Update: Influenza Activity—United States and Worldwide, 2000-01 Season, and Composition of the 2001-02 Influenza Vaccine

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The 2000-01 influenza season was mild in the United States and was the first season since 1995-96 that was not predominated by A (H3N2) viruses. Influenza A (H1N1) viruses predominated in the United States. In some regions, however, influenza B viruses were reported more frequently than influenza A viruses. Worldwide, influenza A (H1N1) and B viruses also predominated in the United States. In some regions, however, influenza B viruses were reported more frequently than influenza A viruses. Worldwide, influenza A (H1N1) and B viruses also predominated. This report summarizes U.S. and worldwide influenza activity during the 2000-01 influenza season and describes the composition of the 2001-02 influenza vaccine.

United States

Influenza activity increased in mid-December and peaked from mid-January through early February. Influenza A (H1N1) viruses predominated; however, the number of influenza type B viruses increased as the season progressed. Influenza B viruses were more frequently identified than influenza A viruses from the week ending February 10 through the week ending May 19 and were the predominant virus type identified in three of the nine surveillance regions.

World Health Organization and National Respiratory and Enteric Virus Surveillance System collaborating laboratories in the United States tested 88,598 respiratory specimens for influenza during October 1, 2000-May 19, 2001; 9962 (11%) were positive. Of these, 5337 (54%) were positive for influenza type A and 4625 (46%) were positive for influenza type B. Of the 2127 subtyped influenza A viruses, 2061 (97%) were type A (H1N1) and 66 (3%) were A (H3N2). Influenza type B viruses were isolated more frequently than type A viruses from the week ending February 10 through the week ending May 19. Influenza type A viruses predominated in the East North Central, South Atlantic, West North Central, and West South Central regions; influenza B viruses predominated in the mid-Atlantic, Mountain, and Pacific regions. The East South Central and New England regions reported approximately equal numbers of influenza A and B viruses. The proportion of specimens testing positive for influenza first increased to ≈10% during the week ending December 23, 2000, peaked at 24% during the week ending January 27, 2001, and declined to <10% during the week ending March 10. The peak percentage of specimens testing positive for influenza during the 2000-01 season was lower than that seen during the previous three seasons when the peak ranged from 28% to 32%.

CDC antigenically characterized 678 influenza viruses received from U.S. laboratories since October 1, 333 (95%) of the 354 influenza A (H1N1) viruses were similar to A/New Caledonia/20/99, the H1N1 component of the 2000-01 influenza vaccine, and 19 (5%) were similar to A/Bayern/07/95. Although A/Bayern-like viruses are distinct from the A/New Caledonia-like viruses, the A/New Caledonia/20/99 vaccine strain produces high titers of antibody that cross-react with A/Bayern/07/95-like viruses. Of the 23 influenza A (H3N2) viruses that were characterized, all were similar to the vaccine strain A/Panama/2007/99. Of the 301 influenza B viruses that were characterized, 33 (11%) were similar to the vaccine strain, B/Beijing/184/93, and 268 (89%) were most closely related to the B/Sichuan/379/99 reference strain.

U.S. influenza sentinel physicians reported that the percentage of patient visits for influenza-like illness (ILI) exceeded baseline levels (0-3%) for 4 consecutive weeks from the week ending January 20 through the week ending February 10. During each of the 4 weeks, 4% of patient visits were for ILI. During the previous three influenza seasons, the peak percentage of patient visits for ILI ranged from 5% to 7%.

On the basis of data from state and territorial epidemiologists’ reports, influenza activity peaked during the weeks ending February 3 and February 10, when 38 states reported regional or widespread influenza activity. The peak of regional influenza activity during consecutive weeks from the week ending November 18 through the week ending March 31. Widespread activity was reported by one or more states during each of the 4 consecutive weeks from the week ending January 6 through the week ending March 10. The peak number of states reporting regional or widespread activity during the previous 3 years ranged from 43 to 46.

As reported through the 122 Cities Mortality Reporting System, the percentage of deaths in the United States associated with pneumonia and influenza (P&I) did not exceed the epidemic threshold during the 2000-01 influenza season. During the previous three seasons, the percentage of deaths attributed to P&I was above the epidemic threshold for 10 consecutive weeks each season.

