

International Overdose Awareness Day — August 31, 2018

August 31, 2018, is International Overdose Awareness Day, a global event to raise awareness that death from drug overdose is preventable. Goals include increasing awareness about the risk for overdose, reducing stigma associated with drug overdose deaths, providing information about community services, and preventing and reducing drug-related harm by supporting evidence-based policy and practice (<https://www.overdoseday.com>).

The opioid overdose epidemic, which killed over 42,000 Americans in 2016 (1), has included three interrelated waves since 1999 (2). The first included increases in overdose deaths related to prescription opioids. In 2010, the second wave began and was characterized by a rapid increase in deaths involving heroin. The third and current wave, which began in 2013, is associated with a rapid increase in deaths involving synthetic opioids, such as illicitly manufactured fentanyl and fentanyl analogs. Varying circumstances have been associated with each of these waves, and their intersection presents a unique challenge for a focused and comprehensive response.

Opioid deaths, particularly those involving illicit opioids, continue to increase. As described in a report in this issue of *MMWR*, illicit opioids were detected in approximately three of four opioid overdose deaths compared with nearly four of 10 for prescription opioids in 11 states examined. Enhanced surveillance for opioid overdose deaths facilitates the classification of deaths involving prescription and illicit opioids as well as identifying missed opportunities for prevention and response. Further information on CDC's state efforts and opioid overdose data is available at <https://www.cdc.gov/drugoverdose/index.html>.

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2. CDC. Understanding the epidemic. Atlanta, GA: US Department of Health and Human Services; CDC, 2017. <https://www.cdc.gov/drugoverdose/epidemic/index.html>

Opportunities to Prevent Overdose Deaths Involving Prescription and Illicit Opioids, 11 States, July 2016–June 2017

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In 2016, 63,632 drug overdose deaths occurred in the United States, 42,249 (66.4%) of which involved opioids (1). The development of prevention programs are hampered by a lack of timely data on specific substances contributing to and circumstances associated with fatal overdoses. This report describes opioid overdose deaths (referred to as opioid deaths) for decedents testing positive for prescription opioids (e.g., oxycodone and hydrocodone), illicit opioids (e.g., heroin, illicitly manufactured fentanyl, and fentanyl analogs), or both prescription and illicit opioids, and describes circumstances surrounding the overdoses, in 11 states participating in CDC's Enhanced State Opioid

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Overdose Surveillance (ESOOS) program.* During July 2016–June 2017, among 11,884 opioid overdose deaths, 17.4% of decedents tested positive for prescription opioids only, 58.7% for illicit opioids only, and 18.5% for both prescription and illicit opioids (type of opioid could not be classified in 649 [5.5%] deaths). Approximately one in 10 decedents had been released from an institutional setting in the month preceding the fatal overdose. Bystanders were reportedly present in approximately 40% of deaths; however, naloxone was rarely administered by a layperson. Enhanced surveillance data from 11 states provided more complete information on the substances involved in and circumstances surrounding opioid overdose deaths. Consistent with other emerging evidence and recommendations,[†] these data suggest prevention efforts should prioritize naloxone distribution to persons misusing opioids or using high dosage prescription opioids and to their family members and friends. In addition, these data suggest a need to expand treatment and support for persons who have experienced

a nonfatal overdose and to expand treatment in detention facilities and upon release.

CDC funds 32 states and the District of Columbia to abstract death certificate and medical examiner and coroner data, including toxicology results, on opioid deaths, through the State Unintentional Drug Overdose Reporting System, a component of ESOOS. Data are collected on death scene investigations (e.g., evidence of illicit or prescription drug use), circumstances occurring close in time to the death (e.g., presence of bystanders), history of substance use treatment, prior history of overdose, and demographics. For all opioid deaths classified as unintentional or of undetermined intent, states list all positive tests for opioid and nonopioid substances (“present” or “detected”), and whether the medical examiner or coroner determined that the substance contributed to the overdose death (“involved”).[§] CDC

*CDC’s Enhanced State Opioid Overdose Surveillance program funded 12 states through a competitive application process in fiscal year 2016, and an additional 20 states and the District of Columbia in fiscal year 2017. Data were available for Kentucky, Maine, Massachusetts, Missouri, New Hampshire, New Mexico, Ohio, Oklahoma, Rhode Island, West Virginia, and Wisconsin for this report. <https://www.cdc.gov/drugoverdose/foa/state-opioid-mm.html>.

[†]<https://www.surgeongeneral.gov/priorities/opioid-overdose-prevention/naloxone-advisory.html>.

[§]State Unintentional Drug Overdose Reporting System (SUDORS) estimates of opioid-involved overdose deaths might differ from those of the National Vital Statistics System because SUDORS uses preliminary death certificate data and collects additional information from medical examiner and coroner reports, which are abstracted within 8 months of death. In SUDORS, an opioid-involved overdose death either was identified through review of the medical examiner/coroner report or had *International Classification of Diseases, Tenth Revision* (ICD-10) underlying cause-of-death codes X40–44 (unintentional) or Y10–Y14 (undetermined) and multiple cause-of-death codes of T40.0, T40.1, T40.2, T40.3, T40.4, or T40.6 on the death certificate. Data for this report were downloaded on June 30, 2018, and might differ from reports using earlier data.

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analyzed demographics, routes of administration, co-use of other substances, and overdose circumstances by involvement of prescription opioids only,[§] illicit opioids only,^{**} or the presence of both prescription and illicit opioids,^{††}

for deaths that occurred during July 2016–June 2017 in 11 ESOOS states.^{§§}

Among 11,884 opioid deaths, 2,066 (17.4%) involved prescription opioids only, 6,975 (58.7%) involved illicit opioids only, and for 2,194 (18.5%) both prescription and illicit opioids were detected; type of opioid could not be classified in 649 (5.5%) deaths, leaving 11,235 deaths for analysis. Among deaths for which both prescription and illicit opioids were detected, medical examiners or coroners determined that both prescription and illicit opioids contributed to 59.2% of deaths, illicit opioids alone contributed to 39.8% of deaths, and prescription opioids alone contributed to 1.0% of deaths. The percentage of deaths involving different opioid types varied across states (Figure), with the highest percentages

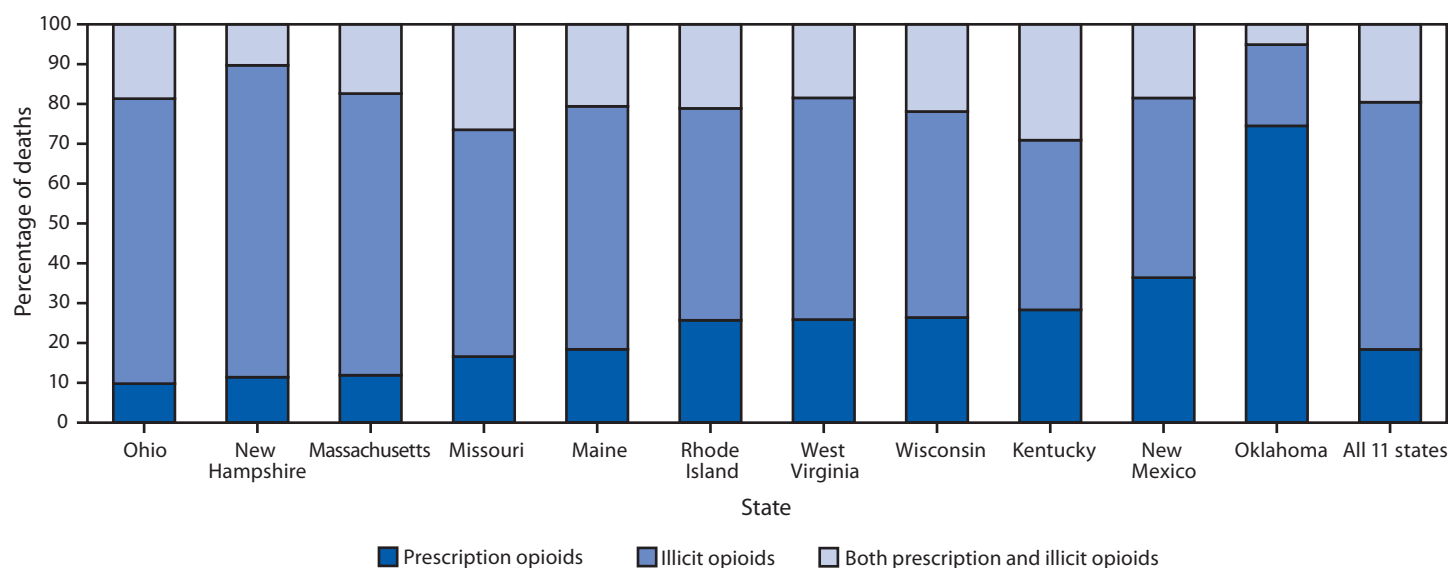
[§] Substances coded as prescription opioids were oxycodone, oxymorphone, hydrocodone, hydromorphone, tramadol, buprenorphine, methadone, meperidine, tapentadol, dextropropofol, levorphanol, propoxyphene, nospapine, pentazocine, and phenacetin. Brand names (e.g., Opana), metabolites (e.g., nortramadol) of these substances, and these substances in combination with nonopioids (e.g., acetaminophen-oxycodone) were included as prescription opioids as well. Morphine and codeine were coded as prescription opioids if scene or other evidence indicated their presence as a result of consumption of prescription morphine or codeine, rather than as a result of metabolism of or impurities of heroin, respectively. Fentanyl was coded as a prescription opioid if scene or other evidence indicated likely consumption of prescription fentanyl rather than illicitly manufactured fentanyl. Decedents might have tested positive for other nonopioid substances. This analysis does not distinguish between prescription drugs prescribed to the decedent and those that were diverted.

^{**} Substances coded as illicit opioids were heroin, fentanyl analogs, and U-47700. Fentanyl was coded as illicit if scene or other evidence indicated that it was more likely to have been illicitly manufactured than pharmaceutical. Decedents might have tested positive for other nonopioid substances.

^{††} Deaths were coded as positive for both prescription and illicit opioids if one or more opioids from both categories were detected on postmortem toxicology testing. Decedents might have tested positive for other nonopioid substances.

^{§§} Twelve states were funded through a competitive application process in September 2016 (Kentucky, Maine, Massachusetts, Missouri, New Hampshire, New Mexico, Ohio, Oklahoma, Pennsylvania, Rhode Island, West Virginia, and Wisconsin). An additional 20 states (Alaska, California, Connecticut, Delaware, Florida, Georgia, Illinois, Indiana, Louisiana, Maryland, Michigan, Minnesota, Nevada, New Jersey, North Carolina, Tennessee, Utah, Vermont, Virginia, and Washington) and the District of Columbia were funded in September 2017. Data were available for 11 of the 33 jurisdictions for this report.

