Centers for Disease Control and Prevention
MMWR
Morbidity and Mortality Weekly Report
Weekly / Vol. 65 / No. 13
April 8, 2016

Sleep Duration and Injury-Related Risk Behaviors Among High School Students — United States, 2007–2013

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Insufficient sleep is common among high school students and has been associated with an increased risk for motor vehicle crashes (*1*), sports injuries (*2*), and occupational injuries (*3*). To evaluate the association between self-reported sleep duration on an average school night and several injury-related risk behaviors (infrequent bicycle helmet use, infrequent seatbelt use, riding with a driver who had been drinking, drinking and driving, and texting while driving) among U.S. high school students, CDC analyzed data from 50,370 high school students (grades 9–12) who participated in the national Youth Risk Behavior Surveys (YRBSs) in 2007, 2009, 2011, or 2013. The likelihood of each of the five risk behaviors was significantly higher for students who reported sleeping ≤7 hours on an average school night; infrequent seatbelt use, riding with a drinking driver, and drinking and driving were also more likely for students who reported sleeping ≥10 hours compared with 9 hours on an average school night. Although insufficient sleep directly contributes to injury risk, some of the increased risk associated with insufficient sleep might be caused by engaging in injury-related risk behaviors. Intervention efforts aimed at these behaviors might help reduce injuries resulting from sleepiness, as well as provide opportunities for increasing awareness of the importance of sleep.

The national YRBS monitors health-risk behaviors among students in public and private high schools and is conducted by CDC in the spring of odd-numbered years. Each national YRBS uses an independent, three-stage cluster sample design to obtain a nationally representative sample of students in grades 9–12. The overall response rates* were 68% in 2007, 71% in 2009, 71% in 2011, and 68% in 2013, and sample sizes ranged from 13,583 (2013) to 16,410 (2009).† Students completed the anonymous, self-administered questionnaires during a single class period.

The combined analytic sample was composed of 50,370 high school students who responded to questions about sleep duration on an average school night (≤4 hours, 5 hours, 6 hours, 7 hours, 8 hours, 9 hours, ≥10 hours); demographic characteristics (sex, grade, and race/ethnicity); and how frequently they used a bicycle helmet (among students who had ridden a bicycle during the past 12 months; responses = never or rarely versus sometimes, most of the time, or always); wore a seatbelt when riding in a car driven by someone else (never or rarely versus sometimes, most of the time, or always); rode in a car or other vehicle with a driver who had been drinking alcohol (i.e., rode with a drinking driver; at least one time during the

* Overall response rate = (number of participating schools/number of eligible sampled schools) × (number of usable questionnaires/number of eligible students sampled).
† Data users manuals (http://www.cdc.gov/healthyyouth/yrbs/data/index.htm).

Continuing Education examination available at http://www.cdc.gov/mmwr/cme/conted_info.html#weekly.

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past 30 days versus 0 times); drove a car or other vehicle when they had been drinking alcohol[§] (i.e., drinking and driving; at least one time during the past 30 days versus 0 times); or texted or e-mailed while driving a car or other vehicle[¶] (i.e., texting while driving; at least 1 day during the past 30 days versus 0 days). The percentage reporting insufficient sleep duration (≤7 hours according to the *Healthy People 2020* sleep objective for adolescents[**]) and distribution of hours of sleep were calculated by survey year, sex, grade, and race/ethnicity; pairwise t-tests and ANOVA (i.e., linear trend) were used to assess crude significant differences.

Because no differences were found in mean sleep duration or prevalence of insufficient sleep duration by survey year, data from all four survey years were aggregated for subsequent analyses. Aggregating the data from four survey years provided adequate sample size for the calculation of low prevalence risk behaviors among students reporting each category of sleep duration. Unadjusted prevalence of each risk behavior was calculated by sleep duration. Pairwise t-tests were used to assess significant differences compared with 9 hours, the median of the sleep duration recommendation for teens by the National Sleep Foundation (*4*). Logistic regression analyses were used to calculate adjusted prevalence ratios (APRs) and 95% confidence intervals (CIs) for the likelihood of each injury-related behavior with a referent sleep duration of 9 hours and were adjusted for sex, grade, and race/ethnicity. All analyses accounted for the sampling weights and complex survey design. P-values of <0.05 were defined to be statistically significant.

