Progress Toward Poliomyelitis Eradication—Ethiopia, 1997–August 2000

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2 tables, 1 figure omitted


Routine Vaccination Coverage
During 1990–1999, reported coverage of children aged 0–11 months with 3 doses of oral poliovirus vaccine (OPV3) ranged from 20%–90%. The last comprehensive coverage survey conducted in 1995 estimated OPV3 coverage at 36%. Preliminary data from the 2000 Ethiopia Demographic and Health Survey estimates average OPV3 coverage at 35%.

Supplemental Vaccination Activities
In 1996, Ethiopia conducted Subnational Immunization Days (SNIDs) for the first time, targeting 2.5 million children aged less than 5 years in nine major cities. Since then, the country has conducted two rounds of National Immunization Days (NIDs) annually. Implementation of NIDs during 1997–1999 and SNIDs in 2000 has reached greater than 90% of the target population, including areas with limited access to routine health services. In 1999, intensified campaigns with delivery of vaccine house-to-house were conducted in three regions (Afar, Benshangul, and Somali) that had performed poorly in previous years. As a result, 541,996 more children were reached in these regions compared with 1998 NIDs, which used only fixed-site vaccinations.

Despite improvements in vaccine delivery, pockets of unvaccinated children remain. During the 2000 house-to-house SNIDs, efforts were made to detect the proportion of children never vaccinated by routine services or during NIDs. Of children vaccinated during the 2000 SNIDs, an average of 25% (range: 1%–100%) had never received vaccine.

Acute Flaccid Paralysis Surveillance
Surveillance for acute flaccid paralysis (AFP) was initiated in 1997. During 1997-August 2000, the nonpolio AFP rate increased from 0.10 to 0.44 per 100,000 children aged <15 years (a sensitive system is defined as ≥1 per 100,000 children aged <15 years).

Surveillance performance among the 11 regions of Ethiopia varies substantially. Wild poliovirus isolates have been isolated in zones (subregional administrative units) where AFP surveillance is improving and reached nonpolio AFP levels >0.5. However, only 26 of Ethiopia’s 71 zones have achieved this level; the more densely populated zones in central Ethiopia have nonpolio AFP rates <0.5, and 25 zones have not reported any AFP cases during 2000. These 25 zones also have very low (<20%) routine OPV3 coverage.

The proportion of adequate stool specimens from AFP case-patients (i.e., two stool specimens collected at an interval of at least 24 hours within 14 days of onset of paralysis and adequately shipped to the laboratory) has improved from 12% in 1998 to 44% in 2000. All stool specimens routinely are split and tested in both the Ethiopia Health and Nutrition Research Institute (EHNRI) polio laboratory and the World Health Organization (WHO) accredited national laboratory in Uganda. The EHNRI polio laboratory is expected to attain WHO accreditation status by the end of 2000.

Incidence of Polio
Until March 2000, AFP surveillance had not detected wild poliovirus in Ethiopia. In March 2000, the Johannesburg polio reference laboratory confirmed isolation of the first wild poliovirus type 1 (P1) in an AFP case from Oromia region with onset of paralysis in October 1999. A second isolate was reported in August 2000, with paralysis onset in March 2000. Neither of these virologically confirmed polio case-patients had received any doses of OPV. Genetic sequencing of polioviruses isolated from these cases revealed that they were indigenous to Ethiopia and unlike those polioviruses isolated in bordering countries.

CDC Editorial Note: Rapid progress has been achieved in implementing polio eradication strategies in Ethiopia, one of the major polio reservoirs in the Africa Region.3 Recent improvements in AFP surveillance led to the detection of indigenous wild poliovirus transmission. In addition, the number of children reached by NIDs and SNIDs has increased annually. The house-to-house approach in parts of the country during 1999 and 2000 resulted in increased coverage of children aged <5 years, especially in hard-to-reach areas.

Routine vaccination activities have been constrained by challenges related...
to program management, training, health sector reform, cold chain maintenance, a largely rural population, and difficult terrain. Low routine OPV3 coverage, suboptimal AFP surveillance, and indigenous wild poliovirus transmission underscore the need for continued high quality NIDs and extra SNIDs.

House-to-house vaccination activities should continue to reach children residing in hard-to-reach areas who have never been vaccinated. A WHO–United Nations Children’s Fund (UNICEF) technical review identified the need for an increased number of mid-level surveillance officers to assist in training, clinician sensitization, and supervision of active AFP surveillance in remote areas. The placement of mid-level surveillance officers in other countries has led to rapid improvement in AFP surveillance indicators.

