FROM THE CENTERS FOR DISEASE CONTROL AND PREVENTION

CDC MMWR MORBIDITY AND MORTALITY WEEKLY REPORT

Update: Outbreak of Poliomyelitis—Dominican Republic and Haiti, 2000-2001

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FROM JULY 12, 2000, THROUGH SEPTEMBER 18, 2001, a total of 21 cases of poliomyelitis (including two fatal cases) were reported from the Caribbean island of Hispaniola, divided between Haiti and the Dominican Republic.[1,2] In the Dominican Republic, 13 of 168 reported cases of acute flaccid paralysis (AFP) were confirmed as polio by isolation of poliovirus type 1 from either patients or their healthy contacts. The median age of the patients was 3 years (range: 9 months-14 years). None was vaccinated adequately. The most recent confirmed case-patient in the Dominican Republic had paralysis onset on January 25, 2001. In Haiti, eight of 40 AFP cases were confirmed virologically; seven of the confirmed cases occurred during January-July 2001. The median age of the patients was 7 years (range: 2-12 years). One patient had received at least 3 doses of oral poliovirus vaccine (OPV). The most recent confirmed case occurred in Haiti and the patient had paralysis onset on July 12, 2001. Eighteen AFP cases from the Dominican Republic and three from Haiti are pending final classification.

This outbreak was the first in the Americas since 1991 and was associated with the circulation of a type 1 OPV-derived virus, having substitutions affecting 1.8% to 4.1% of nucleotides encoding the major capsid protein (VP1). The circulating vaccine-derived poliovirus associated with the outbreak recovered the capacity to cause paralytic disease and widespread person-to-person transmission and was biologically indistinguishable from type 1 wild poliovirus. Contemporary vaccine-derived poliovirus isolates from persons with AFP cases in other countries of the Americas are more closely related (>99.5% VP1 sequence similarity) to the respective OPV strains, are unrelated to the Hispaniola outbreak viruses, and show no evidence of extensive person-to-person transmission. The outbreak in Hispaniola occurred in areas of very low OPV coverage.

In response to the outbreak, health authorities in both countries conducted house-to-house vaccination with OPV. Three rounds of mass vaccination campaigns were conducted in the Dominican Republic in December 2000, and February and April 2001. In each round, approximately 1.2 million OPV doses were administered to an estimated population of 1.1 million children aged <5 years. Haiti conducted two rounds of mass vaccination in February and March 2001. However, these campaigns were hampered by logistic difficulties and heavy rains and reached an estimated 40% of the 1.2 million children aged <5 years. During May-July 2001, a door-to-door and school-based campaign among all 2.3 million children aged <10 years was conducted sequentially in all of the country's departments. Preliminary results suggest that 2.4 million OPV doses were administered, and a second door-to-door campaign is under way.

Travelers to the Dominican Republic and Haiti who are not vaccinated adequately are at risk for polio. Travelers should have received poliovirus vaccination according to national vaccination policies.[3]

Reported by: Ministry of Health, Pan American Health Organization, Santo Domingo, Dominican Republic. Ministry of Health, Pan American Health Organization, Port-au-Prince, Haiti. Caribbean Epidemiology Center Laboratory, Pan American Health Organization, Trinidad and Tobago. Div of Vaccines and Immunization, Pan American Health Organization, Washington, DC. Respiratory and Enteric Viruses Br, Div of Viral and Rickettsial Diseases, National Center for Infectious Diseases; Vaccine Preventable Disease Eradication Div, National Immunization Program, CDC.

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Cigarette Smoking Among Adults—United States, 1999

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1 table and 1 figure omitted

ONE OF THE NATIONAL HEALTH OBJECTIVES for 2010 is to reduce the prevalence of cigarette smoking among adults from 24% in 1998 to ≤12% (objective 27.1a).[1] To assess progress toward this objective, CDC analyzed self-reported data from the 1999 National Health Interview Survey (NHIS) about cigarette smoking among U.S. adults. This report summarizes the findings of this analysis, which indicate that, in 1999, approximately 23.5% of adults were current smokers, representing a modest decline in prevalence since 1993. If states were to invest resources consistent with CDC recommendations and implement proven interventions, the decline in cigarette smoking could be accelerated.