Reported sleep duration during an average school night was ≤4 hours for 6.3% of respondents, 5 hours (10.5%), 6 hours (21.9%), 7 hours (30.1%), 8 hours (23.5%), 9 hours (5.8%), and ≥10 hours (1.8%). Sleep duration varied by sex, grade, and race/ethnicity (Table 1). Female students reported a higher prevalence of insufficient sleep (≤7 hours) than did male students (71.3% versus 66.4%, p<0.001). The percentage reporting insufficient sleep ranged from 59.7% of students in 9th grade to 76.6% of students in 12th grade (p<0.001 for linear trend). Among racial/ethnic groups, the prevalence of insufficient sleep was lowest for American Indian/Alaska Native students (60.3%) and highest for Asian students (75.7%).

Overall, 86.1% of students reported infrequent bicycle helmet use and 8.7% reported infrequent seatbelt use. Twenty-six percent of students reported riding with a drinking driver at least one time during the past 30 days; 8.9% of students reported drinking and driving; and 30.3% reported texting while driving during the past 30 days. Unadjusted prevalence

[§] In the 2013 survey, for the first time, the response options for this question included "I did not drive a car or other vehicle during the past 30 days." For compatibility with the results for the drinking and driving question in the 2007, 2009, and 2011 surveys, students who responded "I did not drive" on the 2013 survey were counted as 0 times and included in the denominators.

[¶] This question was asked for the first time in the 2011 survey. In the 2013 survey, the response options for this question included "I did not drive a car or other vehicle during the past 30 days." For compatibility with the results for the texting or e-mailing and driving question in the 2011 survey, students who responded "I did not drive" on the 2013 survey were counted as 0 days and included in the denominators.

[**] https://www.healthypeople.gov/2020/topics-objectives/topic/sleep-health.

The *MMWR* series of publications is published by the Center for Surveillance, Epidemiology, and Laboratory Services, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30329-4027.

Suggested citation: [Author names; first three, then et al., if more than six.] [Report title]. MMWR Morb Mortal Wkly Rep 2016;65:[inclusive page numbers].

TABLE 1. Duration of sleep on an average school night, by selected characteristics—Youth Risk Behavior Surveys, United States, 2007, 2009, 2011, and 2013.