FIGURE. Percentage of opioid overdose deaths in which prescription opioids only,* illicit opioids only,[†] or both prescription and illicit opioids[§] were detected, by state — 11 states, July 1, 2016–June 30, 2017



* Oxycodone, oxymorphone, hydrocodone, hydromorphone, tramadol, buprenorphine, methadone, meperidine, tapentadol, dextropropofol, levorphanol, propoxyphene, nospapine, pentazocine, and phenacetin. Brand names (e.g., Opana), metabolites (e.g., nortramadol) of these substances, and these substances in combination with nonopioids (e.g., acetaminophen-oxycodone) were included as prescription opioids. Morphine and codeine were coded as prescription opioids if scene or other evidence indicated their presence as a result of consumption of prescription morphine or codeine, rather than as a result of metabolism of or impurities of heroin, respectively. Fentanyl was coded as a prescription opioid if scene or other evidence indicated likely consumption of prescription fentanyl rather than illicitly manufactured fentanyl. Decedents might have tested positive for other nonopioid substances. This analysis does not distinguish whether prescription drugs were prescribed to the decedent or diverted.

[†] Heroin, fentanyl analogs, and U-47700. Fentanyl was coded as illicit if scene or other evidence indicated that it was more likely illicitly manufactured than pharmaceutical. Decedents might have tested positive for other nonopioid substances.

[§] Deaths were coded as positive for both prescription and illicit opioids if one or more opioids from both categories were detected on postmortem toxicology testing. Decedents might have tested positive for other nonopioid substances.

of prescription opioid–only deaths in the West (Oklahoma: 72.2%; New Mexico: 35.0%), and the highest percentages of illicit opioid–only deaths, ranging from 47.6% to 72.1%, in the Northeast (Maine, Massachusetts, New Hampshire, and Rhode Island) and the Midwest (Missouri, Ohio, West Virginia, and Wisconsin). Kentucky had the highest percentage of deaths with both prescription and illicit opioids (26.5%) present, followed by Missouri (25.1%).

Among prescription opioid–only deaths, the median age of decedents was 47 years, 51.0% were female, and 86.2% were non-Hispanic white (white). Among illicit opioid–only deaths, the median age of decedents was 36 years, 73.0% were male, and 81.1% were white. Among deaths for which both prescription and illicit opioids were detected, decedents' median age was 39 years, 70.5% were male, and 83.6% were white (Table).

Evidence of injection drug use was found in approximately half of illicit opioid deaths, but only 6.6% of prescription opioid–only deaths. Other drugs were frequently detected in opioid deaths (Table). Benzodiazepines and gabapentin were detected in 51.6% and 21.6% of prescription opioid–only deaths, respectively. Among illicit opioid–only deaths, cocaine was detected in 34.9% of deaths and benzodiazepines were detected in 24.0% of deaths. Among deaths for which both prescription and illicit opioids were detected, benzodiazepines were detected in 44.5% and cocaine in 34.8%.

Approximately one in 10 decedents had evidence of having been released from an institutional setting in the month preceding the fatal overdose, with the most common settings being jail, prison, or detention facilities when only illicit opioids were involved (4.9%), and hospitals when only prescription opioids were involved (4.1%). Previous drug overdose at any time before the fatal overdose was noted in 15.1% of illicit opioid–only deaths, 13.5% of deaths with both prescription and illicit opioids present, and 9.3% of prescription opioid–only deaths. Bystanders were reported to have been present in 44% of opioid deaths, but naloxone was seldom administered by a layperson (in approximately 4% of deaths involving only illicit opioids and 0.8% of prescription opioid–only deaths).

Discussion

This report is one of the first to use medical examiner and coroner reports across multiple states and provides information that can be used to better inform prevention and response programs related to opioid overdose deaths. Specifically, among these 11 states, there is improved understanding of prescription and illicit opioid involvement, polysubstance use, and potential missed opportunities to intervene to prevent opioid overdose deaths. Previous efforts to differentiate illicit and

prescription opioid deaths were limited by grouping within the same drug categories (e.g., synthetic opioids, excluding methadone) and by the difficulty in determining whether detection of morphine or fentanyl by forensic toxicology testing indicates the presence of prescription or illicit opioids (2,3).^{¶¶} Findings from this analysis indicate that illicit opioids were a major driver of opioid deaths, especially among younger persons, and were detected in approximately three of four deaths overall. Prescription opioids were detected in approximately four of 10 deaths. Illicit opioids predominated in all states except Oklahoma.

Among these 11 states, the evolving opioid overdose epidemic differentially affects states and regions, but most states were simultaneously struggling with a complex mix of prescription and illicit opioid deaths. In this analysis, four polysubstance use patterns highlight an urgent need for targeted and comprehensive action. First, approximately half of prescription opioid–only deaths tested positive for benzodiazepines, which are known to depress the central nervous system and increase the risk of overdose and death.^{***} This high-risk drug-use pattern can be targeted for intervention. Second, gabapentin (a nonopioid medication commonly prescribed for neuropathic pain), was found in approximately one in five prescription opioid–only deaths and in approximately one in 10 deaths in the other groups. Consistent with recent reports (4), the combined use of gabapentin and opioids might be an indicator of high-risk opioid misuse and requires further study. In the illicit opioid–only group, the percentage of deaths testing positive for cocaine and methamphetamine is similar to other reports (5). Finally, extensive use of cocaine and benzodiazepines among deaths where both prescription and illicit opioids were detected highlights the need for prevention and treatment programs to address polysubstance use (6).

Identification of circumstances surrounding overdose deaths can help inform prevention programs and efforts to target resources. Approximately one in 10 decedents had been released from an institution in the month before the fatal overdose. Rhode Island found that expanding enrollment in a medication-assisted treatment program for incarcerated persons was associated with a 60% decrease in postincarceration overdose deaths (7). For the 14% of decedents with previous overdoses, there might have been opportunities for linkage to care and treatment services, especially if the overdose involved an emergency department

^{¶¶} <http://ajph.aphapublications.org/doi/10.2105/AJPH.2017.304265>.

^{***} The Food and Drug Administration issued its strongest warning against combining benzodiazepines with opioids because of risk for overdose. <https://www.fda.gov/Drugs/DrugSafety/ucm518473.htm>.

TABLE. Demographic characteristics of persons who died from opioid overdose and overdose circumstance factors by substance type — 11 states, July 1, 2016–June 30, 2017

Characteristic	Type of opioid(s) detected		
	Prescription opioids only N = 2,066 (17.4%)	Illicit opioids only N = 6,975 (58.7%)	Prescription and illicit opioids N = 2,194 (18.5%)
	No. (%)	No. (%)	No. (%)
Type of opioid(s) contributing to death*			
Prescription opioids only	2,066 (100.0)	—†	22 (1.0)
Illicit opioids only	—†	6,975 (100.0)	873 (39.8)
Both illicit and prescription opioids	—†	—†	1,299 (59.2)
Age group (yrs)[§]			
15–24	79 (3.8)	714 (10.2)	130 (5.9)
25–34	307 (14.9)	2,346 (33.6)	608 (27.7)
35–44	500 (24.2)	1,825 (26.2)	635 (28.9)
45–54	621 (30.1)	1,298 (18.6)	485 (22.1)
55–64	456 (22.1)	708 (10.2)	291 (13.3)
≥65	103 (5.0)	84 (1.2)	45 (2.1)
Median age (interquartile range) in years [¶]	47 (37–55)	36 (29–47)	39 (32–50)
Sex[§]			
Male	1,013 (49.0)	5,089 (73.0)	1,546 (70.5)
Female	1,053 (51.0)	1,886 (27.0)	648 (29.5)
Race and Hispanic origin[§]			
White, non-Hispanic	1,780 (86.2)	5,657 (81.1)	1,833 (83.6)
Black, non-Hispanic	100 (4.8)	685 (9.8)	206 (9.4)
Other, non-Hispanic	51 (2.5)	88 (1.3)	16 (0.7)
Hispanic	100 (4.8)	421 (6.0)	112 (5.1)
Route of administration			
Evidence of injection [§]	136 (6.6)	3,428 (49.2)	958 (43.7)
No evidence of injection; evidence of other route [§]	783 (37.9)	1,194 (17.1)	382 (17.4)
Evidence of snorting [§]	85 (10.9)	743 (62.2)	210 (55.0)
Evidence of ingestion [§]	669 (85.4)	416 (34.8)	211 (55.2)
Evidence of smoking [§]	34 (4.3)	251 (21.0)	54 (14.1)
Evidence of transdermal	65 (8.3)	—†	—†
No evidence of route [§]	1,147 (55.5)	2,353 (33.7)	854 (38.9)
Other substance(s) detected			
Cocaine ^{§,**}	207 (10.0)	2,432 (34.9)	763 (34.8)
Methamphetamines [§]	155 (7.5)	737 (10.6)	277 (12.6)
Benzodiazepines [§]	1,065 (51.6)	1,676 (24.0)	976 (44.5)
Gabapentin [§]	447 (21.6)	564 (8.1)	326 (14.9)
Released from an institution 1 month before death			
Released from any institutional setting [§]	140 (6.8)	726 (10.4)	200 (9.1)
Released from jail, prison, or detention facility [§]	22 (1.1)	343 (4.9)	67 (3.1)
Released from residential alcohol or substance use treatment facility [§]	22 (1.1)	216 (3.2)	53 (2.5)
Released from a hospital [§]	81 (4.1)	107 (1.6)	54 (2.6)
Previous drug overdose[§]	193 (9.3)	1,053 (15.1)	297 (13.5)
Died when bystander was present	860 (41.6)	3,072 (44.0)	987 (45.0)
Naloxone administered by layperson^{§,††}	8 (0.8)	169 (4.3)	52 (4.4)

* By definition, for the categories of “only prescription opioids” and “only illicit opioids” detected, the contributing opioid type could only be “prescription opioids only” or “illicit opioids only,” respectively. For the category of “both prescription and illicit opioids,” either prescription opioids only, illicit opioids only, or both could have contributed to death. Decedents might have tested positive for other nonopioid substances. This analysis does not distinguish between prescription drugs prescribed to the decedent and those that were diverted.

† Data suppressed because of <5 deaths, or suppressed to prohibit calculation of other suppressed cell.

§ Indicates overall chi-squared test statistic has a p-value <0.001.

¶ Statistically significant differences in mean age (p-value <0.001) were identified for prescription opioid deaths (46.3 years), illicit opioid deaths (38.4 years), and prescription and illicit (40.9 years).

** Indicates the presence of cocaine or a cocaine metabolite.

†† Among deaths for which naloxone administration status was known: 1,032 for prescription opioids only, 3,927 for illicit opioids only, and 1,173 for prescription and illicit opioids.

visit (8). The proportions of decedents with evidence of recent release from an institution and of a previous overdose were higher among deaths involving illicit opioids. Similar to earlier findings (9), approximately half of the decedents overdosed when bystanders were present. Although distribution of naloxone to laypersons has rapidly expanded and been determined to be effective,^{†††} broader distribution and education about overdose signs and symptoms are needed.

The findings in this report are subject to at least five limitations. First, because there is no national standard for forensic toxicology testing, testing protocols vary across jurisdictions, which affects whether substances were detected. Second, jurisdictions vary in how they classify whether substances with positive toxicology results contribute to death. Third, evidence of overdose-specific circumstances should be interpreted with caution because it relies upon availability of information within medical examiner and coroner reports and focuses on information from a period close to death; thus, prevalence of circumstances is likely underestimated. Fourth, missing information might have resulted in some misclassification of prescription and illicit substance use; however, this was minimized by using detailed toxicology results and scene evidence. Finally, the results are limited to the 11 participating states and cannot be generalized to the United States.

