	1	3	—	0	1	1	4
Oregon§	—	0	2	6	16	—	0	8	12	16	—	0	2	3	8
Washington	3	1	4	18	23	1	1	8	12	16	—	0	3	3	4
American Samoa	—	0	0	—	—	—	0	0	—	—	N	0	0	N	N
C.N.M.I.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Guam	—	0	0	—	—	—	0	0	—	—	—	0	0	—	—
Puerto Rico	—	0	4	7	9	—	0	5	2	19	—	0	0	—	—
U.S. Virgin Islands	—	0	0	—	—	—	0	0	—	—	—	0	0	—	—

C.N.M.I.: Commonwealth of Northern Mariana Islands.

U: Unavailable. —: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts. Med: Median. Max: Maximum.

* Incidence data for reporting year 2008 and 2009 are provisional.

† Data for acute hepatitis C, viral are available in Table I.

§ Contains data reported through the National Electronic Disease Surveillance System (NEDSS).

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending May 16, 2009, and May 10, 2008 (19th week)*

Reporting area	Pertussis					Rabies, animal					Rocky Mountain spotted fever				
	Current week	Previous 52 weeks		Cum 2009	Cum 2008	Current week	Previous 52 weeks		Cum 2009	Cum 2008	Current week	Previous 52 weeks		Cum 2009	Cum 2008
		Med	Max				Med	Max				Med	Max		
United States	83	235	1,690	3,885	2,749	23	74	119	1,066	1,336	10	39	150	270	142
New England	—	18	35	170	342	4	8	21	104	122	—	0	2	3	1
Connecticut	—	0	4	6	24	—	3	17	44	57	—	0	0	—	—
Maine†	—	1	7	31	12	—	1	5	18	24	—	0	2	3	—
Massachusetts	—	12	30	105	270	—	0	0	—	—	—	0	1	—	1
New Hampshire	—	1	5	19	9	1	1	7	10	13	—	0	1	—	—
Rhode Island†	—	1	6	3	22	—	0	3	8	9	—	0	2	—	—
Vermont†	—	0	2	6	5	3	1	6	24	19	—	0	0	—	—
Mid. Atlantic	13	23	64	333	324	7	18	30	231	264	—	2	29	7	26
New Jersey	—	3	12	26	52	—	0	0	—	—	—	0	6	—	15
New York (Upstate)	5	6	41	69	97	7	9	20	113	129	—	0	29	1	3
New York City	—	0	21	33	36	—	0	2	—	8	—	0	2	4	4
Pennsylvania	8	10	33	205	139	—	8	17	118	127	—	0	2	2	4
E.N. Central	24	37	238	795	582	2	2	28	17	20	—	2	15	9	9
Illinois	—	13	45	164	56	—	1	20	6	6	—	1	10	6	8
Indiana	—	2	158	73	15	—	0	2	—	1	—	0	3	—	—
Michigan	2	8	21	183	67	2	1	9	11	10	—	0	1	1	—
Ohio	22	12	57	347	418	—	0	7	—	3	—	0	4	2	1
Wisconsin	—	2	7	28	26	N	0	0	N	N	—	0	1	—	—
W.N. Central	7	30	872	801	211	4	5	17	91	75	2	4	33	24	15
Iowa	—	4	21	41	33	—	0	5	6	5	—	0	2	—	1