Characteristic	No.†	Sleep duration %* (95% CI)							
		≤7 hrs (insufficient sleep)	≤4 hrs	5 hrs	6 hrs	7 hrs	8 hrs	9 hrs	≥10 hrs
Total	**50,370**	**68.8 (68.0–69.6)**	**6.3 (5.9–6.7)**	**10.5 (10.1–11.0)**	**21.9 (21.3–22.4)**	**30.1 (29.5–30.7)**	**23.5 (22.9–24.2)**	**5.8 (5.5–6.2)**	**1.8 (1.7–2.0)**
Survey year									
2007	11,939	69.0 (67.0–70.9)	5.8 (5.3–6.5)	10.0 (9.2–10.9)	22.8 (21.6–24.1)	30.4 (29.4–31.3)	23.5 (21.9–25.3)	5.8 (5.2–6.5)	1.6 (1.3–2.1)
2009	14,477	69.1 (67.5–70.6)	5.5 (4.9–6.1)	10.1 (9.0–11.4)	21.9 (20.9–23.0)	31.6 (30.4–32.9)	23.4 (22.2–24.5)	5.8 (5.3–6.4)	1.7 (1.5–2.1)
2011	11,904	68.6 (67.2–69.9)	6.6 (6.0–7.2)	10.8 (10.0–11.6)	21.7 (20.6–22.7)	29.6 (28.4–30.9)	24.0 (22.9–25.1)	5.7 (5.1–6.4)	1.7 (1.4–2.0)
2013	12,050	68.4 (66.9–69.9)	7.4 (6.6–8.3)	11.3 (10.5–12.2)	21.2 (20.2–22.2)	28.5 (27.5–29.5)	23.2 (22.2–24.2)	6.0 (5.3–6.8)	2.4 (2.0–2.8)
Sex									
Female	25,327	71.3 (70.4–72.1)	6.3 (5.9–6.8)	12.1 (11.5–12.8)	23.8 (23.0–24.6)	29.0 (28.3–29.8)	22.0 (21.3–22.8)	5.1 (4.7–5.5)	1.6 (1.4–1.8)
Male	25,043	66.4 (65.4–67.4)	6.3 (5.8–6.7)	9.0 (8.4–9.6)	20.0 (19.4–20.7)	31.1 (30.2–32.0)	25.0 (24.2–25.8)	6.6 (6.1–7.1)	2.1 (1.8–2.3)
Grade									
9th	12,551	59.7 (58.6–60.8)	5.8 (5.2–6.4)	8.4 (7.7–9.0)	17.2 (16.4–18.0)	28.3 (27.3–29.3)	28.5 (27.5–29.5)	9.1 (8.4–9.9)	2.7 (2.3–3.2)
10th	12,083	67.4 (66.1–68.8)	5.9 (5.3–6.5)	9.7 (8.9–10.6)	20.6 (19.6–21.7)	31.2 (30.0–32.4)	24.7 (23.6–25.8)	5.8 (5.2–6.4)	2.1 (1.8–2.5)
11th	12,784	73.3 (72.0–74.5)	6.8 (6.1–7.4)	11.9 (11.1–12.7)	24.4 (23.4–25.4)	30.2 (29.2–31.3)	21.0 (19.9–22.1)	4.4 (4.0–4.9)	1.3 (1.1–1.6)
12th	12,952	76.6 (75.4–77.8)	6.9 (6.3–7.5)	12.7 (11.9–13.6)	26.3 (25.1–27.5)	30.7 (29.6–31.9)	18.9 (17.9–19.9)	3.4 (3.0–4.0)	1.1 (0.8–1.4)
Race/Ethnicity									
White§	22,330	68.3 (67.3–69.4)	5.1 (4.7–5.5)	9.5 (8.9–10.1)	21.6 (20.8–22.3)	32.2 (31.4–33.0)	24.7 (23.8–25.5)	5.6 (5.2–6.1)	1.3 (1.2–1.5)
Black§	9,701	71.2 (69.9–72.5)	9.2 (8.5–10.0)	13.9 (12.8–15.2)	23.9 (22.9–24.8)	24.2 (23.1–25.3)	19.9 (18.9–20.9)	5.7 (5.1–6.3)	3.2 (2.8–3.8)
Hispanic	13,452	67.0 (65.5–68.5)	6.7 (6.1–7.4)	10.5 (9.7–11.4)	20.7 (19.8–21.7)	29.1 (28.1–30.1)	23.7 (22.7–24.9)	6.9 (6.3–7.6)	2.3 (2.0–2.7)
American Indian/ Alaska Native§	769	60.3 (52.4–67.6)	9.8 (6.7–14.1)	8.9 (6.6–12.0)	18.2 (15.1–21.9)	23.3 (20.1–26.9)	25.9 (21.7–30.7)	8.4 (5.4–12.9)	5.4 (3.8–7.5)
Asian§	1,751	75.7 (72.7–78.5)	8.3 (6.4–10.6)	13.2 (11.1–15.6)	26.4 (23.9–29.1)	27.8 (25.2–30.5)	18.1 (15.7–20.7)	4.4 (3.1–6.3)	1.8 (1.1–2.9)
Native Hawaiian/ Pacific Islander§	399	68.3 (62.1–73.9)	12.8 (9.0–18.0)	10.8 (7.5–15.2)	20.4 (16.7–24.7)	24.3 (19.3–30.2)	24.2 (19.1–30.1)	5.1 (3.1–8.5)	—¶
Multiracial§	1,968	72.0 (69.2–74.7)	9.2 (7.6–11.1)	13.3 (11.4–15.5)	22.9 (20.4–25.6)	26.6 (24.1–29.2)	21.0 (18.5–23.6)	4.9 (3.8–6.3)	2.1 (1.4–3.2)

Abbreviation: CI = confidence interval.
* Weighted percentages.
† Unweighted n's.
§ Non-Hispanic.
¶ Unreliable estimate. Relative standard error ≥0.3.

of all five injury-related risk behaviors varied by sleep duration (Table 2). The likelihood of each of the five risk behaviors was significantly higher (APR >1.0) among students with sleep durations ≤7 hours; infrequent seatbelt use, riding with drinking driver, and drinking and driving were also more likely among students reporting sleeping ≥10 hours compared with 9 hours (Table 3). The likelihood of drinking and driving was also significantly higher among students sleeping 8 hours compared with 9 hours.