The 1999 NHIS adult core questionnaire was administered by personal interview to a nationally representative sample (n=30,801) of the U.S. noninstitutionalized civilian population aged ≥18 years; the overall response rate was 69.6%. Respondents were asked, "Have you smoked ≥100 cigarettes in your entire life?" and "Do you now smoke cigarettes every day, some days, or not at all?" Current smokers were persons who reported both having smoked ≥100 cigarettes during their

lifetime and who smoked every day or some days. Former smokers were those who had smoked ≥100 cigarettes during their lifetime but currently did not smoke. Attempts to quit were determined by asking current smokers, "During the past 12 months, have you stopped smoking for 1 day or longer because you were trying to stop smoking?" Data were adjusted for nonresponses and weighted to provide national estimates. Confidence intervals (CIs) were calculated using SUDAAN.

In 1999, an estimated 46.5 million adults (23.5% [95% CI=±0.6]) were current smokers. Overall, 19.2% (95% CI=±0.6) of adults were everyday smokers and 4.3% (95% CI=±0.3) were some day smokers. The prevalence of smoking was higher among men (25.7% [95% CI=±0.9]) than women (21.5% [95% CI=±0.7]). Among racial/ethnic groups, Hispanics (18.1% [95% CI=±1.3]) and Asians/Pacific Islanders (15.1% [95% CI=±3.1]) had the lowest prevalence of cigarette use; American Indians/Alaska Natives had the highest prevalence (40.8% [95% CI=±8.6]). Adults who had earned a General Educational Development diploma had the highest smoking prevalence (44.4% [95% CI=±4.5]); persons with masters, professional, and doctoral degrees had the lowest prevalence and met the 2010 objective (8.5% [95% CI=±1.3]). Prevalence was highest among persons aged 18-24 years (27.9% [CI=±1.9]) and 25-44 years (27.3% [CI=±1.0]) and lowest among those aged ≥65 years (10.6% [CI=±0.9]). The prevalence of smoking was highest among adults living below the poverty level* (33.1% [(95% CI=±2.0]) compared with those living at or above the poverty level (23.4% [95% CI=±0.7]), and lowest among those with unknown poverty status (20.2% [95% CI=±1.2]).

In 1999, an estimated 45.7 million adults (23.1% [95% CI=±0.6]) were former smokers; 25.8 million were men and 19.9 million were women. Former smokers constituted 49.5% (95% CI=±1.0) of persons who had ever smoked ≥100 cigarettes. Among current smokers, an estimated 15.7 million (41.3% [95% CI=±1.5]) had stopped smoking at least 1 day during the preceding 12 months because they were trying to quit.

During 1998-1999, significant changes in smoking prevalence[2] did not occur; however, since 1993, the prevalence of current smoking has slowly declined. To assess changes over time, 1993 data were compared with 1999 data.[ref]3 Overall prevalence of current smoking declined significantly from 1993 (25.0% [95% CI=±0.7]) to 1999 (23.5% [95% CI=±0.6]). Data for 2000 (23.3% [95% CI=±0.6]) and preliminary data for January-March 2001 (22.3% [95% CI=±1.1]) suggest a continuing decline.[4]

During 1993-1999, no significant changes were observed in current smoking prevalence for any racial/ethnic group or for the population living below the poverty level; however, reductions were reported in adults with 12 years of education (from 29.2% [95% CI=±1.2] to 26.3% [95% CI=±1.1]), and in persons aged 45-64 years (from 26.0% [95% CI=±1.3] to 23.3% [95% CI=±1.0]). Prevalence of smoking among persons aged 18-24 years has not increased significantly; this age group continues to have the highest smoking prevalence.[2]

Reported by: Epidemiology Br, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.

CDC Editorial Note: After 4 years during which the prevalence of current smoking among U.S. adults remained unchanged,[2] data from 1999 indicated a slow but significant decline; however, the 2010 objective of ≤12% for adult smoking prevalence will not be met unless the rate of decline increases significantly. The 2000 report of the Surgeon General[5] concluded that the 2010 objective could be met if comprehensive approaches to tobacco control were implemented fully.

Increasing the unit price of tobacco products, smoking bans and restrictions, and mass media education campaigns for tobacco-use cessation are among the recommended measures[5,6] to increase quitting among a wide range of smokers. The decline in smoking prevalence that began in 1997 may be explained, in part, by the December 1997–December 1999 increase in taxes and wholesale prices that resulted in a 49% price increase.[7]

The findings in this report are subject to at least two limitations. First, questionnaires and data collection procedures for NHIS have changed since 1993. In 1995, the sample was redesigned; in 1997, questions on tobacco use were moved from supplementary questionnaires to the adult core questionnaire. It is impossible to assess how these changes affected prevalence estimates and trend analysis or comparisons; therefore, statistical trend analysis from the years preceding 1997 should be approached with caution. Second, because the NHIS sample size of some racial/ethnic populations was small (e.g., American Indians/Alaska Natives), data for a single year might be unstable. Combining data from several years would produce more reliable estimates.