Worldwide

During October 2000-April 2001, influenza A (H1N1) and influenza B viruses circulated widely in Africa, the Americas, Asia, and Europe and influenza A (H3N2) viruses were reported sporadically. Influenza A (H1N1) viruses pre-
Most influenza A (H1N1) viruses isolated worldwide were similar to A/New Caledonia/20/99 (H1N1). Both A/New Caledonia/20/99 and A/Bayern/07/95-like (H1N1) viruses circulated in the United States. Although these viruses antigenically are distinct, antibodies produced against A/New Caledonia/20/99 react at equivalent levels with A/Bayern/07/95-like viruses; therefore, A/New Caledonia/20/99 was retained in the 2001-02 influenza vaccine.

Most influenza A (H3N2) viruses isolated during the 2000-01 season were similar to A/Panama/2007/99 and A/Moscow/10/99-like (H3N2) viruses. Antibodies produced following vaccination with the 2000-01 vaccine containing the A/Panama/2007/99 (H3N2) virus reacted equally well with recent influenza A (H3N2) viruses and the vaccine strain; therefore, VRBPAC recommended that an influenza A/Moscow/10/99-like (H3N2) virus be retained in the 2001-02 vaccine. Because of its growth properties, U.S. vaccine manufacturers will use the antigenically equivalent virus, A/Panama/2007/99.

Most influenza B isolates were related more closely to the antigenic drift variant B/Sichuan/379/99 than the 2000-01 B/Beijing/184/93-like vaccine strain, B/Yamanashi/166/98. Antibodies produced against the B/Yamanashi/166/98 vaccine strain cross-reacted with B/Sichuan/379/99-like viruses; however, antibodies were lower in titer and frequency against B/Sichuan/379/99-like viruses than B/Yamanashi/166/98-like viruses. Therefore, VRBPAC recommended that the influenza B component be updated for the 2001-02 vaccine to an influenza B/Sichuan/379/99-like virus. For the B/Sichuan/379/99-like virus, U.S. manufacturers will use one of the antigenically equivalent viruses B/Johannesburg/05/99, B/Victoria/504/2000, or B/Guangdong/120/2000.

Composition of the 2001-02 Influenza Vaccine

The Food and Drug Administration’s Vaccines and Related Biological Products Advisory Committee (VRBPAC) recommended that the 2001-02 trivalent influenza vaccine for the United States contain A/New Caledonia/20/99-like (H1N1), A/Moscow/10/99-like (H3N2), and B/Sichuan/379/99-like viruses. This recommendation was based on antigenic analyses of recently isolated influenza viruses, epidemiologic data, and postvaccination serologic studies in humans.

Influenza A (H3N2) viruses were identified more frequently than influenza A viruses in Canada, China, Egypt, Iceland, Ireland, Hong Kong/China, Portugal, Sweden, and the United Kingdom. Influenza type B viruses also were reported in Argentina, Australia, Brazil, Chile, India, Israel, Japan, Republic of Korea, Mauritius, Paraguay, Peru, Singapore, Taiwan, Thailand, and throughout Europe.

Influenza A (H3N2) viruses were identified in Argentina, Australia, Brazil, Bulgaria, Canada, Chile, China, Czech Republic, Denmark, France, Germany, Hong Kong/China, Ireland, Italy, Japan, Republic of Korea, Peru, Portugal, Russia, Singapore, South Africa, Spain, Switzerland, Taiwan, and Thailand. Unsubtyped influenza A viruses were reported from Belarus, Chile, Colombia, Malaysia, Nepal, Paraguay, and Slovenia.

CDC Editorial Note: Influenza A (H1N1) and B viruses co-circulated in the United States and worldwide during the 2000-01 influenza season. Influenza A (H3N2) viruses were isolated sporadically and no country reported widespread activity as a result of influenza A (H3N2) viruses. Seasons in which influenza A (H1N1) and/or influenza B viruses predominate typically have been less severe than seasons in which influenza A (H3N2) viruses circulate widely. The level of influenza activity reported this season was consistent with a number of other A (H1N1) and B predominant years.