Among 11 states, illicit opioids were a major driver of opioid overdose deaths; however, prescription opioids also contribute to a substantial number of these deaths. Interventions should be guided by the substances detected and contributing to overdose deaths in a given locale and might differ for overdoses involving prescription or illicit opioids, or both. For example, for preventing illicit opioid overdose, integrating public health strategies within public safety (e.g., law enforcement providing linkages to care for persons with substance use disorders), as well as using syringe services programs for naloxone distribution, providing access to treatment, and addressing blood borne infections might have a larger impact. To prevent prescription opioid overdose, strategies might emphasize prescription drug monitoring programs, face-to-face education of prescribers by trained health care professionals, typically pharmacists, physicians, or nurses (a process known as academic detailing), and implementation of the CDC Guideline for Prescribing Opioids for Chronic Pain (10). However, interventions should not focus exclusively on one opioid type because the epidemic continues to evolve, and use of opioids along with other substances is common. Continued and increased attention should capitalize on opportunities for overdose prevention including linking to treatment during and upon release from an institution or after a nonfatal overdose and expanding naloxone access.

^{†††} <https://www.bmj.com/content/346/bmj.f174>.

Summary

What is already known about this topic?

In 2016, opioids were involved in 42,249 U.S. overdose deaths.

What is added by this report?

Among 11 reporting states, most (58.7%) opioid overdose deaths involved illicit opioids only, followed by those where both illicit and prescription opioids were detected (18.5%); 17.4% of deaths involved prescription opioids only. Bystanders to the overdose, who could potentially intervene, were documented in 44% of deaths; however, laypersons rarely administered naloxone.

What are the implications for public health practice?

Development of overdose prevention programs should consider the types of opioids contributing to deaths, link persons to treatment during and upon release from an institution or after a nonfatal overdose, and expand naloxone distribution to laypersons.

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Racial/Ethnic Disparities in Tobacco Product Use Among Middle and High School Students — United States, 2014–2017

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During the past few decades, wide disparities in tobacco product use have been documented among the largest racial/ethnic groups in the United States (1,2); however, little is known about tobacco product use among youths from racial/ethnic groups other than whites, blacks, and Hispanics. Surveillance reports typically aggregate these racial/ethnic minorities into a single category because of small sample sizes (3). To assess tobacco product use among U.S. middle and high school students from seven racial/ethnic groups (non-Hispanic whites [whites], non-Hispanic blacks [blacks], Hispanics, non-Hispanic Asians [Asians], non-Hispanic American Indian/Alaska natives [AI/ANs], non-Hispanic Native Hawaiians/Other Pacific Islanders [NHOPIs], and non-Hispanic multiracial persons [multiracial]), CDC analyzed pooled data from the 2014–2017 National Youth Tobacco Surveys (NYTS). Prevalence of ever (≥1 time in lifetime) and current (≥1 time in past 30 days) use of seven tobacco products (cigarettes, cigars, smokeless tobacco, electronic cigarettes [e-cigarettes], hookahs, pipes, and bidis) was assessed; any tobacco product use was defined as use of one or more tobacco products, including hand-rolled cigarettes. During 2014–2017, ever-use of any tobacco product among U.S. middle and high school students was as follows: NHOPIs (45.1%), AI/ANs (43.8%), multiracial persons (38.2%), Hispanics (35.1%), blacks (32.3%), whites (32.0%), and Asians (16.3%). Current use of any tobacco product was as follows: NHOPIs (23.4%), AI/ANs (20.6%), multiracial persons (16.5%), whites (15.3%), Hispanics (14.6%), blacks (11.5%), and Asians (5.0%). Among black middle and high school students, cigars were the most common product currently used, whereas e-cigarettes were the most commonly used product for all other racial/ethnic groups. Comprehensive and sustained implementation of evidence-based, population-level tobacco control interventions could reduce prevalence and disparities in tobacco product use among U.S. youths.

NYTS is a cross-sectional, voluntary, school-based, paper-and-pencil questionnaire administered to U.S. middle (grades 6–8) and high (grades 9–12) school students. A three-stage cluster sampling procedure was used to generate a nationally representative sample of U.S. students attending public and private schools.* For these analyses, data were pooled across four cycles of NYTS (2014–2017) to allow sufficient sample size to assess tobacco product use among each of the racial/ethnic groups. Averaged annual sample size and response rate were 19,566 and 69.1%, respectively.†

* https://www.cdc.gov/tobacco/data_statistics/surveys/nyts/index.htm.

† Annual sample sizes and response rates were 22,007: 73.3% (2014); 17,711: 63.4% (2015); 20,675: 71.6% (2016); and 17,872: 68.1% (2017).

Participants were asked about ever[§] and current[¶] use of seven tobacco product types: cigarettes, cigars (cigars, cigarillos, and little cigars), smokeless tobacco products (chewing tobacco, snuff, dip, snus, and dissolvable tobacco), e-cigarettes, hookahs, pipes, and bidis. Use of hand-rolled cigarettes was also assessed in the survey, but is not reported separately as a distinct tobacco product type because the majority (88.7%) of persons who reported smoking hand-rolled cigarettes also smoked regular cigarettes. Any tobacco product use was defined as use of ≥1 tobacco product types, including hand-rolled cigarettes.

Data were weighted to adjust for differential nonresponse and to yield nationally representative estimates. Pooled sample sizes for each racial/ethnic group during 2014–2017 included 32,358 whites, 11,664 blacks, 21,337 Hispanics, 3,321 Asians, 1,213 AI/ANs, 456 NHOPIs, and 4,106 multiracial persons. Prevalence was calculated overall and by race/ethnicity; within each racial/ethnic group, prevalence was further stratified by sex and school level (middle or high school). Comparisons between and within each racial/ethnic group were performed with chi-squared tests, with statistical significance defined as $p < 0.05$. Estimates with relative standard errors ≥30% were suppressed. Differences in any tobacco product use across groups were assessed using Poisson regression models adjusted for sex and school level. Adjusted prevalence ratios (aPRs) with 95% confidence intervals (CIs) were calculated, with the group with the lowest prevalence (Asians) serving as the referent group.

Ever-Use of Tobacco Products

During 2014–2017, ever-use of any tobacco product among U.S. middle and high school students was highest among NHOPIs (45.1%) and AI/ANs (43.8%), and did not significantly differ between these groups. Compared with these two groups, prevalence of ever-use of any tobacco product was significantly lower among multiracial persons (38.2%), Hispanics (35.1%), blacks (32.3%), whites (32.0%), and Asians (16.3%) (Table 1).

[§] Respondents were asked whether they have ever used/smoked the respective tobacco products, or asked “Which of the following tobacco products have you ever tried, even just one time?,” depending on the type of products and survey years. Those who provided an affirmative response or specified their product(s) from the product list provided were classified as ever tobacco product users.

[¶] Respondents were asked on how many days in the past 30 days they used/smoked the respective tobacco products, or asked “In the past 30 days, which of the following products have you used on at least one day?,” depending on the type of products and survey years. Those who answered ≥1 day or specified their product(s) from the product list provided were classified as current tobacco product users.

TABLE 1. Prevalence of ever use* of tobacco products among middle and high school students, by race/ethnicity†, sex, and school level — National Youth Tobacco Survey, United States, 2014–2017[§]