Discussion

Unintentional injuries are the leading cause of death for adolescents, with approximately two thirds of these fatalities related to road traffic crashes (*5*). Excessive sleepiness, which is most often a result of not getting adequate sleep, has been shown to increase the risk for motor vehicle crashes and other unintentional injury among adolescents (*1–3*). Although insufficient sleep contributes to injury risk directly by slowing reaction time, impairing ability to pay attention, or causing a driver to fall asleep (*6*), this study provides evidence that some of the increased risk associated with insufficient sleep might be caused by engaging in injury-related risk behaviors. In addition to a higher likelihood of engaging in injury-related risk behaviors among students who reported typically sleeping ≤7 hours on school nights, infrequent seatbelt use, riding with a drinking driver, and drinking and driving were also more likely for students sleeping ≥10 hours compared with 9 hours. Although short and long sleep might simply be associated with other adolescent risk behaviors, insufficient sleep might cause persons to take more risks and disregard the possibility of negative consequences (*7*). However, depression might contribute to both sleep problems and participation in risk behaviors. Sleep problems, including both not sleeping enough and sleeping too much, are common symptoms of depression; one study found that adolescents who reported more depressive symptoms were more likely to engage in several injury-related risk behaviors, including infrequent seatbelt use, infrequent bicycle helmet use, and drinking and driving (*8*).

The findings in this report are subject to at least two limitations. First, the data were self-reported and the extent of any underreporting or overreporting cannot be determined. However, the survey questions demonstrate good test-retest reliability.†† Second, the survey is not representative of school-aged youths

†† http://www.ncbi.nlm.nih.gov/pubmed/12359379.

TABLE 2. Prevalences and 95% confidence intervals (CIs) of unintentional injury risk behaviors among high school students, by sleep duration — Youth Risk Behavior Surveys, United States, 2007, 2009, 2011, and 2013

	Sleep duration % (95% CI)						
Risk behavior	**≤4 hrs**	**5 hrs**	**6 hrs**	**7 hrs**	**8 hrs**	**9 hrs**	**≥10 hrs**
Infrequent bicycle helmet use	91.2 (89.2–92.9)*	90.6 (88.8–92.2)*	87.9 (86.3–89.4)*	85.7 (83.9–87.5)*	83.4 (81.2–85.4)	81.7 (79.2–84.0)	82.5 (78.0–86.2)
Infrequent seatbelt use	22.8 (20.6–25.2)*	12.5 (11.2–14.1)*	9.3 (8.3–10.5)*	6.5 (5.6–7.4)	5.9 (5.1–6.8)	5.5 (4.5–6.6)	13.1 (10.6–16.0)*
Rode with a drinking driver	36.8 (34.7–39.0)*	31.5 (29.8–33.3)*	28.2 (26.9–29.4)*	25.1 (23.8–26.3)*	21.4 (20.2–22.7)	19.8 (17.9–21.8)	24.0 (20.5–27.9)*
Drinking and driving	16.6 (14.9–18.4)*	11.2 (10.0–12.5)*	10.1 (9.2–11.1)*	8.3 (7.5–9.2)*	6.7 (6.1–7.4)*	4.7 (3.9–5.7)	9.9 (7.5–12.9)*
Texting while driving	32.7 (29.3–36.3)*	34.8 (31.6–38.1)*	33.4 (31.2–35.7)*	31.5 (28.7–34.5)*	26.1 (23.9–28.4)*	20.9 (17.7–24.4)	24.8 (20.5–29.7)

* Prevalence significantly different from 9 hours (p<0.05).

TABLE 3. Adjusted prevalence ratios (APRs)* and 95% confidence intervals (CIs) for unintentional injury risk behaviors among high school students, by sleep duration — Youth Risk Behavior Surveys, United States, 2007, 2009, 2011, and 2013