Although influenza epidemics typically peak during December-March in the temperate regions of the Northern Hemisphere, sporadic cases and outbreaks can occur during the summer. The influenza season typically peaks during May-August in temperate regions of the Southern Hemisphere, and epidemics can occur at any time of the year in the tropics. U.S. physicians should consider influenza when diagnosing a febrile respiratory illness during the summer, particularly among persons who have traveled recently in the tropics, Southern Hemisphere, or with large international groups. Persons at increased risk for influenza-related complications who were not vaccinated during the preceding fall or winter should consider receiving influenza vaccine, if available, before traveling to the tropics, Southern Hemisphere, or with large international groups. Persons at increased risk for influenza-related complications who have received the previous season’s vaccine during the summer should be vaccinated with the current influenza vaccine during the following fall or winter.
Fatal Pediatric Lead Poisoning—New Hampshire, 2000

**MMWR. 2001;50:457-459**

**FATAL PEDIATRIC LEAD POISONING IS RARE** in the United States because of multiple public health measures that have reduced blood lead levels (BLLs) in the population. However, the risk for elevated BLLs among children remains high in some neighborhoods and populations, including children living in older housing with deteriorated lead paint.

This report describes the investigation of the first reported death of a child from lead poisoning since 1990. The investigation implicated leaded paint and dust in a home environment as the most likely source of the poisoning. Lead poisoning can be prevented by correcting lead hazards, especially in older housing, and by screening children at risk according to established guidelines.

On March 19, 2000, a 2-year-old girl was seen at a community hospital emergency department with a low-grade fever and vomiting of approximately 1 day's duration. The child had been well since arriving in New Hampshire from Egypt with her Sudanese refugee family 3 weeks earlier. Laboratory findings included a microcytic anemia (hemoglobin: 7.6 g/dL; lower limit of normal: 11.5 g/dL) with occasional basophilic stippling of red blood cells. A throat swab streptococcal antigen screening test was positive. She was discharged from the emergency department with prescriptions for an antibiotic and antiemetic to treat presumed strep throat. However, her vomiting worsened, and she was admitted to the same hospital on April 17, and then transferred to a tertiary-care hospital the next day. On April 19, approximately 5 hours after the transfer, she became unresponsive, apneic, and hypotensive. She was intubated and placed on a ventilator. Computed tomography of the head showed diffuse cerebral edema and dilated ventricles. Later that day, the results of a blood test drawn on April 18 showed a BLL of 391 µg/dL and an erythrocyte protoporphyrin level of 541 µg/dL. Chelation therapy was initiated with intramuscular British antilewisite and intravenous calcium ethylenediaminetetraacetic acid. Despite a decrease in her BLL to 72 µg/dL, and treatment for increased intracranial pressure, including surgical ventricular drainage, she remained comatose without spontaneous respirations, brain electrical activity, and intracranial blood flow. She was pronounced brain dead on April 21.

An autopsy found diffuse cerebral edema. A hair sample lead concentration was 31 µg/g in the distal centimeter and 67 µg/g in the proximal centimeter, indicating a large increase in lead exposure during the preceding month. Radiographs of the left knee were equivocal for growth arrest lines that can occur in chronic lead poisoning. A bone marrow sample showed no stainable iron, indicating iron deficiency.

On April 19, the Manchester Health Department and New Hampshire Department of Health and Human Services (NHDHHS) initiated an investigation, including interviews and blood lead tests of the patient’s family and an inspection of her residence. In addition, to assess a possible contribution of lead exposure from the child’s previous residence in Egypt, the Field Epidemiology Training Program of the Egyptian Ministry of Health obtained soil and dust samples from that location.