Expanded access to treatment for nicotine dependence (e.g., FDA-approved pharmacotherapy and individual, group, and telephone counseling) will help more persons stop smoking. One method to increase access to treatment is to reduce out-of-pocket costs by covering therapies as a standard insurance benefit.[1,5,7] *Best Practices for Comprehensive Tobacco Control*[8] recommends that cessation interventions be incorporated into comprehensive, statewide programs. Following the implementation of a comprehensive program, the Arizona Department of Health Services Tobacco Education and Prevention Program reported that prevalence among adult smokers decreased from 23.1% to 18.3% during 1996-1999, and the proportion of Arizona smokers who reported that a health-care provider had both asked them about their tobacco use and advised them to quit increased significantly during this pe-

riod.[9] To eliminate tobacco-related disease nationwide, comprehensive tobacco control programs similar to those in Arizona must focus on groups with high levels of smoking prevalence, including persons aged 18-24 years, with low incomes, with low education levels, and American Indians/Alaska Natives.

Seven states (Arizona, Indiana, Maine, Massachusetts, Mississippi, Ohio, and Vermont) are funding tobacco prevention and control programs at the minimum level recommended by CDC.[10] If all states invested resources consistent with these recommendations and spent resources on proven interventions, the decline in prevalence could be accelerated.

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*Poverty thresholds for 1998 from the Bureau of the Census, Economics and Statistics Administration, U.S. Department of Commerce.
†The first year NHIS asked about some day smoking was 1991; refinements were made to the questions in 1992. Since 1993, the full sample of adults has been asked identical questions about some day smoking.

Pesticide-Related Illnesses Associated With the Use of a Plant Growth Regulator—Italy, 2001

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DURING JANUARY-FEBRUARY 2001, EIGHT cases of acute illness in the county of Ragusa, Italy, were reported to the Italian National Institute for Health (INIH) by the Milan Poison Control Center (MPCC) and were attributed to exposure to Dormex®, a plant growth regulator with hydrogen cyanamide as the active ingredient. These cases were identified during a pilot project for acute pesticide-related illness surveillance. Subsequent active case finding at healthcare clinics by the Ragusa Occupational Health Unit identified six additional cases. MPCC identified nine cases in other areas of Italy. Of the 23 cases of acute illness, 22 resulted from occupational exposure during mixing and/or applying of Dormex®, and one was from unintentional ingestion. This report summarizes the investigation of these cases, which implicates a pesticide as the causative agent and demonstrates the usefulness of surveillance for detecting pesticide-related illnesses.

All 22 workers were male with a median age of 41 years (range: 16-76 years). It is not known whether personal protection equipment was used. Eighteen of the workers reported dermatologic manifestations, including macular or papular rash (11), erythema/hyperemia (nine), pruritus (two), and caustic burns to the hand (two). Two workers reported eye irritation. Fourteen workers had systemic signs and/or symptoms characteristic of adverse effects of the active ingredient, including tachycardia (four), weakness (four), dizziness (four), palpitations (three), headache (three), vomiting and/or nausea (three), dyspnea (three), and hypotension (one). Of 21 persons initially treated in an emergency department, 12 (52%) were hospitalized; one person was treated by a local physician. Thirteen patients had low severity effects (i.e., minimal effects that rapidly resolved), and nine had moderate severity effects (i.e., nonlife threatening effects that are more pronounced, prolonged, or of a systemic nature) (CDC, unpublished data, 2001).

The nonoccupational case occurred in a man aged 44 years who unintentionally ingested the product that had been placed in a plastic water bottle in the refrigerator. He became seriously ill with third degree shock, coma, miosis, and hepatic necrosis and required care in an intensive care unit.

In May 2001, INIH notified the Italian Ministry of Health (IMH) about the outbreak. IMH, which acts as the regulatory agency for pesticides and agricultural products, suspended use of the product in Italy.