	Sleep duration APR (95% CI)						
Risk behavior	**≤4 hrs**	**5 hrs**	**6 hrs**	**7 hrs**	**8 hrs**	**9 hrs**	**≥10 hrs**
Infrequent bicycle helmet use	1.12 (1.08–1.15)	1.11 (1.07–1.15)	1.08 (1.05–1.11)	1.06 (1.02–1.09)	1.02 (1.00–1.06)	1.00 (Ref)	0.99 (0.94–1.05)
Infrequent seatbelt use	4.50 (3.66–5.54)	2.60 (2.10–3.22)	1.92 (1.60–2.31)	1.28 (1.06–1.54)	1.13 (0.95–1.36)	1.00 (Ref)	2.38 (1.82–3.11)
Rode with a drinking driver	1.84 (1.64–2.06)	1.58 (1.41–1.77)	1.42 (1.27–1.58)	1.27 (1.15–1.40)	1.09 (0.98–1.20)	1.00 (Ref)	1.20 (1.01–1.42)
Drinking and driving	3.14 (2.52–3.92)	2.10 (1.71–2.58)	1.84 (1.49–2.27)	1.51 (1.27–1.81)	1.32 (1.06–1.64)	1.00 (Ref)	2.19 (1.60–3.00)
Texting while driving	1.26 (1.05–1.51)	1.29 (1.11–1.49)	1.22 (1.05–1.41)	1.19 (1.03–1.37)	1.10 (0.95–1.27)	1.00 (Ref)	1.24 (1.00–1.55)

* Adjusted for sex, grade, and race/ethnicity.

who do not attend school. Nationwide, in 2012, approximately 3% of persons aged 16–17 years were not enrolled in a high school program and had not completed high school.[§§]

The National Sleep Foundation recommends that adolescents aged 14–17 years sleep 8–10 hours per night (*4*). To help ensure that adolescents get adequate sleep, they can practice good sleep hygiene (i.e., habits that promote good sleep). These habits include going to bed and getting up at the same time each day both during the school week and weekends, minimizing light exposure in the evenings, and keeping computers and other electronic devices, such as computers, video games, and cell phones, out of the bedroom.[¶¶] Parents can help by setting bedtimes and limiting when (only before a set time or "media curfew") and where (not in their bedrooms) their teenagers can use electronic devices. Early school start times contribute to insufficient sleep among adolescents. Delaying school start times has been proposed as a means of allowing adolescents to get adequate sleep (*9*). Some students naturally need more sleep than their peers, but waking up and feeling unrested in spite of adequate sleep might be an indication of a problem such as poor sleep quality or an underlying health condition. Poor sleep quality might result from poor sleep hygiene, a bad sleep environment (e.g., too warm, too noisy, or cell phones in the bedroom), or a sleep disorder. In addition, long sleep durations might be a symptom of depression.

[§§] http://nces.ed.gov/pubs2015/2015015.pdf.

[¶¶] More tips for good sleep are available from the National Sleep Foundation (https://sleepfoundation.org/sleep-tools-tips/healthy-sleep-tips).

Various resources are available to address injury-related risk behaviors. Public health practitioners can refer to systematic reviews of interventions included in The Community Guide (http://www.thecommunityguide.org/mvoi/index.html). Some evidence exists that health care providers, in collaboration with health educators, might be able to change adolescent injury-related behavior through screening and brief counseling, followed by a visit by a health educator (*9*,*10*). Information for parents of teen drivers on what they can do to encourage safe driving by their teens is available at http://www.cdc.gov/parentsarethekey/parents/index.html.

Summary

What is already known about this topic?

Insufficient sleep is common among high school students and is associated with an increased risk for unintentional injury from drowsy driving crashes and other causes.

What is added by this report?

Students who reported sleeping ≤7 hours on school nights were more likely to report several injury-related risk behaviors (infrequent bicycle helmet use, infrequent seatbelt use, riding with a driver who had been drinking, drinking and driving, and texting while driving) compared with students who sleep 9 hours.

What are the implications for public health practice?

High school faculty and administrators, as well as parents of high school students, should be made aware of the increased likelihood for risky behavioral choices among students who do not get enough sleep.