Characteristic	Any tobacco [¶]	Electronic cigarettes	Cigarettes	Cigars (cigars/cigarillos/little cigars)	Hookah	Smokeless tobacco**	Pipe tobacco	Bidis
	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)
All MS and HS students	32.5 (32.1–32.9)	22.9 (22.5–23.3)	20.2 (19.9–20.6)	16.1 (15.7–16.4)	11.5 (11.2–11.7)	9.2 (9.0–9.5)	2.7 (2.5–2.8)	1.3 (1.2–1.4)
White								
Overall	32.0 (31.4–32.7)	23.9 (23.3–24.4)	19.9 (19.4–20.5)	15.8 (15.3–16.3)	10.5 (10.1–10.9)	11.6 (11.2–12.1)	3.0 (2.8–3.3)	1.3 (1.1–1.4)
Male	34.6 (33.7–35.5) ^{††}	25.6 (24.8–26.5) ^{††}	20.8 (20.0–21.6) ^{††}	20.0 (19.2–20.7) ^{††}	10.8 (10.2–11.4)	16.8 (16.1–17.5) ^{††}	4.1 (3.8–4.5) ^{††}	1.7 (1.5–2.0) ^{††}
Female	29.5 (28.5–30.4)	22.0 (21.2–22.9)	19.0 (18.2–19.7)	11.5 (10.9–12.1)	10.2 (9.7–10.8)	6.4 (5.9–6.9)	1.9 (1.6–2.1)	0.7 (0.6–0.9)
MS	14.9 (14.1–15.7) ^{††}	10.4 (9.7–11.0) ^{††}	8.5 (7.9–9.2) ^{††}	4.2 (3.8–4.7) ^{††}	2.6 (2.3–2.9) ^{††}	4.8 (4.3–5.2) ^{††}	0.9 (0.7–1.1) ^{††}	0.4 (0.3–0.6) ^{††}
HS	44.7 (43.8–45.6)	33.8 (33.0–34.7)	28.3 (27.5–29.1)	24.4 (23.6–25.2)	16.4 (15.7–17.0)	16.7 (16.0–17.4)	4.6 (4.2–5.0)	1.9 (1.6–2.1)
Black								
Overall	32.3 (31.2–33.5)	16.0 (15.1–16.8)	18.0 (17.1–18.9)	19.9 (18.9–20.8)	9.5 (8.8–10.2)	3.2 (2.8–3.6)	1.0 (0.7–1.2)	1.0 (0.7–1.2)
Male	32.3 (30.7–34.0)	17.6 (16.3–18.9) ^{††}	18.9 (17.5–20.2)	20.5 (19.1–21.8)	8.6 (7.7–9.6) ^{††}	4.4 (3.7–5.1) ^{††}	1.2 (0.8–1.5)	1.0 (0.7–1.4)
Female	32.3 (30.7–33.9)	14.3 (13.1–15.4)	17.1 (15.8–18.4)	19.3 (17.9–20.6)	10.5 (9.5–11.4)	1.8 (1.4–2.3)	0.8 (0.5–1.0)	0.9 (0.6–1.2)
MS	19.0 (17.5–20.5) ^{††}	10.6 (9.5–11.7) ^{††}	10.7 (9.6–11.9) ^{††}	9.0 (7.9–10.0) ^{††}	4.5 (3.7–5.3) ^{††}	2.1 (1.6–2.6) ^{††}	0.8 (0.5–1.0)	0.5 (0.3–0.7) ^{††}
HS	42.3 (40.7–43.9)	19.9 (18.6–21.1)	23.3 (22.0–24.6)	27.9 (26.5–29.3)	13.2 (12.2–14.3)	3.9 (3.2–4.5)	1.1 (0.8–1.5)	1.3 (1.0–1.7)
Hispanic								
Overall	35.1 (34.3–36.0)	26.0 (25.2–26.7)	22.2 (21.5–22.9)	15.7 (15.1–16.4)	15.0 (14.4–15.6)	7.7 (7.2–8.1)	2.7 (2.4–2.9)	1.7 (1.5–1.9)
Male	36.4 (35.2–37.6) ^{††}	28.1 (27.0–29.1) ^{††}	23.4 (22.3–24.4) ^{††}	17.6 (16.7–18.5) ^{††}	14.4 (13.5–15.2)	9.7 (9.0–10.4) ^{††}	3.2 (2.8–3.7) ^{††}	1.8 (1.5–2.1)
Female	33.8 (32.6–35.0)	23.9 (22.8–24.9)	20.9 (19.9–21.9)	13.8 (13.0–14.7)	15.5 (14.6–16.4)	5.6 (5.0–6.1)	2.0 (1.7–2.3)	1.5 (1.2–1.9)
MS	20.8 (19.7–22.0) ^{††}	15.6 (14.7–16.6) ^{††}	12.4 (11.5–13.3) ^{††}	7.9 (7.1–8.6) ^{††}	7.9 (7.1–8.6) ^{††}	4.7 (4.1–5.3) ^{††}	1.6 (1.3–1.9) ^{††}	1.0 (0.8–1.3) ^{††}
HS	46.7 (45.5–47.8)	34.4 (33.3–35.4)	30.1 (29.1–31.1)	22.0 (21.1–22.9)	20.6 (19.7–21.5)	9.8 (9.1–10.5)	3.4 (3.0–3.8)	2.1 (1.8–2.4)
Asian								
Overall	16.3 (14.6–17.9)	11.0 (9.6–12.4)	10.3 (9.0–11.6)	4.7 (3.8–5.5)	5.7 (4.8–6.6)	3.1 (2.3–3.9)	0.8 (0.5–1.2)	0.4 (0.2–0.6)
Male	18.5 (16.1–20.9) ^{††}	12.2 (10.3–14.2)	12.1 (10.2–14.1) ^{††}	5.8 (4.5–7.0) ^{††}	5.4 (4.2–6.6)	3.9 (2.8–5.1) ^{††}	1.0 (0.5–1.5)	— ^{§§}
Female	13.8 (11.7–16.0)	9.6 (7.7–11.5)	8.2 (6.5–9.9)	3.5 (2.4–4.6)	6.0 (4.6–7.5)	2.2 (1.2–3.2)	— ^{§§}	— ^{§§}
MS	5.4 (3.8–6.9) ^{††}	4.6 (3.2–6.1) ^{††}	4.8 (3.2–6.4) ^{††}	2.1 (1.1–3.1) ^{††}	1.6 (0.9–2.4) ^{††}	1.7 (0.8–2.7) ^{††}	— ^{§§}	— ^{§§}
HS	24.4 (21.9–26.9)	15.7 (13.6–17.8)	14.2 (12.3–16.2)	6.6 (5.3–7.8)	8.7 (7.2–10.1)	4.1 (3.0–5.2)	1.2 (0.6–1.7)	— ^{§§}
AI/AN								
Overall	43.8 (39.2–48.4)	29.8 (25.7–33.9)	31.4 (27.2–35.7)	23.1 (19.2–26.9)	14.2 (11.1–17.3)	18.6 (14.6–22.5)	5.3 (3.0–7.6)	2.0 (1.1–3.0)
Male	43.7 (37.7–49.8)	31.4 (26.0–36.7)	32.1 (26.7–37.4)	25.0 (19.9–30.1)	12.7 (9.0–16.3)	23.5 (17.9–29.1) ^{††}	8.5 (4.5–12.6)	— ^{§§}
Female	44.2 (37.0–51.4)	27.9 (21.5–34.4)	31.1 (24.4–37.9)	20.9 (15.0–26.9)	16.3 (11.0–21.6)	12.3 (7.0–17.6)	— ^{§§}	— ^{§§}
MS	28.5 (22.2–34.7) ^{††}	18.6 (13.5–23.7) ^{††}	20.8 (15.3–26.3) ^{††}	12.0 (7.5–16.6) ^{††}	6.6 (3.8–9.3) ^{††}	13.1 (8.2–17.9) ^{††}	— ^{§§}	— ^{§§}
HS	62.8 (56.5–69.2) ^{††}	43.2 (36.9–49.5)	44.4 (38.1–50.7)	36.1 (29.9–42.3)	23.5 (17.8–29.1)	25.4 (19.2–31.7)	7.9 (4.3–11.4)	3.1 (1.3–4.9)
NHOPI								
Overall	45.1 (38.4–51.8)	34.1 (28.0–40.2)	29.4 (23.4–35.4)	22.4 (17.1–27.8)	20.6 (15.5–25.6)	13.1 (8.9–17.4)	4.7 (2.2–7.3)	— ^{§§}
Male	49.7 (40.9–58.5)	37.3 (29.2–45.5)	33.2 (25.1–41.4)	28.0 (20.4–35.6)	21.6 (14.7–28.5)	20.4 (13.3–27.5)	— ^{§§}	— ^{§§}
Female	40.8 (30.7–50.9)	31.0 (21.8–40.2)	25.2 (16.2–34.2)	15.2 (7.6–22.8)	19.1 (11.6–26.6)	— ^{§§}	— ^{§§}	— ^{§§}
MS	23.5 (15.9–31.1) ^{††}	18.7 (12.0–25.4) ^{††}	14.8 (8.6–21.1) ^{††}	10.7 (5.2–16.2) ^{††}	10.5 (4.7–16.2) ^{††}	8.3 (3.5–13.0)	— ^{§§}	— ^{§§}
HS	60.7 (52.4–69.0)	44.9 (36.6–53.2)	39.6 (31.2–48.0)	30.6 (22.8–38.4)	27.5 (20.1–34.8)	16.6 (10.3–22.9)	— ^{§§}	— ^{§§}
Multiracial								
Overall	38.2 (36.1–40.2)	26.6 (24.8–28.5)	24.9 (23.1–26.7)	18.5 (16.9–20.1)	14.3 (12.8–15.8)	10.1 (8.8–11.5)	4.5 (3.4–5.6)	1.6 (1.1–2.1)
Male	38.8 (35.8–41.8)	28.2 (25.5–30.8)	25.2 (22.7–27.7)	21.5 (19.1–23.9) ^{††}	14.3 (12.2–16.4)	14.0 (11.9–16.2) ^{††}	6.1 (4.5–7.7) ^{††}	1.8 (1.1–2.6)
Female	37.6 (34.7–40.4)	25.1 (22.6–27.7)	24.6 (22.0–27.2)	15.6 (13.6–17.7)	14.3 (12.2–16.4)	6.7 (4.9–8.4)	3.2 (1.6–4.7)	1.4 (0.7–2.0)
MS	24.5 (21.6–27.5) ^{††}	15.8 (13.3–18.3) ^{††}	15.2 (12.7–17.7) ^{††}	8.7 (6.9–10.5) ^{††}	5.7 (3.8–7.6) ^{††}	6.8 (4.8–8.8) ^{††}	— ^{§§}	0.9 (0.4–1.4) ^{††}
HS	48.4 (45.7–51.1)	34.6 (32.1–37.2)	32.2 (29.7–34.6)	25.8 (23.5–28.1)	20.7 (18.6–22.9)	12.6 (10.8–14.5)	6.1 (4.6–7.6)	2.1 (1.3–2.9)

Abbreviations: AI/AN = American Indians/Alaska Natives; CI = confidence interval; HS = high school; MS = middle school; NHOPI = Native Hawaiian/Other Pacific Islander.

* Respondents were asked whether they had ever used/smoked the respective tobacco products, or asked “Which of the following tobacco products have you ever tried, even just one time?” depending on the type of products and survey years. Those who provided an affirmative response or specified their product(s) from the product list provided were classified as ever tobacco product users.

† All racial/ethnic groups assessed are non-Hispanic unless otherwise specified

§ Data were pooled across four cycles of National Youth Tobacco Surveys (2014–2017) to increase precision of estimates among racial/ethnic minorities. Pooled sample sizes for each racial/ethnic group during 2014–2017 were 32,358 whites; 11,664 blacks; 21,337 Hispanics; 3,321 Asians; 1,213 AI/ANs; 456 NHOPIs; and 4,106 multiracial persons.

¶ Any tobacco product use was defined as use of one or more tobacco product types, including hand-rolled cigarettes. Smokers of hand-rolled cigarettes are not reported separately because the vast majority (88.7%) of these persons reported ever smoking regular cigarettes.

** Chewing tobacco/snuff/dip/snus/dissolvable tobacco.

†† Prevalence significantly different within demographic subgroups (male versus female; MS versus HS) ($p < 0.05$).

§§ Estimates not presented because relative standard error $\geq 30\%$.

Males reported significantly higher ever-use of any tobacco product than did females among whites (males: 34.6% versus females: 29.5%); Hispanics (36.4% versus 33.8%); and Asians (18.5% versus 13.8%); no significant gender differences were observed for the other racial/ethnic groups. Across all racial/ethnic groups, ever-use of any tobacco product was significantly higher among high school students than among middle school students. By specific product, e-cigarettes were the most commonly ever-used tobacco product, both overall (22.9%) and among all racial/ethnic groups except black and AI/AN students, who reported higher ever-use of cigars (19.9%) and cigarettes (31.4%), respectively (Table 1).

Current Use of Tobacco Products

Current use of any tobacco product was highest among NHOPIs (23.4%) and AI/ANs (20.6%), followed by multiracial persons (16.5%); whites (15.3%); Hispanics (14.6%), blacks (11.5%), and Asians (5.0%) (Table 2). Compared with Asians, and controlling for sex and school level, current use of any tobacco product was significantly higher among blacks (aPR = 2.33; 95% CI = 1.91–2.82), Hispanics (2.97; 2.46–3.58), whites (3.08; 2.56–3.71), multiracial persons 3.37; 2.74–4.13), NHOPIs (4.61; 3.44–6.19), and AI/ANs (4.84; 3.78–6.21) (Figure). Males reported significantly higher current use of any tobacco product than did females among whites (males: 18.0% versus females: 12.6%), Hispanics (15.6% versus 13.5%), NHOPIs (29.6% versus 16.8%), and multiracial persons (19.5% versus 13.8%); no significant gender differences were observed for the other racial/ethnic groups. Across all racial/ethnic groups, current use of any tobacco product was significantly higher among high school students than among middle school students. E-cigarettes were the most common currently used tobacco product overall (9.2%) and among all racial/ethnic groups except black students (Table 2), among whom the most common currently used product was cigars (6.7%), followed by e-cigarettes (5.1%).

Discussion

Marked disparities in tobacco product use exist among U.S. youths by race/ethnicity. Tobacco product use is higher among NHOPIs and AI/ANs, with nearly one in two NHOPI (45.1%) and AI/AN (43.8%) youths reporting ever using at least one tobacco product. Early exposure to nicotine during adolescence can lead to stronger addiction to tobacco products (2), and tobacco product experimentation is a critical step in developing dependence (4). Given that most adult smokers first try cigarettes before age 18 years, and that progression from occasional to daily smoking typically occurs during early adulthood (2), these disparities among youths might contribute to the higher rates of tobacco product use among adults from these racial/ethnic groups (1,5).

Summary

What is known about this topic?

Wide disparities in tobacco product use have been documented among the largest U.S. racial/ethnic groups; however, apart from the three most populous groups (non-Hispanic whites, non-Hispanic blacks, and Hispanics), little is known about tobacco use among youths from other racial/ethnic groups.

What is added by this report?

During 2014–2017, ever-use and current use of any tobacco product among U.S. middle and high school students were highest among Native Hawaiians/Other Pacific Islanders and American Indians/Alaska Natives and lowest among Asians.

What are the implications for public health practice?