Kansas	—	2	12	70	26	—	1	6	36	33	—	0	0	—	—
Minnesota	—	2	808	155	31	—	0	11	18	16	—	0	0	—	—
Missouri	5	14	51	449	95	4	1	8	15	5	2	3	32	23	14
Nebraska†	2	4	32	77	17	—	0	0	—	—	—	0	4	1	—
North Dakota	—	0	24	2	—	—	0	9	3	8	—	0	1	—	—
South Dakota	—	0	10	7	9	—	0	4	13	8	—	0	1	—	—
S. Atlantic	14	25	71	529	257	2	28	66	471	686	4	16	72	177	49
Delaware	—	0	3	5	2	—	0	0	—	—	—	0	5	1	2
District of Columbia	—	0	2	—	1	—	0	0	—	—	—	0	1	—	1
Florida	14	7	20	164	59	—	0	22	49	138	1	0	3	2	2
Georgia	—	3	9	74	19	—	6	47	102	144	2	1	9	9	10
Maryland†	—	3	10	33	41	—	7	17	102	155	—	1	7	13	11
North Carolina	—	0	65	152	59	N	2	4	N	N	—	9	55	129	11
South Carolina†	—	2	10	52	29	—	0	0	—	—	—	1	9	9	3
Virginia†	—	3	24	44	42	—	11	24	183	211	1	2	15	13	7
West Virginia	—	0	2	5	5	2	1	6	35	38	—	0	1	1	2
E.S. Central	3	10	33	212	86	—	3	7	34	61	1	4	23	31	22
Alabama†	—	2	11	59	18	—	0	0	—	—	—	1	8	8	10
Kentucky	1	4	15	89	13	—	1	4	22	12	—	0	1	—	—
Mississippi	—	1	5	17	37	—	0	1	—	1	—	0	3	1	3
Tennessee†	2	2	14	47	18	—	2	6	12	48	1	3	19	22	9
W.S. Central	18	38	383	520	222	—	0	9	16	34	3	2	132	15	13
Arkansas†	—	2	38	27	23	—	0	6	12	20	—	0	60	3	1
Louisiana	—	2	7	34	6	—	0	0	—	—	—	0	2	—	2
Oklahoma	—	0	40	9	4	—	0	9	4	13	—	0	71	2	4
Texas†	18	31	303	450	189	—	0	1	—	1	3	1	6	10	6
Mountain	3	15	31	288	367	—	2	9	35	20	—	1	3	4	6
Arizona	—	2	10	49	98	N	0	0	N	N	—	0	2	1	3
Colorado	—	3	12	91	59	—	0	0	—	—	—	0	1	—	—
Idaho†	3	1	5	32	16	—	0	2	—	—	—	0	1	—	—
Montana†	—	0	4	9	58	—	0	4	10	—	—	0	1	1	—
Nevada†	—	0	3	6	12	—	0	5	—	1	—	0	2	—	—
New Mexico†	—	1	10	29	22	—	0	2	14	14	—	0	1	1	1
Utah	—	4	19	71	97	—	0	6	—	1	—	0	1	1	2
Wyoming†	—	0	2	1	5	—	0	4	11	4	—	0	2	—	—
Pacific	1	24	98	237	358	4	4	13	67	54	—	0	1	—	1
Alaska	—	3	21	27	29	—	0	2	8	12	N	0	0	N	N
California	—	6	24	22	179	4	3	12	59	41	—	0	0	—	—
Hawaii	—	0	3	10	4	—	0	0	—	—	N	0	0	N	N
Oregon†	—	3	37	81	53	—	0	2	—	1	—	0	1	—	1
Washington	1	6	76	97	93	—	0	0	—	—	—	0	0	—	—
American Samoa	—	0	0	—	—	N	0	0	N	N	N	0	0	N	N
C.N.M.I.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Guam	—	0	0	—	—	—	0	0	—	—	N	0	0	N	N
Puerto Rico	—	0	1	1	—	—	1	5	15	24	N	0	0	N	N
U.S. Virgin Islands	—	0	0	—	—	N	0	0	N	N	N	0	0	N	N