Acknowledgment

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References

1. Martiniuk AL, Senserrick T, Lo S, et al. Sleep-deprived young drivers and the risk for crash: the DRIVE prospective cohort study. JAMA Pediatr 2013;167:647–55. http://dx.doi.org/10.1001/jamapediatrics.2013.1429
2. Milewski MD, Skaggs DL, Bishop GA, et al. Chronic lack of sleep is associated with increased sports injuries in adolescent athletes. J Pediatr Orthop 2014;34:129–33. http://dx.doi.org/10.1097/BPO.0000000000000151
3. Graves JM, Miller ME. Reduced sleep duration and history of work-related injuries among Washington state adolescents with a history of working. Am J Ind Med 2015;58:464–71. http://dx.doi.org/10.1002/ajim.22416
4. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. Sleep Health 2015;1:40–3. http://dx.doi.org/10.1016/j.sleh.2014.12.010
5. CDC. Vital signs: unintentional injury deaths among persons aged 0–19 years—United States, 2000–2009. MMWR Morb Mortal Wkly Rep 2012;61:270–6.
6. Williamson A, Lombardi DA, Folkard S, Stutts J, Courtney TK, Connor JL. The link between fatigue and safety. Accid Anal Prev 2011;43:498–515. http://dx.doi.org/10.1016/j.aap.2009.11.011
7. Harrison Y, Horne JA. The impact of sleep deprivation on decision making: a review. J Exp Psychol Appl 2000;6:236–49. http://dx.doi.org/10.1037/1076-898X.6.3.236
8. Testa CR, Steinberg L. Depressive symptoms and health-related risk-taking in adolescence. Suicide Life Threat Behav 2010;40:298–305.
9. Adolescent Sleep Working Group, Committee on Adolescence, Council on School Health. School start times for adolescents. Pediatrics 2014;134:642–9. http://dx.doi.org/10.1542/peds.2014-1697
10. Ozer EM, Adams SH, Orrell-Valente JK, et al. Does delivering preventive services in primary care reduce adolescent risky behavior? J Adolesc Health 2011;49:476–82. http://dx.doi.org/10.1016/j.jadohealth.2011.02.011

Varying Estimates of Sepsis Mortality Using Death Certificates and Administrative Codes — United States, 1999–2014

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Sepsis is a clinical syndrome caused by a dysregulated host response to infection (*1*). Because there is no confirmatory diagnostic test, the diagnosis of sepsis is based on evidence of infection and clinical judgement. Both death certificates and health services utilization data (administrative claims) have been used to assess sepsis incidence and mortality, but estimates vary depending on the surveillance definition and data source. To highlight the challenges and variability associated with estimating sepsis mortality, CDC compared national estimates of sepsis-related mortality based on death certificates using the CDC WONDER database with published sepsis mortality estimates generated using administrative claims data from hospital discharges reported in the Nationwide Inpatient Sample, Healthcare Cost and Utilization Project, Agency for Healthcare Research and Quality (*2*). During 2004–2009, using data rounded to thousands, the annual range of published sepsis-related mortality estimates based on administrative claims data was 15% to 140% higher (range = 168,000–381,000) than annual estimates generated using death certificate data (multiple causes) (range = 146,000–159,000). Differences in sepsis-related mortality reported using death certificates and administrative claims data might be explained by limitations inherent in each data source. These findings underscore the need for a reliable sepsis surveillance definition based on objective clinical data to more accurately track national sepsis trends and enable objective assessment of the impact of efforts to increase sepsis awareness and prevention.