After living in Egypt for approximately 18 months, on March 9, 2000, the family had moved to Manchester into an apartment constructed before 1920. A wall in a sibling’s bedroom had multiple holes from which the patient had been seen removing and ingesting plaster. Two of seven samples of plaster with the adhering surface paint contained lead at levels of 5% and 12%. Peeling paint (35% lead) was present on the balusters and floor (3% lead) of a porch outside the apartment entrance where the patient sometimes had played. She also had played near and looked out of a living room window that occasionally was opened during meal preparation. A wipe sample of dust from the window well showed 6732 µg lead/ft², well above the hazardous level of 800 µg/ft². NHDHHS ordered the apartment owner to correct the lead hazards identified during the inspection. The patient’s family relocated to another dwelling.

BLLs in the mother and three siblings (ages 5, 11, and 15 years) ranged from 4-12 µg/dL. The family did not use or possess nontraditional remedies, food supplements, cosmetics, or ceramic eating or drinking containers acquired abroad. No one in the household was employed or had lead-related hobbies. Measurements of stable lead isotopes in selected environmental samples and the patient’s blood showed that the isotopic lead composition of the porch paint and window well dust in the her Manchester apartment matched the composition of lead in her blood more closely than did the isotopic composition of other samples, including those from her previous residence in Egypt.

**REFERENCES**


*The four components of the influenza surveillance system have been described.* Information reported as of June 5, 2001.

†Temperature ≥ 100.0°F (≥37.8°C) and either cough or sore throat in the absence of a known cause.

‡Levels of activity are (1) no activity; (2) sporadic—sporadically occurring ILI or cultureconfirmed influenza in counties with no outbreaks detected; (3) regional—outbreaks of ILI or cultureconfirmed influenza in counties with a combined population of ≥50% of the state’s population; and 4) widespread—outbreaks of ILI or cultureconfirmed influenza in counties with a combined population of ≥50% of the state’s population.

§Before the 1999-2000 season, the case definition for P&I deaths was modified. CDC analysis estimated that the revised case definition resulted in an average increase in baseline P&I mortality estimates of 0.8% for 1999-2000. Thus, the 122 cities P&I mortality baseline and epidemic threshold for the 2000-01 season have been adjusted upward. The epidemic threshold is 1.645 standard deviations above the seasonal baseline. The expected seasonal baseline is projected using a robust regression procedure in which a periodic regression model is applied to observed percentages of deaths from P&I since 1983.
CDC Editorial Note: Lead encephalopathy is a life-threatening complication of lead poisoning that can occur in young children who have very high BLLs (>70-100 µg/dL). Nonspecific symptoms (e.g., lethargy, sporadic vomiting, and constipation) can occur at BLLs >50-70 µg/dL and may precede the abrupt onset of frank encephalopathy characterized by persistent vomiting, ataxia, altered consciousness, coma, and seizures. In this report, the child’s anemia with basophilic stippling also suggested lead poisoning. However, symptoms or signs cannot be used to reliably diagnose or exclude lead poisoning; a BLL must be measured whenever lead poisoning is suspected. In young children, BLLs >70 µg/dL or elevated BLLs with symptoms suggesting encephalopathy require prompt inpatient treatment with chelating agents to rapidly reduce BLLs. Providing appropriate intensive care for children with encephalopathy can prevent death, although severe permanent brain damage can occur despite treatment.

During the 1950s and 1960s, acute, often fatal, lead encephalopathy was a common cause of pediatric admissions to urban hospitals. The subsequent decline in fatal lead poisoning cases is attributable to reduced lead exposure from multiple sources, institution of lead screening programs, and improved treatment of lead poisoning. Despite the reduction in severe lead poisoning, in some U.S. counties, >20% of young children tested have BLLs ≥10 µg/dL, high enough to adversely affect learning and development.