Polio eradication priorities in Ethiopia include (1) implementing high-quality NIDs (planned for November and December 2000 and tentatively planned for 2001), (2) ensuring high-quality house-to-house vaccination campaigns in hard-to-reach areas, (3) strengthening routine vaccination, (4) strengthening facility-based active AFP surveillance to reach certification standards (nonpolio AFP rate of $\leq 1.0$) in all zones, (5) supporting the national laboratory to attain WHO accreditation, and (6) coordinating cross-border vaccination and surveillance activities to detect possible importation of wild poliovirus from neighboring countries. Meeting these challenges will require the continued support of polio eradication partners.†

### References

3 available

†Focal mass campaigns in high-risk areas over a short period (days to weeks) in which two doses of OPV are administered to all children, usually aged $<5$ years, regardless of vaccination history, with an interval of 4-6 weeks between doses.

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### Cigarette Smoking Among Adults—United States, 1998

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*One of the national health objectives for 2010 is to reduce the prevalence of cigarette smoking among adults to no more than 12% (objective 21.1a).*

To assess progress toward meeting this objective, CDC analyzed self-reported data from the 1998 National Health Interview Survey (NHIS) Sample Adult Core Questionnaire about cigarette smoking among U.S. adults. This report summarizes the findings of this analysis, which indicate that, in 1998, $24.1\%$ of adults were current smokers.

The 1998 NHIS Core Questionnaire was administered to a nationally representative sample ($n=32,440$) of the U.S. noninstitutionalized civilian population aged $\geq 18$ years; the overall response rate for the survey was $73.9\%$.

Participants were asked, “Have you smoked at least 100 cigarettes in your lifetime?” and “Do you now smoke cigarettes every day, some days, or not at all?” Current smokers were persons who reported both having smoked greater than or equal to 100 cigarettes during their lifetime and having smoked every day or some days at the time of the interview. Former smokers were those who had smoked $\geq 100$ cigarettes during their lifetime but did not currently smoke. Attempts to quit were determined by asking current smokers, “During the past 12 months, have you stopped smoking for one day or longer because you were trying to stop smoking?” Data were adjusted for nonresponse and weighted to provide national estimates. Confidence intervals (CIs) were calculated using SUDAAN.

In 1998, an estimated 47.2 million adults ($24.1\%$), comprising 24.8 million men ($26.4\%$) and 22.4 million women ($22.0\%$), were current smokers. Overall, $19.7\%$ ($95\%$ CI = $\pm 0.6$) of adults were every-day smokers, and $4.2\%$ ($95\%$ CI = $\pm 0.3$) were some-day smokers (every-day smokers constituted $82.4\%$ [$95\%$ CI = $\pm 1.0$] of all smokers). Prevalence of smoking was highest among persons aged 18-24 years (27.9\%) and aged 25-44 years (27.5\%), and lowest among persons aged $\geq 65$ years (10.9\%). Prevalence of current smoking was highest among American Indians/Alaska Natives (40.0\%), intermediate among non-Hispanic whites (25.0\%) and non-Hispanic blacks (24.7\%), and lowest among Hispanics (19.1\%) and Asians/Pacific Islanders (13.7\%). Adults with $\geq 16$ years of education had the lowest smoking prevalence (11.3\%), achieving the 2010 goal of reducing smoking rates to no more than 12%. Current smoking prevalence was highest among persons with 9-11 years of education (36.8%). Smoking prevalence was higher among persons living below the poverty level (32.3\%) than among those living at or above the poverty level (23.5\%).

In 1998, an estimated 44.8 million adults (22.9\% [$95\%$ CI = $\pm 0.6$]) were former smokers, comprising 25.7 million men and 19.1 million women. Former smokers constituted 48.7\% (95\% CI = $\pm 1.0$) of persons who had ever smoked $\geq 100$ cigarettes. Among current daily smokers in 1998, an estimated 15.2 million (39.2\% [$95\%$ CI = $\pm 1.4$]) had stopped smoking for at least 1 day during the preceding 12 months because they were trying to stop smoking.

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**CDC Editorial Note:** The findings in this report suggest that the goal of reducing the prevalence of cigarette smoking among adults to $\leq 12\%$ by 2010 will require aggressive public health efforts to implement comprehensive tobacco-control programs nationwide. The 1998 NHIS data also demonstrate substantial differences in smoking prevalence across populations.

In 1998, smoking prevalence among persons aged 18-24 years was as high as the prevalence among persons aged 5 years, regardless of vaccination history, with an interval of 4-6 weeks between doses.