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U: Unavailable. —: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts. Med: Median. Max: Maximum.

* Incidence data for reporting year 2008 and 2009 are provisional.

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TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending May 16, 2009, and May 10, 2008 (19th week)*

Reporting area	Streptococcal diseases, invasive, group A				<i>Streptococcus pneumoniae</i> , invasive disease, nondrug resistant† Age <5 years					
	Current week	Previous 52 weeks		Cum 2009	Cum 2008	Current week	Previous 52 weeks		Cum 2009	Cum 2008
		Med	Max				Med	Max		
United States	47	99	237	2,324	2,579	20	35	124	722	830
New England	—	5	31	149	161	—	1	12	21	41
Connecticut	—	0	26	43	12	—	0	11	—	—
Maine§	—	0	3	8	12	—	0	1	—	1
Massachusetts	—	3	10	60	105	—	1	3	15	32
New Hampshire	—	1	4	24	15	—	0	1	4	7
Rhode Island§	—	0	8	4	8	—	0	2	—	1
Vermont§	—	0	3	10	9	—	0	1	2	—
Mid. Atlantic	13	18	38	437	550	2	4	33	109	103
New Jersey	—	1	9	3	97	—	1	4	14	32
New York (Upstate)	9	6	25	167	170	2	2	17	57	40
New York City	—	4	12	90	108	—	0	31	38	31
Pennsylvania	4	6	18	177	175	N	0	2	N	N
E.N. Central	7	17	43	467	513	4	6	18	107	149
Illinois	—	4	11	107	147	—	1	5	14	43
Indiana	—	3	23	77	62	—	0	13	11	16
Michigan	3	3	10	80	95	—	1	5	29	41
Ohio	2	4	13	134	140	4	1	6	41	26
Wisconsin	2	1	10	69	69	—	0	3	12	23
W.N. Central	3	5	37	184	212	6	2	14	65	54
Iowa	—	0	0	—	—	—	0	0	—	—
Kansas	—	0	5	28	24	N	0	1	N	N
Minnesota	1	0	34	66	101	6	0	8	28	24
Missouri	—	1	8	51	51	—	1	4	27	19
Nebraska§	2	1	3	27	18	—	0	1	3	4
North Dakota	—	0	2	2	7	—	0	3	3	2
South Dakota	—	0	2	10	11	—	0	2	4	5
S. Atlantic	14	22	46	513	515	2	6	14	143	159
Delaware	—	0	1	7	6	—	0	0	—	—
District of Columbia	—	0	2	—	5	N	0	0	N	N
Florida	7	5	12	128	118	2	1	6	34	24
Georgia	6	5	13	123	107	—	2	6	43	47
Maryland§	—	3	10	77	95	—	1	3	29	34
North Carolina	—	2	12	53	62	N	0	0	N	N
South Carolina§	—	1	5	35	34	—	1	6	26	25
Virginia§	1	3	9	70	69	—	0	2	3	25
West Virginia	—	1	4	20	19	—	0	2	8	4
E.S. Central	3	4	10	102	84	—	1	6	28	50
Alabama§	N	0	0	N	N	N	0	0	N	N
Kentucky	—	1	5	18	18	N	0	0	N	N
Mississippi	N	0	0	N	N	—	0	2	—	14
Tennessee§	3	3	8	84	66	—	1	6	28	36
W.S. Central	3	10	75	203	207	5	6	46	126	115
Arkansas§	—	0	2	9	6	1	0	3	12	7
Louisiana	—	0	2	6	9	—	0	3	12	5
Oklahoma	2	2	16	79	53	1	1	7	26	38
Texas§	1	6	59	109	139	3	4	34	76	65
Mountain	4	10	22	209	287	1	4	16	109	138
Arizona	4	3	8	65	93	—	2	10	64	63
Colorado	—	3	8	76	73	—	1	4	20	30
Idaho§	—	0	2	3	10	1	0	1	3	2
Montana§	N	0	0	N	N	N	0	0	N	N
Nevada§	—	0	1	3	6	—	0	1	—	2
New Mexico§	—	2	7	40	72	—	0	3	11	20
Utah	—	1	6	21	28	—	0	4	11	20
Wyoming§	—	0	1	1	5	—	0	1	—	1
Pacific	—	3	9	60	50	—	1	5	14	21
Alaska	—	0	4	8	12	—	0	4	9	11
California	N	0	0	N	N	N	0	0	N	N
Hawaii	—	3	8	52	38	—	0	2	5	10
Oregon§	N	0	0	N	N	N	0	0	N	N
Washington	N	0	0	N	N	N	0	0	N	N
American Samoa	—	0	8	—	16	N	0	0	N	N
C.N.M.I.	—	—	—	—	—	—	—	—	—	—
Guam	—	0	0	—	—	—	0	0	—	—
Puerto Rico	N	0	0	N	N	N	0	0	N	N
U.S. Virgin Islands	—	0	0	—	—	N	0	0	N	N

C.N.M.I.: Commonwealth of Northern Mariana Islands.

U: Unavailable. —: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts. Med: Median. Max: Maximum.

* Incidence data for reporting year 2008 and 2009 are provisional.

† Includes cases of invasive pneumococcal disease, in children aged <5 years, caused by *S. pneumoniae*, which is susceptible or for which susceptibility testing is not available (NNDSS event code 11717).

§ Contains data reported through the National Electronic Disease Surveillance System (NEDSS).

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