Comprehensive and sustained implementation of evidence-based, population-level tobacco control interventions could reduce prevalence of and disparities in tobacco product use among U.S. youths.

Use of specific tobacco products varied by race/ethnicity. Ever-use was highest for cigarettes among AI/AN students (31.4%), highest for cigars among black students (19.9%), and highest for e-cigarettes among all other racial/ethnic groups. E-cigarettes were the most common currently used tobacco product among youths overall (9.2%) and all racial/ethnic groups except blacks, among whom cigar smoking was most prevalent. Given that cigar smoking has historically been higher among black adults than other racial/ethnic groups (6,7), these findings suggest distinct acculturation and social norms regarding tobacco use across racial/ethnic groups.

Observed disparities in tobacco product use might also be attributable to racial/ethnic variations in targeted tobacco industry advertising, marketing, and promotional activities (1,2,8). For example, some cigarettes have been promoted using tribal icons and logos to attract AI/AN persons (1). In addition, mentholated and other flavored tobacco products have been heavily promoted to certain racial/ethnic minority populations, including black communities (9). Flavored additives can mask the harshness of tobacco products, which might make it easier for nonusers to try their first tobacco product (9).

The findings in this report are subject to at least three limitations. First, tobacco product use was self-reported and might have been subject to recall and social desirability bias. Second, small sample sizes of some subgroups within the assessed racial/ethnic categories resulted in imprecise estimates that could not be reported. Finally, these analyses used pooled data across 4 years, and therefore do not reflect possible secular trends in prevalence and disparities in youth tobacco product use.

Evidence-based strategies that have been proven to reduce youth tobacco use include tobacco product price increases, policies that protect persons from secondhand smoke exposure

TABLE 2. Prevalence of current use* of tobacco products among middle and high school students, by race/ethnicity†, sex, and school level — National Youth Tobacco Survey, United States, 2014–2017[§]

Characteristic	Any tobacco [¶]	Electronic cigarettes	Cigarettes	Cigars (cigars/cigarillos/little cigars)	Hookah	Smokeless tobacco**	Pipe tobacco	Bidis
	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)	% (95% CI)
All MS and HS students	14.3 (14–14.6)	9.2 (9.0–9.5)	5.8 (5.6–6.0)	5.3 (5.1–5.5)	4.4 (4.2–4.5)	4.0 (3.8–4.2)	0.9 (0.8–1.0)	0.6 (0.5–0.6)
White								
Overall	15.3 (14.8–15.8)	10.2 (9.8–10.6)	6.6 (6.3–6.9)	5.2 (4.9–5.5)	3.7 (3.5–4.0)	5.0 (4.7–5.3)	0.9 (0.7–1.0)	0.4 (0.3–0.5)
Male	18.0 (17.3–18.7) ^{††}	11.7 (11.1–12.3) ^{††}	7.0 (6.6–7.5) ^{††}	7.2 (6.7–7.7) ^{††}	3.8 (3.5–4.2)	7.8 (7.3–8.4) ^{††}	1.2 (1.0–1.4) ^{††}	0.6 (0.4–0.8) ^{††}
Female	12.6 (12–13.3)	8.6 (8.1–9.2)	6.1 (5.6–6.6)	3.2 (2.9–3.5)	3.6 (3.3–4.0)	2.2 (1.8–2.5)	0.5 (0.4–0.7)	0.2 (0.2–0.3)
MS	4.9 (4.4–5.3) ^{††}	3.5 (3.1–3.9) ^{††}	1.9 (1.6–2.2) ^{††}	1.2 (1.0–1.5) ^{††}	1.1 (0.8–1.3) ^{††}	1.6 (1.3–1.9) ^{††}	0.4 (0.2–0.5) ^{††}	0.2 (0.1–0.4) ^{††}
HS	23.0 (22.2–23.8)	15.1 (14.5–15.7)	10.0 (9.5–10.6)	8.2 (7.7–8.6)	5.7 (5.3–6.1)	7.5 (7.1–8.0)	1.2 (1.0–1.4)	0.6 (0.4–0.7)
Black								
Overall	11.5 (10.7–12.3)	5.1 (4.6–5.7)	3.3 (2.8–3.7)	6.7 (6.1–7.3)	3.5 (3.1–4.0)	1.5 (1.2–1.7)	0.6 (0.4–0.7)	0.6 (0.4–0.8)
Male	12.2 (11.0–13.4)	5.8 (5.0–6.5) ^{††}	4.1 (3.4–4.8) ^{††}	7.6 (6.6–8.5) ^{††}	3.3 (2.6–3.9)	2.0 (1.5–2.4) ^{††}	0.7 (0.4–1.0)	0.6 (0.3–0.8)
Female	10.8 (9.7–11.9)	4.4 (3.7–5.1)	2.3 (1.8–2.8)	5.9 (5.2–6.7)	3.7 (3.1–4.4)	0.9 (0.5–1.2)	0.4 (0.2–0.6)	0.6 (0.3–0.9)
MS	5.7 (4.9–6.6) ^{††}	3.5 (2.8–4.1) ^{††}	1.7 (1.2–2.1) ^{††}	2.5 (2.0–3.1) ^{††}	1.8 (1.3–2.3) ^{††}	1.2 (0.7–1.6)	0.5 (0.2–0.8)	0.4 (0.2–0.6)
HS	15.8 (14.6–17.0)	6.3 (5.5–7.1)	4.3 (3.7–5.0)	9.9 (8.9–10.8)	4.8 (4.1–5.4)	1.6 (1.2–2.0)	0.6 (0.4–0.8)	0.8 (0.5–1.0)
Hispanic								
Overall	14.6 (14.0–15.2)	9.9 (9.4–10.4)	5.7 (5.3–6.1)	5.3 (4.9–5.7)	6.2 (5.8–6.6)	3.5 (3.2–3.8)	1.2 (1.0–1.4)	0.8 (0.7–1.0)
Male	15.6 (14.7–16.5) ^{††}	11.3 (10.6–12.1) ^{††}	6.3 (5.7–6.9) ^{††}	5.9 (5.4–6.5) ^{††}	5.7 (5.2–6.2) ^{††}	4.6 (4.1–5.2) ^{††}	1.4 (1.1–1.6)	0.9 (0.7–1.1)
Female	13.5 (12.6–14.3)	8.5 (7.8–9.1)	5.0 (4.5–5.6)	4.6 (4.1–5.1)	6.6 (6.0–7.3)	2.2 (1.9–2.6)	1.0 (0.8–1.2)	0.7 (0.5–0.9)
MS	8.2 (7.4–9.0) ^{††}	6.0 (5.4–6.6) ^{††}	3.1 (2.6–3.6) ^{††}	2.6 (2.2–2.9) ^{††}	3.8 (3.2–4.3) ^{††}	2.5 (2.1–3.0) ^{††}	0.9 (0.6–1.1) ^{††}	0.6 (0.4–0.8) ^{††}
HS	19.6 (18.7–20.5)	12.9 (12.2–13.7)	7.6 (7.0–8.2)	7.3 (6.7–7.8)	8.0 (7.4–8.6)	4.0 (3.5–4.4)	1.4 (1.1–1.6)	0.9 (0.7–1.1)
Asian								
Overall	5.0 (4.1–5.9)	3.6 (2.9–4.4)	2.0 (1.4–2.5)	1.1 (0.7–1.6)	1.9 (1.4–2.4)	0.7 (0.4–1.0)	— ^{§§}	— ^{§§}
Male	5.8 (4.5–7.2)	4.1 (3.0–5.3)	2.0 (1.2–2.7)	1.4 (0.7–2.1)	1.8 (1.1–2.5)	0.9 (0.4–1.3)	— ^{§§}	— ^{§§}
Female	4.1 (2.9–5.3)	3.1 (2.0–4.2)	2.0 (1.1–2.8)	0.9 (0.4–1.3)	2.0 (1.2–2.8)	— ^{§§}	— ^{§§}	— ^{§§}
MS	1.8 (0.9–2.6) ^{††}	1.5 (0.8–2.2) ^{††}	— ^{§§}	— ^{§§}	— ^{§§}	— ^{§§}	— ^{§§}	— ^{§§}
HS	7.4 (5.9–8.8)	5.2 (4.0–6.5)	2.5 (1.7–3.3)	1.5 (0.9–2.2)	2.8 (2.0–3.6)	0.7 (0.4–1.1)	— ^{§§}	— ^{§§}
AI/AN								
Overall	20.6 (17.0–24.3)	12.7 (10.1–15.3)	10.3 (7.6–13.1)	6.8 (4.8–8.7)	5.9 (4.1–7.8)	7.7 (5.0–10.3)	— ^{§§}	— ^{§§}
Male	21.8 (17.1–26.6)	14.5 (10.8–18.2)	10.0 (6.7–13.3)	7.8 (5.0–10.7)	4.9 (2.7–7.1)	8.1 (5.1–11.1)	— ^{§§}	— ^{§§}
Female	19.1 (13.3–24.9)	10.4 (6.8–14.1)	10.9 (6.1–15.6)	5.6 (3.0–8.1)	7.2 (4.0–10.4)	6.9 (2.2–11.5)	— ^{§§}	— ^{§§}
MS	9.0 (6.2–11.9) ^{††}	6.4 (3.8–8.9) ^{††}	5.2 (2.6–7.8) ^{††}	3.0 (1.5–4.6) ^{††}	— ^{§§}	3.3 (1.7–5.0) ^{††}	— ^{§§}	— ^{§§}
HS	35.4 (28.8–42.1)	20.1 (15.5–24.8)	16.7 (11.6–21.9)	11.4 (7.5–15.2)	9.9 (6.5–13.4)	13.0 (7.7–18.3)	— ^{§§}	— ^{§§}
NHOPI								
Overall	23.4 (17.6–29.2)	18.0 (13.1–22.9)	9.5 (5.4–13.6)	11.1 (7.0–15.1)	9.5 (6.1–12.9)	7.3 (4.2–10.4)	— ^{§§}	— ^{§§}
Male	29.6 (21.0–38.3) ^{††}	25.7 (17.9–33.6) ^{††}	13.1 (6.9–19.3)	15.8 (9.1–22.6)	10.9 (5.7–16.1)	12.0 (6.3–17.7)	— ^{§§}	— ^{§§}
Female	16.8 (9.4–24.2)	9.5 (4.4–14.5)	— ^{§§}	— ^{§§}	7.3 (3.1–11.5)	— ^{§§}	— ^{§§}	— ^{§§}
MS	10.1 (4.7–15.5) ^{††}	10.8 (5.2–16.4) ^{††}	— ^{§§}	— ^{§§}	— ^{§§}	— ^{§§}	— ^{§§}	— ^{§§}
HS	32.8 (24.3–41.3)	23.1 (15.9–30.3)	12.5 (6.3–18.7)	14.2 (8.1–20.2)	11.1 (6.4–15.8)	8.5 (4.1–12.9)	— ^{§§}	— ^{§§}
Multiracial								
Overall	16.5 (15.0–18.0)	10.6 (9.3–11.9)	6.6 (5.6–7.7)	6.0 (5.1–6.9)	5.8 (4.9–6.8)	4.0 (3.2–4.9)	1.5 (1.0–2.0)	— ^{§§}
Male	19.5 (17.0–21.9) ^{††}	12.5 (10.4–14.5) ^{††}	8.0 (6.4–9.6) ^{††}	7.7 (6.1–9.3) ^{††}	6.1 (4.7–7.6)	6.9 (5.3–8.6) ^{††}	2.0 (1.2–2.9)	— ^{§§}
Female	13.8 (12.0–15.7)	8.9 (7.3–10.5)	5.4 (4.1–6.8)	4.6 (3.6–5.6)	5.6 (4.4–6.9)	1.5 (0.9–2.1)	1.0 (0.4–1.6)	— ^{§§}
MS	7.6 (6.0–9.1) ^{††}	5.3 (4.0–6.6) ^{††}	2.6 (1.8–3.4) ^{††}	2.2 (1.5–3.0) ^{††}	2.4 (1.5–3.2) ^{††}	2.2 (1.4–3.1) ^{††}	— ^{§§}	— ^{§§}
HS	23.1 (20.8–25.4)	14.5 (12.5–16.5)	9.6 (8.0–11.3)	8.8 (7.4–10.3)	8.4 (6.9–9.9)	5.4 (4.1–6.7)	2.0 (1.2–2.7)	— ^{§§}

Abbreviations: AI/AN = American Indians/Alaska Natives; CI = confidence interval; HS = high school; MS = middle school; NHOPI = Native Hawaiian/Other Pacific Islander.