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25-44 years. Historically, smoking prevalence has been highest among persons aged 25-44 years and significantly lower among persons aged 18-24 years. Recent increases among persons aged 18-24 years may reflect the aging of the cohort of high school students among whom current smoking rates were high during the 1990s. In addition, the increase may indicate increased initiation of smoking among young adults. The high prevalence of smoking among young adults indicates a need to focus tobacco-use prevention and treatment programs on both adolescents and young adults.

Smoking prevalence reported for racial/ethnic subgroups showed few changes from 1997 to 1998. Prevalence of current smoking among American Indians/Alaska Natives remained the highest. State and regional surveys indicate that the prevalence of smoking cessation among American Indians/Alaska Natives remains relatively low. Although many factors contribute to the high prevalence of smoking among American Indians/Alaska Natives, it is important to develop culturally appropriate prevention and control measures that distinguish between the use of manufactured tobacco products and the ceremonial use of tobacco.

National health objectives for 2010 that are focused on eliminating population disparities reinforce the need for greater surveillance and culturally responsive approaches to tobacco use across communities. In the United States, population disparities in smoking prevalence have been consistent from 1993 through 1998. For example, in 1993, an 8.3 (95% CI = ±2.5) percentage-point difference in smoking prevalence existed between those at or above the poverty level and those below (23.8% and 32.1%, respectively). In 1998, the difference was 8.8 (95% CI = ±1.9) percentage points (23.5% and 32.3%, respectively). Similarly, differences in prevalence among various educational groups have not been reduced. In 1993, the difference between those with 9-11 years of education and those with ≥16 years was 23.3 (95% CI = ±3.0) percentage points (36.8% and 13.5%, respectively). In 1998, the difference was 25.5 (95% CI = ±2.3) percentage points (36.8% and 11.3%, respectively). The relation between tobacco use and increased risk for failing or dropping out of high school demonstrates the necessity of reaching these students through school-based programs before they leave school. Differences in prevalence among racial/ethnic subgroups have not been reduced. For example, in 1993, the difference between non-Hispanic whites and American Indians/Alaska Natives was 13.3 (95% CI = ±8.7) percentage points (25.4% and 38.7%, respectively). In 1998, the difference between non-Hispanic whites and American Indians/Alaska Natives was 15.0 (95% CI = ±9.8) percentage points (25.0% and 40.0%, respectively). The reduction of tobacco-related health disparities requires communities, states, and national organizations to take a multidisciplinary approach to tobacco prevention and control.

The findings in this report are subject to at least two limitations. Because the questionnaire for the 1997 NHIS was redesigned completely, trend analysis or comparison with data from years before 1997 should be conducted with caution. Second, the sample size of certain subgroups (e.g., American Indians/Alaska Natives) was small, possibly resulting in unstable estimates.

Although comprehensive programs are critical in reducing the burden of tobacco use, short-term decreases in tobacco-related morbidity and mortality can be achieved only by helping current smokers quit. To assist in this process, the U.S. Department of Health and Human Services has released guidelines with specific evidence-based recommendations for tobacco-use treatment. Recommended interventions include individual, group, or telephone counseling that offers practical advice about and support for quitting; support from family and friends also improves success rates. In addition, all smokers trying to quit should be encouraged to use a medication approved by the Food and Drug Administration, either nicotine replacement therapy (gum, inhaler, nasal spray, or patch) or a non-nicotine pharmacologic aid (bupropion). To ensure that smokers interested in quitting receive appropriate treatment, health-care systems must make routine screening of tobacco use the standard of care and monitor (through quality assurance processes) the provision of appropriate interventions to smokers. Improving access to treatment by reducing cost barriers also increases the number of quitters.

A comprehensive approach to tobacco control will require treatment for nicotine dependence and efforts at national, state, and local levels to reduce youth smoking, promote smoke-free environments, support countermarketing efforts, enforce laws and regulations, and eliminate disparities in tobacco use among population subgroups. Increased attention must be focused on groups that show no decline in smoking prevalence, including persons aged 18-24 years, adults with low education levels, and American Indians/Alaska Natives. Approaches with the widest scope (i.e., economic, regulatory, and comprehensive) are likely to have the greatest long-term population impact.

REFERENCES

Foodborne Botulism From Eating Home-Pickled Eggs—Illinois, 1997

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DURING NOVEMBER 1997, THE ILLINOIS Department of Public Health was notified by a local physician about a pos-
Possible case of foodborne botulism. This report summarizes the case investigation, which implicated home-pickled eggs as the cause.