Reported by: F Davanzo, L Faraoni, Milan Poison Control Center; G Miceli, M Conticello, L Bongiovanni, Ragusa Occupational Health Unit; T Ballard, L Settimi, M Rubbiani, I Marcello, S Bascherini, Italian National Institute of Health. L Mehler, MD, California Dept of Pesticide Regulation, Sacramento. Surveillance Br, Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

CDC Editorial Note: This report describes the adverse health effects of hydrogen cyanamide, the active ingredient in Dormex®, which is a plant growth regulator designed to stimulate more uniform budbreak following dormancy, resulting in more uniform flowering and maturity at harvest. Dormex® is applied by nebulization with an atomizer. Adverse health effects from contact with hydrogen cyanamide include severe irritation and ulceration of the eyes, skin, and respiratory tract.[1,2] It also inhibits aldehyde dehydrogenase and can produce the acetaldehyde syndrome (e.g., vomiting, parasympathetic hyperactivity, dyspnea, hypotension, tachycardia, and confusion) when exposure coincides with alcohol use.[2]

Hydrogen cyanamide is classified in the European Union as "toxic" if swallowed, "harmful" in contact with skin, "irritating" to eyes and skin, and ca-

pable of producing sensitization after skin contact. The U.S. Environmental Protection Agency (EPA) places both the active ingredient (hydrogen cyanamide) and the product (Dormex®), which contains 50% hydrogen cyanamide, into the acute toxicity category I (danger).* The Dormex® product label provided by the manufacturer to EPA indicates that the following personal protective equipment must be used by applicators and other handlers of this product: chemical-resistant suit, chemical-resistant gloves, chemical-resistant footwear, eye and face protection, and a respirator with either an organic vapor-removing cartridge with a prefilter approved for pesticides or a canister approved for pesticides.

On the basis of experimental trials of the product, Dormex® was classified in Italy as "harmful" if swallowed, "harmful" in contact with the skin, "irritating" to the eyes and skin, capable of causing serious damage to the eyes, and of causing sensitization after skin contact. This corresponds to EPA acute toxicity category II. The product sold in Italy was for use only by licensed applicators and required wearing suitable protective clothing, gloves, and eye and face protection.

Since 1981, only five cases of acute pesticide-related illness associated with hydrogen cyanamide have been identified in the United States (CDC, unpublished data, 2001). All five patients were exposed in California. No cases were identified in the other seven states with acute pesticide-related illness surveillance programs or by the Toxic Exposure Surveillance System, which collects poisoning reports submitted by approximately 85% of U.S. poison control centers. The low number of U.S. cases compared with Italy may be related to greater precautions required by the label of the U.S.-distributed product.

The findings in this report are subject to at least two limitations. First, because active surveillance for acute pesticide-related illness cases was conducted in Ragusa only, patients who sought health care in other parts of Italy may have been missed. Second, lack of detailed information on the events surrounding exposure may have precluded identification of additional risk factors for hydrogen cyanamide-related illness.

Although use of Dormex® in Italy began in 2000, only three cases of acute illness associated with this product were identified by MPCC in 2000 (i.e., before establishment of the pilot surveillance program). One occurred in Ragusa and the other two were from other regions in southern Italy. These data suggest that fewer cases occurred in 2000 compared with 2001. Because emergency department medical records in Ragusa for 2000 were not available to the Occupational Health Unit, the total number of Ragusa cases that occurred in 2000 is unknown. The establishment of the pilot surveillance system in January 2001 probably enabled the detection of this outbreak through active case-finding and the use of a standardized form. Ragusa was selected for this pilot program, in part, because it is an area characterized by greenhouse cultivation of fruits and vegetables with extensive use of pesticides and because of heightened awareness of pesticide-related illnesses by the Ragusa Occupational Health Unit.

These findings demonstrate the usefulness of surveillance for detecting emerging pesticide problems.[3] In addition, this outbreak suggests the need for international uniformity in both the acute toxicity category assigned to a pesticide and in the detailed recommendations and requirements provided on the pesticide label.

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*EPA classifies all pesticide products into one of four acute toxicity categories based on established criteria (40 CFR Part 156). Pesticides with the greatest toxicity are in category I, and those with the least are in category IV.

Influence of Homicide on Racial Disparity in Life Expectancy—United States, 1998

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LIFE EXPECTANCY (LE) IS AN IMPORTANT indicator of the health of populations. Since the early 1900s, when estimates of LE began to be tabulated in the United States, the LE of blacks has been lower than that of whites.[1] Homicide, which disproportionately affects blacks, particularly young males, contributes to this difference in LE. To examine the associations between homicide, LE, and race, CDC analyzed 1998 mortality files from the National Center for Health Statistics (NCHS). This report summarizes the results of that analysis, which indicate that, in 1998, the LE for blacks was approximately 6 years shorter than for whites and that, after heart disease and cancer, homicide was the next largest contributor to the 6-year discrepancy. Violence prevention strategies (e.g., programs for youth offenders) have been implemented for the general population. More research is needed to determine an approach to target the male black population and to reduce LE disparity.