* Respondents were asked on how many days in the past 30 days they used/smoked the respective tobacco products, or asked “In the past 30 days, which of the following products have you used on at least one day?” depending on the type of products and survey years. Those who answered more than one day(s) or specified their product(s) from the product list provided were classified as current tobacco product users.

† All racial/ethnic groups assessed are non-Hispanic, unless otherwise specified.

§ Data were pooled across four cycles of NYTS (2014–2017) to increase precision of estimates among racial/ethnic minorities. Pooled sample sizes for each racial/ethnic group during 2014–2017 were 32,358 whites; 11,664 blacks; 21,337 Hispanics; 3,321 Asians; 1,213 AI/ANs; 456 NHOPIs; and 4,106 multiracial persons.

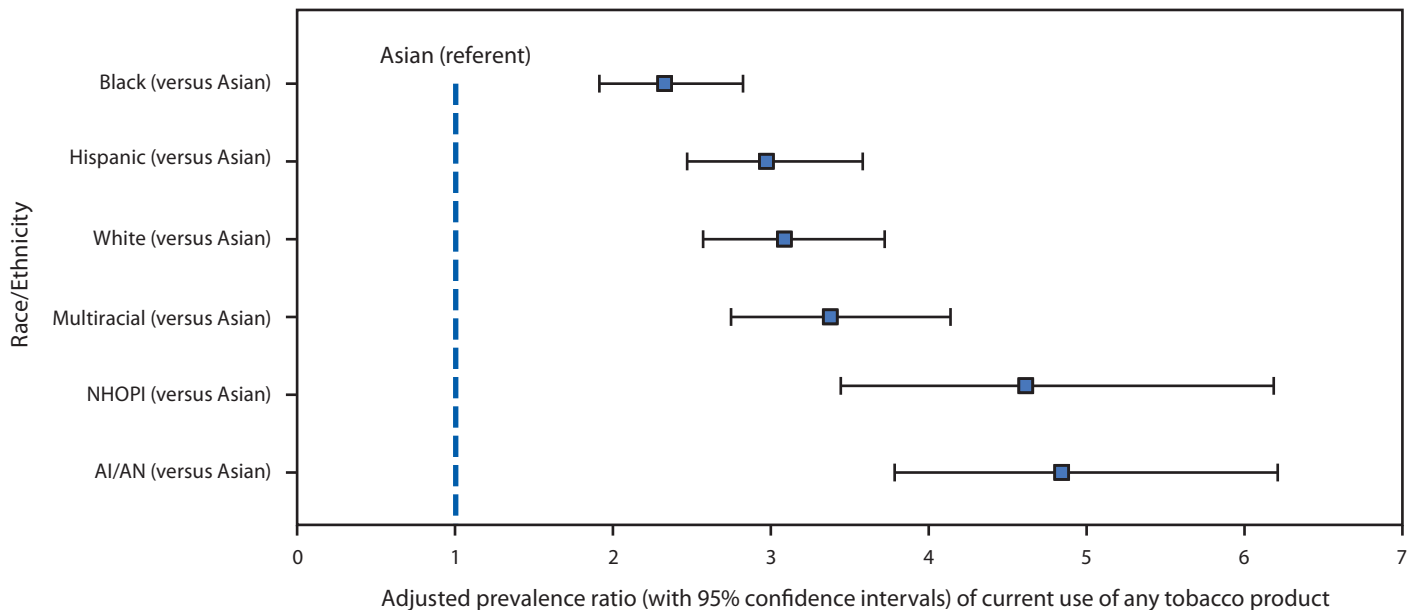
¶ Any tobacco product use was defined as use of one or more tobacco product types, including hand-rolled cigarettes. Smokers of hand-rolled cigarettes are not reported separately because the vast majority (88.7%) of these individuals reported ever smoking regular cigarettes.

** Chewing tobacco/snuff/dip/snus/dissolvable tobacco.

†† Prevalence significantly different within demographic subgroups (male versus female; MS versus HS) ($p < 0.05$).

§§ Estimates not presented because relative standard error $\geq 30\%$.

FIGURE. Adjusted prevalence ratios* of current use of any tobacco product† among middle and high school students — National Youth Tobacco Survey, United States, 2014–2017^{§,¶}



Abbreviations: AI/AN = American Indians/Alaska Natives; NHOPI = Native Hawaiian/Other Pacific Islander.

* Adjusted prevalence ratios and 95% confidence intervals (presented as whiskers) were obtained using Poisson regression models adjusted for sex and school level, with the group with the lowest prevalence of current use of any tobacco product (Asians, 5.0%) serving as the referent. Among other racial and ethnic groups, prevalence was NHOPIs 23.4%; AI/ANs 20.6%; multiracial 16.5%; whites 15.3%; Hispanics 14.6%; and blacks 11.5%.

† Current (≥ 1 time in the past 30 days) use of any tobacco product was defined as current use of one or more tobacco product types: cigarettes, cigars (including cigarillos and little cigars), smokeless tobacco (including chewing tobacco, snuff, dip, snus, and dissolvable tobacco), electronic cigarettes, hookahs, pipes, bidis, and hand-rolled cigarettes.

§ Data were pooled across four cycles of National Youth Tobacco Survey (2014–2017) to increase precision of estimates among racial and ethnic minorities. Pooled sample sizes for each racial/ethnic group during 2014–2017 were 32,358 whites; 11,664 blacks; 21,337 Hispanics; 3,321 Asians; 1,213 AI/ANs; 456 NHOPIs; and 4,106 multiracial.

¶ All racial/ethnic groups assessed are non-Hispanic unless otherwise specified.

from combustible tobacco and e-cigarette aerosol, advertising and promotion restrictions, national public education campaigns, and strategies to reduce youth access to flavored tobacco products, including menthol (1,2,9). In addition, states and communities have worked to reduce youth tobacco use by raising the minimum age for sale of tobacco products to 21 years (10). As of August 2018, six states (California, Hawaii, Maine, Massachusetts, New Jersey, and Oregon) and several hundred localities have raised the minimum age of tobacco product sales to 21 years.** Ensuring that these interventions reach all population groups, coupled with targeted strategies that acknowledge sociocultural dimensions of tobacco use among racial/ethnic groups, could reduce tobacco product use and tobacco-related disparities among U.S. youths (2,5).

** <https://tobacco21.org/>.

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Cholera Outbreak in Dadaab Refugee Camp, Kenya — November 2015–June 2016

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Dadaab Refugee camp in Garissa County, Kenya, hosts nearly 340,000 refugees in five subcamps (Dagahaley, Hagadera, Ifo, Ifo2, and Kambioos) (1). On November 18 and 19, 2015, during an ongoing national cholera outbreak (2), two camp residents were evaluated for acute watery diarrhea (three or more stools in ≤ 24 hours); *Vibrio cholerae* serogroup O1 serotype Ogawa was isolated from stool specimens collected from both patients. Within 1 week of the report of index cases, an additional 45 cases of acute watery diarrhea were reported. The United Nations High Commissioner for Refugees and their health-sector partners coordinated the cholera response, community outreach and water, sanitation, and hygiene (WASH) activities; Médecins Sans Frontières and the International Rescue Committee were involved in management of cholera treatment centers; CDC performed laboratory confirmation of cases and undertook GIS mapping and postoutbreak response assessment; and the Garissa County Government and the Kenya Ministry of Health conducted a case-control study. To prevent future cholera outbreaks, improvements to WASH and enhanced disease surveillance systems in Dadaab camp and the surrounding area are needed.

Investigation and Findings

Case ascertainment. A suspected cholera case was defined as the occurrence of acute watery diarrhea in any person aged ≥ 2 years seen at a camp health facility on or after November 18, 2015, or in a child aged < 2 years who was epidemiologically linked to a confirmed cholera case. Stool specimens were collected from one of every 2–3 patients with suspected cholera and tested using standard microbiological methods*; cholera isolates were tested for antimicrobial resistance by disc-diffusion. Suspected cases with a stool culture positive for *V. cholerae* were considered to be laboratory-confirmed (3). Demographic and clinical data were recorded for all suspected and confirmed cases. Characteristics of cholera cases were described, and case fatality rates calculated. Geographic information system software was used to map the calculated cumulative attack rates by age, sex, and residential block in

each of the five subcamps. Spatial clustering of cholera cases by block, adjusted to the block's population density, was evaluated using the software's Average Nearest Neighbor function (4), which indicated clustering in some blocks.

During November 18, 2015–June 6, 2016, a total of 1,797 cases of cholera, including 1,548 suspected and 249 confirmed, were reported among the camp's 348,781 residents (Figure); 20 cases that occurred in persons from the host community and were treated in the camp health facilities are included. Males accounted for 904 (51%) cases. The overall attack rate was 5.1 per 1,000 residents, with the highest attack rate occurring in children aged 2–4 years (16.9); attack rates varied by subcamp (Table 1). Fourteen deaths were reported (case fatality rate = 0.79%).

After identification of the two index cases on November 18 and 19, the outbreak quickly spread in the subcamps of Hagadera (attack rate = 8.0 per 1,000; peak = December 18–27), Dagahaley (7.7; November 28–December 7), and Kambioos (5.3; December 28–January 6) (Table 1); among 252 residential blocks in these three subcamps, 195 (77%) reported at least one case. Fewer cases were reported in Ifo (attack rate = 0.6 per 1,000) and Ifo2 (1.7), where only 10%–30% of residential blocks reported at least one case. Incidence among affected residential blocks ranged from 0.1% to 20%; spatial clustering of cases occurred in all the subcamps within the residential blocks ($p < 0.01$).

Identification of risk factors. In December 2015, the Kenya Ministry of Health conducted a case-control study in the subcamps most affected (Dagahaley and Hagadera) to identify risk factors for cholera. Persons with suspected or confirmed cholera (one per household) clinically evaluated before December 31, 2015, were eligible for inclusion. Eligible controls were Dagahaley or Hagadera residents aged ≥ 2 years with no history of acute watery diarrhea during the same period. Each case-patient was frequency matched to two controls by subcamp and age group (2–4 years, 5–14 years, 15–24 years, and ≥ 25 years). A standardized questionnaire was developed that adopted some questions from previous efforts, and it was administered to case-patients and controls (or their caregivers) to collect demographic and exposure information. Partially adjusted odds ratios (ORs) and 95% confidence intervals

*Rapid diagnostic tests (RDT) for cholera were also used for cholera diagnosis in the camp; however, cholera RDT testing and results were not collected systematically, and are not presented here.