On November 23, 1997, a previously healthy 68-year-old man became nauseated, vomited, and complained of abdominal pain. During the next 2 days, he developed diplopia, dysarthria, and respiratory impairment, necessitating hospitalization and mechanical ventilation. Physical examination confirmed multiple cranial nerve abnormalities, including extraocular motor palsy and diffuse flaccid paralysis. Possible botulism was diagnosed, and a one-vial dose of trivalent (types A, B, and E) antitetanus toxoid was administered. A sample of the patient’s serum collected before antitetanus administration demonstrated the presence of type B botulinum toxin.

A food history revealed no exposures to home-canned products; however, the patient had eaten pickled eggs that he had prepared 7 days before onset of illness; gastrointestinal symptoms began 12 hours after ingestion. The patient recovered after prolonged supportive care.

The pickled eggs were prepared using a recipe that consisted of hard-boiled eggs, commercially prepared beets and hot peppers, and vinegar. The intact hard-boiled eggs were peeled and punctured with toothpicks then combined with the other ingredients in a glass jar that closed with a metal screw-on lid. The mixture was stored at room temperature and occasionally was exposed to sunlight.

Cultures revealed *Clostridium botulinum* type B, and type B toxin was detected in samples of the pickled egg mixture at CDC’s National Botulism Surveillance and Reference Laboratory. *C. botulinum* was cultured from the pickling liquid, beets, and egg yolk. The concentration of preformed type B toxin was 1000 times greater in the egg yolks than in the pickling liquid and was undetected in the beets. Peppers from the original commercial container contained no detectable toxin, and bacterial cultures of the peppers did not yield *C. botulinum*. Beets from the original commercial containers were not available. The pH of the pickling liquid was 3.5 (i.e., adequate to prevent *C. botulinum* germination and toxin formation. However, the pH of the egg yolk was not determined [normal egg yolk pH: 6.8]).

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**CDC Editorial Note:** Botulism is a paralytic illness caused by the neurotoxin produced by the bacterium *C. botulinum*. Paralysis first affects the cranial nerves, then the skeletal muscles; untreated intoxications can lead to dense flaccid paralysis, respiratory failure, and death.1,2

Although rare and sporadic, foodborne botulism is a persistent cause of morbidity and mortality in the United States. In 1997, an annual survey of state epidemiologists and directors of state public health laboratories identified 24 cases of foodborne botulism with one associated death (CDC, unpublished data, 1998). During 1989-1998, a median of 23 cases (range: 17-42 cases) of foodborne botulism was reported each year with a median of one death (range: 0-2 deaths).

*C. botulinum* spores are ubiquitous. Safe food preservation methods destroy spores or inhibit their germination and growth. Conditions that promote germination and growth of *C. botulinum* spores include absence of oxygen (anaerobic conditions), low acidity (pH greater than 4.6), temperatures greater than 39°F (4°C), and high moisture content. Most foodborne botulism cases that occur in the United States are the result of improperly home-canned foods. This is the first reported case of botulism related to eating pickled eggs. The amount of toxin detected in the recovered egg yolk suggested that bacterial growth was concentrated in that portion of the egg. Intact eggs that have been hard-boiled should be free of bacteria or spores. Pricking cooked eggs may introduce *C. botulinum* spores into the yolk. Portions of the yolk that remained anaerobic and inadequately pickled (i.e., not acidified to pH less than or equal to 4.6) may have allowed *C. botulinum* spores to germinate, grow, and form toxin. Setting the pickling jar in sunlight provided warmth that facilitated bacterial growth and toxin production.

To reduce the risk for botulism when pickling, food items should be washed and cooked adequately, and utensils, containers, and other surfaces in contact with food, including cutting boards and hands, should be cleaned thoroughly with soap and warm water. Containers (e.g., jars and lids) in which pickling will occur should be sterilized (e.g., placed in boiling water for the prescribed period published in the container instructions). ADEquate acidification to a pH less than or equal to 4.6 is essential. Refrigeration at 39°F (4°C) during pickling is advisable, especially in foods that may be acidified inadequately such as whole eggs. Once opened, any canned or pickled food should be refrigerated. Pricking, poking holes, or otherwise handling whole eggs in a manner that might allow spores or bacteria into the yolk should be avoided.

When foodborne botulism is suspected, clinicians and public health investigators should inquire about the preparation and eating of foods preserved by any home method (e.g., canning, pickling, curing, and fermenting). Persons seeking advice on home-food preservation should consult their local county or university cooperative extension service, or contact the U.S. Department of Agriculture Food Safety Hotline, telephone (800) 535-4555. CDC provides epidemiologic consultation and laboratory diagnostic services for suspected botulism cases and authorizes release of botulinum antitoxin. Through state health departments, these services are available 24 hours a day from CDC.

**REFERENCES**

3 available