The likely sources of lead poisoning for the child in this report—deteriorated lead paint and elevated levels of lead-contaminated house dust—are found in an estimated 24 million U.S. dwellings, 4.4 million of which are home to one or more children aged <6 years (U.S. Department of Housing and Urban Development, unpublished data, 2001). Lead hazards are especially common in homes built before 1960 (58%). Although the patient’s pica and iron deficiency probably contributed to the severity of her lead poisoning, by increasing ingestion and absorption of lead, all children living in homes with lead hazards are at increased risk for developing elevated BLLs. Children who are refugees, adoptees, or recent immigrants may be at increased risk for elevated BLLs, possibly related to lead exposure in their country of origin or to continued use of certain lead-containing traditional remedies or cosmetics. However, such children also are at risk for exposure to leaded paint hazards in older U.S. housing. In addition to ensuring that such children are screened after arrival in the United States, lead poisoning prevention programs and health-care providers should ensure that families receive timely education about lead hazards. Federal regulations require that property sellers and landlords provide families with information about lead poisoning and about any known lead hazards in a dwelling before its sale or lease. Agencies providing health and social services to refugees and immigrants should become familiar with these regulations and ensure that appropriate information is provided to families in a language they can understand.

REFERENCES
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First Report of AIDS

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TWENTY YEARS AGO, ON JUNE 5, 1981, MMWR published a report of five cases of Pneumocystis carinii pneumonia (PCP) among previously healthy young men in Los Angeles. All of the men were described as “homosexuals”; two had died. Local clinicians and the Epidemiology Intelligence Service (EIS) Officer stationed at the Los Angeles County Department of Public Health, prepared the report and submitted it for MMWR publication in early May 1981. Before publication, MMWR editorial staff sent the submission to CDC experts in parasitic and sexually transmitted diseases. The editorial note that accompanied the published report stated that the case histories suggested a “cellular-immune dysfunction related to a common exposure” and a “disease acquired through sexual contact.” The report prompted additional case reports from New York City, San Francisco, and other cities. At about the same time, CDC’s investigation drug unit, the sole distributor of pentamidine, the therapy for PCP, began to receive requests for the drug from physicians also to treat young men. In June 1981, CDC developed an investigative team to identify risk factors and to develop a case definition for national surveillance. Within 18 months, epidemiologists conducted studies and prepared MMWR reports that identified all of the major risks factors for acquired immunodeficiency syndrome (AIDS). In March 1983, CDC issued recommendations for prevention of sexual, drug-related, and occupational transmission based on these early epidemiologic studies and before the cause of the new, unexplained illness was known.

MMWR has published more than 400 reports about human immunodeficiency virus (HIV) and AIDS and remains a primary source of information about the epidemiology, surveillance, prevention, care, and treatment of HIV and AIDS. This anniversary issue provides new reports on the epidemiologic features and impact of HIV/AIDS on communities in the United States and in other countries. A compilation of notable MMWR reports on HIV and AIDS is available at http://www.cdc.gov/mmwr/hiv_aids20.html. A video that includes interviews with participants in these first AIDS investigations and
New strategies are needed to maintain and accelerate progress in HIV/AIDS prevention that sustain and reinvigorate communities most severely affected during the early years of the epidemic, particularly men who have sex with men and to meet the evolving needs of an increasingly diverse epidemic. Efforts also must be tailored to equip racial/ethnic minority communities with the skills and knowledge to prevent HIV infection.

Highly active antiretroviral therapies have improved the length and quality of life for HIV-infected persons. However, some infected persons on treatment assume that they are not infectious and engage in behavior that increases risk for transmission. In addition, some persons may have decreased concern about infection because of advances in treatment. Increases in risk behaviors and rates of sexually transmitted diseases among men who have sex with men have been reported from multiple cities, which may herald an increase in HIV transmission.

CDC begins the third decade of HIV/AIDS with a new strategic plan designed to reduce annual infections by half within 5 years. This three-part plan includes: (1) intensifying efforts to help all infected persons learn their HIV status; (2) establishing new prevention programs to help HIV-infected persons establish and maintain safer behaviors, combined with improved linkages to treatment and care; and (3) expanding highly targeted prevention programs to reach all HIV-negative persons at greatest risk. Additional information about the HIV strategic plan is available at http://www.cdc.gov/nchstp/od/news/prevention.pdf

HIV prevention programs contribute to healthier behaviors and reduce the number of new HIV infections in the United States. An expanded and sustained commitment to prevention on a global, national, community, and personal level is required to further reduce the number of new infections and of persons living with HIV.

REFERENCES

*Use of trade names is for identification only and does not imply endorsement by CDC or the U.S. Department of Health and Human Services.