FIGURE. Suspected and confirmed cholera cases (N = 1,797), by week of illness onset — Dadaab refugee camp, Kenya, November 18, 2015–June 6, 2016

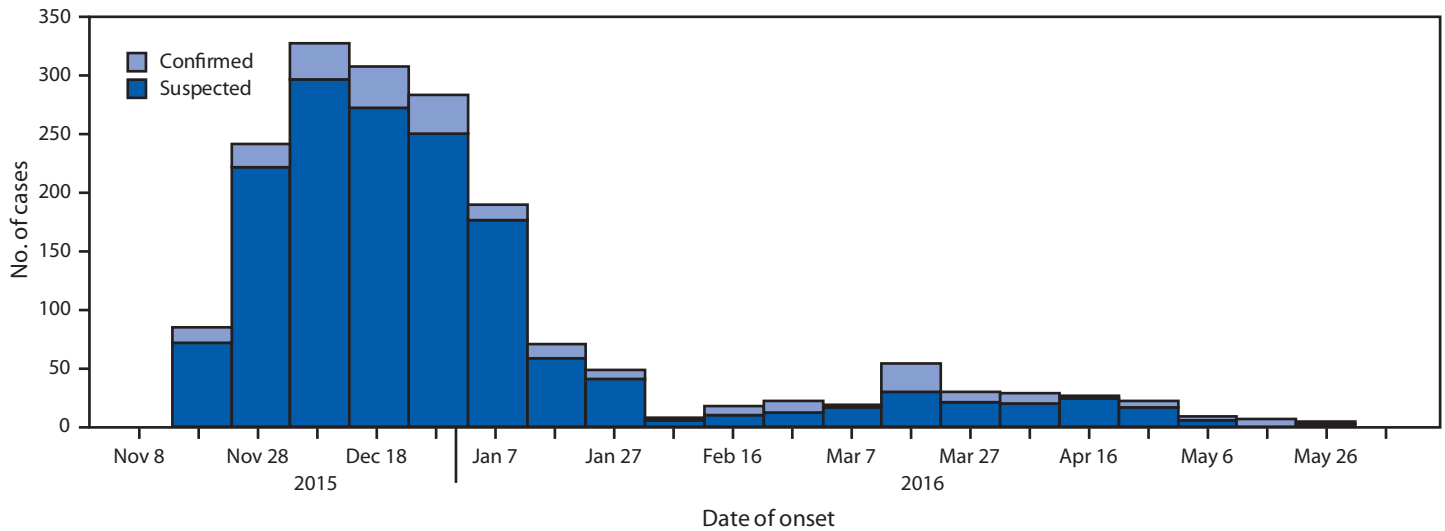


TABLE 1. Cholera attack rate and case fatality rate, by age and subcamp — Dadaab refugee camp, Kenya, November 18, 2015–June 6, 2016

Subcamp/ Age group	Total population	No. of cases*	Attack rate (per 1,000)	No. of deaths (CFR %)
Overall	348,781	1,777	5.1	14 (0.79)
Subcamp				
Hagadera	106,925	860	8.0	6 (0.70)
Dagahaley	87,617	678	7.7	5 (0.73)
Kambioos	19,612	103	5.3	2 (1.94)
Ifo2	49,814	83	1.7	1 (1.20)
Ifo	84,813	53	0.6	0 (—)
Age group (yrs)				
2–4	32,882 [†]	555	16.9	6 (1.08)
5–14	109,808	550	5.0	3 (0.55)
15–24	75,430	253	3.4	0 (—)
≥25	108,739	420	3.9	5 (1.19)

Abbreviation: CFR = case fatality rate.
 * Twenty cholera cases that occurred in the host community are not included.
[†] Estimated based on the assumption that among 54,804 children aged 0–4 years, 60% were aged 2–4 years.

(CIs) were calculated. Unconditional logistic regression using stepwise forward selection was used for building a multivariate model. Independent variables with p-values ≤0.2 in univariate analysis were considered for inclusion. Adjusted ORs (aORs) and 95% CIs were calculated from the final multivariate model.

From a calculated sample size of 38 cases and 76 controls, 32 case-patients and 64 controls were enrolled in the case-control study (Table 2). Identified risk factors for suspected or confirmed cholera included observation by interviewer of 1) human fecal and solid waste in a compound, 2) soiled communal latrines or self-reported open defecation, 3) swimming in rainwater pools, 4) sharing of food from a common plate, and 5) reported sharing of a latrine with someone with

TABLE 2. Reported exposures among 32 cholera case-patients and 64 controls during a cholera outbreak — Dadaab refugee camp, Kenya, December 2015

Exposure*	No. reporting exposure (%)		Partially adjusted OR (95% CI) [†]	Adjusted OR (95% CI) [†]
	Cases (N = 32)	Controls (N = 64)		
Use of soiled communal latrine	13 (41)	3 (4.7)	14 (3.6–54.0)	— [§]
Visible solid and human waste in compound	15 (47)	5 (7.8)	10 (3.3–32.0)	7.7 (2.0–30.0)
Swimming in rainwater pools	6 (19)	2 (3.1)	7.2 (1.4–38.0)	— [§]
Sharing latrine with a person with diarrhea	11 (34)	6 (9.4)	5.1 (1.7–15.0)	— [§]
Practicing open defecation	23 (72)	23 (36)	4.5 (1.8–12.0)	13.0 (3.0–61.0)
Sharing food from a common plate	32(100)	42(66)	3.4 (1.5–9.9)	5.9 (1.5–23.0)
Always washing hands with soap and water after using latrine	16 (50)	52 (81)	0.3 (0.1–0.8)	— [§]
Owning household latrine	19 (59)	53 (83)	0.3 (0.1–0.8)	— [§]

Abbreviations: CI = confidence interval; OR = odds ratio.
 * All participants used communal piped water from tap stands; levels of free residual chlorine and fecal coliforms unknown.
[†] Unconditional large sample logistic regression. All partially adjusted models included age and residence. Final ORs are adjusted for age, residence, visible solid and human waste in compound, practicing open defecation, and sharing food from a common plate.
[§] Adjusted OR not calculated because p-value >0.05 for partially adjusted bivariate association.

diarrhea. Always washing hands with soap and water after using a latrine and household latrine ownership were protective. Living in a compound with visible human and solid waste (aOR = 7.7; 95% CI = 2.0–30.0), self-reported open defecation (13.0; 3.0–61.0), and sharing food on a common plate

(5.9; 1.5–23.0) remained significant in the final multivariate model. No evidence of disease clustering by ethnic background or geographic origin was found.

Assessment of outbreak control measures. In late January 2016, as the outbreak waned, CDC and the United Nations High Commissioner for Refugees conducted site visits in four subcamps to assess outbreak control measures. Residual chlorine levels were below outbreak standards in various water sources, including tap stands (outbreak standard = 1.0 mg/L) and households (outbreak standard = 0.5 mg/L), and handwashing facilities in schools, markets, and eateries were insufficient. Pools of stagnant water where children played were observed near affected residential blocks in Dagahaley and Ifo2 subcamps. Although the average number of persons per latrine in Dadaab met the international standard for refugee camps (one latrine per 20 persons) (5,6), in some subcamps, up to 60 persons were observed to be sharing one latrine. In addition, at the outbreak onset in November 2015, only 168 community health workers were in the camp (approximately one per 2,000 residents), one quarter of the internationally recommended standard of one per 500 residents (5). Some households anecdotally reported cases of cholera in multiple household members, although this information was not systematically collected.

Assessment of antibiotic susceptibility of cholera isolates. *V. cholerae* serogroup O1, serotype Ogawa was isolated from 312 (39%) of 791 stool specimens. All isolates were sensitive to tetracycline, ceftriaxone, cotrimoxazole, gentamycin, and chloramphenicol; 97% were sensitive to ciprofloxacin. All isolates had intermediate sensitivity to erythromycin and were resistant to furazolidone and nalidixic acid.

Public Health Response

Cholera treatment centers were established by Médecins Sans Frontières and the International Rescue Committee, and active surveillance for cases of acute watery diarrhea was enhanced. A health promotion and hygiene campaign was conducted, primarily through mobilization of community health workers (from 168 during the first few weeks of the outbreak to 286) and hygiene promoters and use of media networks, especially radio. Frequent coordination meetings were held among stakeholders to provide updates and revise recommendations. Water from boreholes was chlorinated, soap was distributed, and bedding and latrine disinfection in affected households was carried out. WASH partners were advised to maintain adequate chlorination levels, install additional tap stands and latrines (especially in unofficial settlement areas), and install additional handwashing facilities in schools, eateries, and marketplaces.

Summary

What is already known about this topic?

Cholera, caused by infection with the bacterium *Vibrio cholerae* through ingestion of contaminated food or water, can spread rapidly in densely populated settings such as refugee camps.

What is added by this report?

During November 18, 2015–June 6, 2016, the largest cholera outbreak (1,797 cases; attack rate 5.1 per 1,000) in the history of Dadaab refugee camp in Kenya occurred. Significant risk factors included living in a compound where open defecation, visible human and solid waste, and eating from a shared plate were common. Chlorine levels in water were below standard, and handwashing facilities were insufficient.

What are the implications for public health practice?

Improvements to water and sanitation, expansion of capacity for community outreach, and enhanced camp security and disease surveillance systems in Dadaab camp and the surrounding area are urgently needed.

Discussion

Cholera is an acute diarrheal illness caused by the toxin secreted during infection with *V. cholerae* bacterium after ingestion of contaminated food or water. The infection is frequently mild or asymptomatic; however, approximately 5%–10% of infected persons develop severe disease and profuse watery diarrhea. Without prompt treatment, persons with severe disease can die within hours (7). Cholera outbreaks can spread rapidly in densely populated settings such as refugee camps (8). Rapid detection and control of cholera outbreaks is a goal for the implementation of the World Health Organization's International Health Regulations and for global health security.

This cholera outbreak is the largest reported in Dadaab refugee camp since its establishment in 1992. Existing laboratory capacity and effective disease surveillance in the camp facilitated early detection and eventual control of the outbreak. Antimicrobial susceptibility testing confirmed that *V. cholerae* circulating in the camp was susceptible to antibiotics used in treating patients with severe cholera (9).

All subcamps were affected, but the highest rates occurred in the two largest subcamps. These two areas had poorer water drainage and were in proximity to food markets where persons from areas with active cholera transmission interact. These factors might have contributed to the large number of cases and high attack rates in these areas. Furthermore, in these subcamps, large numbers of housing structures were outside the official boundaries of the camp, in areas that had poorer water and sanitation infrastructure. Inadequate residual chlorine levels in drinking water and the presence of standing

bodies of water used for swimming and bathing suggest possible waterborne transmission routes. Reported infection of multiple household members and spatial case clustering among residential blocks suggest household transmission or common exposures to a contaminated food or water source.