NCHS mortality files for 1998[2] and the multiple-decrement life table[3] were used to examine differences between whites and blacks. These methods were used to partition the contribution to LE at birth by selected causes of death using the *International Classification of Diseases, Ninth Revision, (ICD-9)* codes* for the four major race-sex groups (black-males, black-females, white-males, white-females) in the United States. The contribution in years for each cause of death to the black/white differential and statistical tests of difference (Z-scores) were determined using Survival software,[4] with whites as the referent group. Causes of death used were based on the leading causes of

death in 1998 for the total population and for both racial populations. Other causes of death were categorized as "all other causes."

In the United States during 1998, whites lived 6.2 years longer than blacks. Among the leading causes of death that contributed to the difference were heart disease (1.7 years; 27.4%), cancer (1.2 years; 19.4%), homicide (0.6 years; 9.7%), stroke (0.5 years; 8.1%), and "all other causes" (1.9 years; 30.6%). The LE differential was 6.4 years for males and 4.4 years for females. Among males, some of the leading causes of death that contributed to the LE differential were heart disease (1.2 years; 19.0%), cancer (1.0 years; 15.6%), and homicide (0.9 years; 14.1%), and among females were heart disease (1.2 years; 27.3%), cancer (0.5 years; 11.4%), and perinatal disease (e.g., birth trauma, birth asphyxia, ectopic pregnancy, and maternal death) (0.4 years; 9.1%). Stroke and human immunodeficiency virus (HIV) accounted for 0.3 years (6.8%) and 0.3 years (6.8%), respectively, of the LE differential among females and 0.4 years (6.3%) and 0.6 years (9.4%), respectively, among males. Homicide among black females contributed 0.2 years (4.5%) to the LE differential.

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CDC Editorial Note: The findings in this report document racial disparities in LE, which were attributable mainly to blacks having a shorter LE than whites for each examined cause of death (except suicide). For the total U.S. population in 1998, homicide ranked 13th among causes of death,[5] accounting for <1% of all deaths. However, homicide accounted for approximately 10% of the LE differential. This finding suggests that causes of death that rank low for the total population may be important targets to address in attempting to eliminate the LE gap between these populations.

During 1985, the U.S. Department of Health and Human Services conducted the first analyses using health indicators that documented the health status of minority populations and found that approximately 60,000 excess deaths (i.e., the difference between the number of deaths observed in a racial/ethnic group and the number of deaths that would have occurred in that group if it had the same death rate as the non-Hispanic white population) occurred among blacks each year in the United States.[6] Health disparities between blacks and the general population have been attributed to less access to health care and to health-care coverage. Risk factors for violence include living at or below the poverty level, living in single parent households, and having poor academic performance and/or exposure to neighborhood violence (e.g., gangs).[7]

The 1998 publication of *The Initiative to Eliminate Racial and Ethnic Disparities in Health* indicated a commitment to eliminating longstanding racial/ethnic disparities in health status by 2010. The initiative focuses on six key areas of health that disproportionately affect multiple racial/ethnic minority groups at all ages[8]: infant mortality, cancer screening and management, cardiovascular disease, diabetes, HIV, and vaccination coverage. The findings in this report are consistent with previous findings that show homicide to be a leading contributor to the difference in LE between blacks and whites[9] and underscore the need to include homicide among the key areas.

The findings in this report are subject to at least three limitations. First, incorrect diagnoses or errors can result in inaccuracies in death records. Second, although approximately 99% of deaths in the United States are reported systematically,[5] denominator data (population estimates) that refer to race or color may be inaccurate.[5] Third, several assumptions (e.g., that life expectancy is aged 85 years) that could be technically flawed were made in constructing the life table model in this analysis.[3]

Preventing homicide requires integrated approaches from multiple disciplines, including criminal justice, education, social services, community advocacy, and public health. Strategies for preventing violence among youth (e.g., social-cognitive, mentoring, and family-based approaches) have been described in *Best Practices to Prevent Violence by Children and Adolescents: A Sourcebook for Community Action*[10] *and in the* Surgeon General's Report on Youth Violence.[7] These prevention programs and strategies could be implemented by educators, public health practitioners, and law enforcement agencies to target black males. Reducing the racial LE differential in homicide will improve the health of blacks in the United States and thus reduce racial disparities in health.

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*Codes 042-044; 140-208; 390-398, 402, 404-429; 430-438; 760-779; E950-E959; E810-E825, E958.5, E988.5; E960-E978.