Limited promotion of hygiene messaging during the early weeks of the outbreak could also have increased vulnerability to cholera transmission. Sustained and intensive hygiene promotion and WASH interventions in the most affected blocks were also recommended.

The findings in this report are subject to at least two limitations. First, camp insecurity affected case finding and study enrollment, precluding achieving the calculated sample size for the case-control study. Second, other cases of acute watery diarrhea might have been included as cases of suspected cholera, reducing the power of the study.

The last reported cholera case in the camp occurred on June 6, 2016, and the outbreak was declared over on June 21, 2016. Thereafter, two small clusters of cases not associated with traveling outside the camp or contact with visitors outside the camp were reported in July (seven cases) and August (five cases) 2016. During April–August 2017, a cholera outbreak involving 511 cases occurred after flooding that destroyed approximately 9,000 latrines. In September 2017, four cases were reported, and during October–December 2017, 109 cases were reported. Both outbreaks were controlled immediately with no deaths. Improvements to WASH and disease surveillance are needed to prevent future outbreaks.

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Notes from the Field:

Fatal *Vibrio anguillarum* Infection in an Immunocompromised Patient — Maine, 2017

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In July 2017, a woman aged 65 years was evaluated at a hospital emergency department in Maine for an approximately 10-cm area of necrosis on her left lower leg identified as likely skin and soft tissue infection. The patient noted pain in the area that morning and was unable to walk when examined later that day. Computed tomography indicated extensive cellulitis in the area; she was hospitalized and treated with intravenous antibiotics. The Maine Health and Environmental Testing Laboratory identified *Vibrio anguillarum* from blood cultures collected after admission and before starting treatment; stool and wound cultures were not collected. Approximately 36 hours after she first arrived at the emergency department, the patient developed septic shock and multiorgan failure, dying 2 days after admission.

The patient spent the summer in Maine and lived the rest of the year in a southern coastal state. She was in Maine during her exposure period and illness and had reported vomiting and diarrhea 9 days before seeking treatment at the hospital, which had resolved in 1 day, and one additional diarrhea episode the night before admission. Her husband reported that she had eaten a lobster roll from a local restaurant and a cod loin cooked at home approximately 10 days before the onset of leg pain and a lobster and other seafood dip purchased at a grocery store approximately 5 days before onset of leg pain; he could not recall specifically when each of these foods was consumed in relation to the onset of vomiting and diarrhea. None of the seafood was reported to have been raw or undercooked. The patient had also waded at a beach as recently as the day before seeking treatment, but she did not have any known wounds. Her husband reported that she was bitten by a greenhead fly while at the beach, but the location of the fly bite was not known. A traceback investigation was performed on the seafood she consumed; no informative findings were reported. Her previous medical history included multiple myeloma and amyloidosis, both of which were being treated with immunosuppressive drugs.

Vibriosis is an underrecognized and underreported infection caused by species of the family Vibrionaceae other than toxigenic *Vibrio cholerae* O1 and O139, which cause cholera (1,2). Typically, Vibrionaceae cause gastrointestinal illness with mild to severe watery diarrhea, although they can also cause bacteremia,

wound, or extraintestinal infections. The most common transmission modes involve consumption of raw or undercooked shellfish or contact with seawater. Persons with liver disease, cancer, diabetes, HIV, thalassemia, or who receive immunosuppressive therapy are at increased risk for serious infection. In the United States, vibriosis causes an estimated 80,000 illnesses, 500 hospitalizations, and 100 deaths annually (2).

Vibrio bacteria are gram-negative bacilli naturally found in coastal waters. Their growth and concentration increases with warmer water temperatures, leading to a seasonal distribution of *Vibrio* infections, with most occurring from summer through early autumn (3). *V. anguillarum* (previously known as *Listonella anguillarum*) is normally a pathogen of fish, crustaceans, and bivalves and causes considerable economic losses in the fishing and aquaculture industries (4). This is the first reported instance of *V. anguillarum* associated with human illness.

Recent studies report increasing sea surface temperatures in the coastal North Atlantic corresponding with increased abundance and spread of *Vibrio* bacteria and increased *Vibrio*-associated illnesses in the United States (5,6). Vibriosis is preventable by ensuring that shellfish and seafood are fully cooked before consuming and by either avoiding exposure to seawater when a wound is present or wearing a waterproof dressing and washing wounds after seawater exposure. This immunocompromised patient had exposures to seafood and seawater. With multiple potential exposures, the source and route of infection could not be determined, but two possibilities based on the available epidemiologic data include 1) exposure of a small wound or the fly bite to contaminated seawater resulting in cellulitis and invasive blood infection, or 2) consumption of seafood contaminated with *Vibrio* that caused the diarrhea and the subsequent invasive blood infection and cellulitis. In light of this patient's comorbidities, determining whether this pathogen was the sole factor in her decline was not feasible, but the case serves as a reminder that laboratories and physicians should be aware of the possibility of illness from *Vibrio* species outside of those commonly observed.

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Notes from the Field:

Inspection of 59 “Vape Shops” — United States, October–November, 2016

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In 2009, the Food and Drug Administration (FDA) was given the authority to regulate the manufacture, marketing, and distribution of tobacco products (1). Effective August 2016, this authority was extended to all tobacco products, including electronic cigarettes (e-cigarettes) (2). Section 704(a)(1) of the Food, Drug, and Cosmetic Act gives FDA authority to conduct investigations and inspections of manufacturers (i.e., any person who manufactures, fabricates, assembles, processes, or labels a tobacco product or imports a finished tobacco product for sale or distribution in the United States) to ensure compliance with applicable federal requirements (1,2).^{*} In October and November 2016, unannounced inspections were conducted in 59 “vape shops” to learn about business and manufacturing practices, including whether establishments were retailers or manufacturers, and, among manufacturers, how the products sold were manufactured and whether the manufacturers were aware of FDA regulations regarding tobacco products. This report summarizes these first 59 inspections, which showed a lack of quality assurance programs, standard operating procedures, and full labeling of ingredients by the inspected manufacturers.

Vape shops engage in activities that include selling electronic nicotine delivery systems (ENDS), ENDS replacement pieces, and ENDS premixed flavored “e-liquids” and mixing or preparing combinations of liquid nicotine, flavors, and other liquids for sale to consumers. Before an inspection was initiated, a notice of inspection was issued to the most senior staff member at the shop, who also received a summary of the findings once the inspection was closed. Sixty establishments in four states were selected; one smoking lounge that did not sell or manufacture products was excluded from subsequent analysis.

The 59 inspected shops included 31 retailers that only sold finished products, 27 that were both manufacturers and retailers, and one manufacturer that did not sell to consumers. Personnel at all shops reported being aware of FDA tobacco product regulation. The remainder of this report focuses on the 28 manufacturers.

Among the 28 manufacturers, 14 were small businesses employing approximately three persons each; the remaining 14 reported having a parent corporation, a subsidiary, or an

affiliate company. All 28 manufacturers sold brands of tobacco products made by other manufacturers as well as house brands manufactured on-site in an area away from customers. No assessed manufacturers allowed customers to mix their own products on-site.

Among the 28 manufacturers inspected, 25 identified a nicotine concentration on the label of products offered for sale, which ranged from 0–100 mg/mL. During the manufacturing process, the assessed manufacturers generally reported using recipes to make products and employed automated pipettes, graduated burettes, or disposable syringes to measure the volumes of liquid, including nicotine, flavoring, propylene glycol, and vegetable glycerin specified in the recipe. Only one manufacturer reported testing the finished products to ensure the product contained the concentration of nicotine indicated on the label. Six establishments indicated that they request testing records from the manufacturers of the branded tobacco products they sold. None of the establishments had quality assurance programs or practices, standard operating procedures, or standardized job training for manufacturing house brands of tobacco products. Workers received on-the-job training and used recipes.

This is the first assessment of business and manufacturing practices at vape shops, which have only recently become regulated. Although all vape shops inspected were found to be in compliance with the regulatory requirements of FDA that were in effect at the time of the inspection, the lack of quality assurance programs, standard operating procedures, and full labeling of ingredients by the inspected manufacturers suggests that consumers might not receive complete information regarding product contents or purchase products of consistent quality; these concerns might be addressed by future FDA regulatory activities. FDA continues to conduct inspections, which will provide additional information on industry practices and compliance with FDA’s regulatory requirements and might inform state and local vape shop policies.

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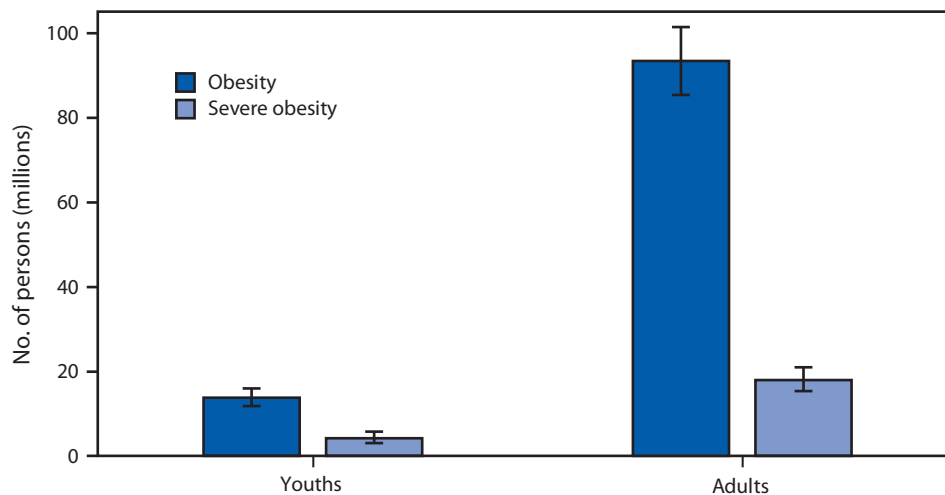
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QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Number* of Youths Aged 2–19 Years and Adults Aged ≥20 Years with Obesity[†] or Severe Obesity[‡] — National Health and Nutrition Examination Survey, 2015–2016



* 95% confidence intervals indicated with error bars. The number of adults and youths with obesity and severe obesity was calculated by multiplying the unadjusted (crude) prevalence by the average population totals for 2015–2016 (74.3 million youths and 234.5 million adults). Prevalence estimates exclude pregnant females.

[†] Body mass index (BMI) is calculated as weight in kilograms divided by height in meters squared. Obesity among youths was defined as sex-specific BMI-for-age at or above the 95th percentile of the CDC growth charts, and obesity among adults was defined as BMI ≥ 30 .

[‡] Severe obesity among youths was defined as sex-specific BMI-for-age at or above 120% of the 95th percentile of the CDC growth charts, and severe obesity among adults was defined as BMI ≥ 40 .

During 2015–2016, in the United States, there were 13.7 million youths (18.5%) with obesity, including 4.2 million youths (5.6%) with severe obesity. During this same period, in the United States, there were 93.3 million adults (39.8%) with obesity, including 17.8 million adults (7.6%) with severe obesity.

Sources: Hales et al. Trends in Obesity and Severe Obesity Prevalence in US Youth and Adults by Sex and Age, 2007–2008 to 2015–2016. <https://jamanetwork.com/journals/jama/fullarticle/2676543>; National Center for Health Statistics, National Health and Nutrition Examination Survey, 2015–2016. <https://www.cdc.gov/nchs/nhanes.htm>.

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