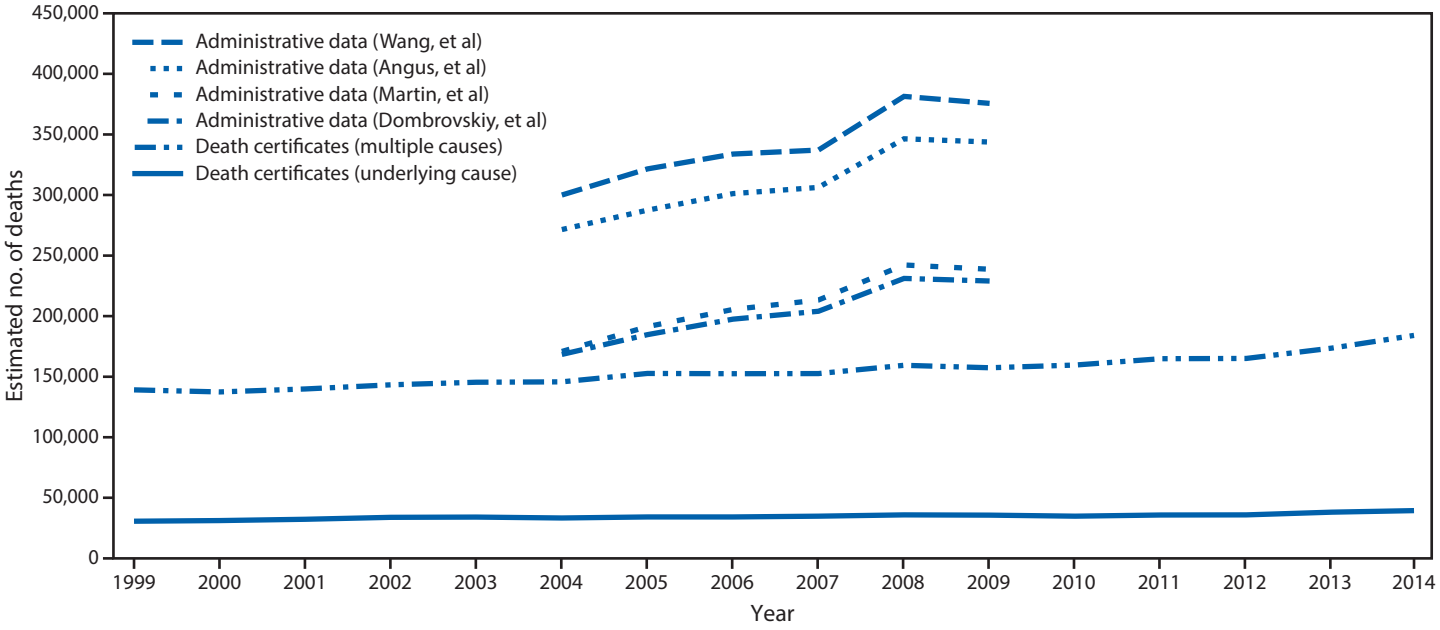
Death certificate data were obtained from multiple cause-of-death records of the National Vital Statistics System, using CDC's WONDER database (http://wonder.cdc.gov/mcd.html). Multiple cause-of-death records include the immediate cause of death (i.e., the final disease or condition resulting in death), up to 20 contributing causes, a single underlying cause of death (i.e., the disease or injury that initiated the events resulting in death), and significant conditions that were present at the time of death but were not a direct link in the chain of events leading to death. All information on death certificates is documented by the certifier (e.g., a physician, medical examiner, or coroner) and subsequently coded by the National Center for Health Statistics in accordance with guidelines specified by the World Health Organization (*3*). National trends in sepsis-related mortality have been previously estimated from administrative claims data obtained from the Nationwide Inpatient Sample, the largest all-payer, publicly-available inpatient database in the United States, using various combinations of the *International Classification of Diseases 9th Revision Clinical Modification* (ICD-9-CM) administrative codes for primary or secondary infection and organ dysfunction to identify severe sepsis.

In a published report (*2*), investigators generated a range of sepsis mortality estimates using four previously established approaches to identifying adult patients (aged ≥18 years) with sepsis using administrative claims data. Two of these approaches (*4*,*5*) defined sepsis using explicit, sepsis-specific ICD-9-CM codes in addition to various codes for infection and organ dysfunction, whereas the other two approaches (*6*,*7*) defined sepsis on the basis of combinations of infection criteria and organ dysfunction as implicit markers for severe sepsis.

Multiple cause-of-death mortality files maintained in the CDC WONDER database were reviewed to analyze deaths (for all ages) from sepsis reported on death certificates during 1999–2014, defined as deaths with diagnoses corresponding to *International Classification of Diseases 10th Revision* (ICD-10) diagnosis codes A40 (streptococcal septicemia) and A41 (other septicemia) listed on the death certificate. The annual sepsis mortality estimates based on death certificates from the WONDER database were then compared with the previously published annual estimates generated based on the ICD-9-CM administrative codes data from the Nationwide Inpatient Sample.

Based on multiple cause-of-death data during 1999–2014, a total of 2,470,666 decedents (6% of all deaths) had sepsis listed among the causes of death (sepsis-related deaths); for 22% of these decedents, sepsis was listed as the underlying cause of death. During this period, the annual number of all reported sepsis-related deaths increased 31%, from 139,086 in 1999 to 182,242 in 2014 (Figure 1). Approximately 15% of all sepsis-related deaths during this period occurred in nonacute care settings (e.g., at home, long-term care facilities, hospice, and unknown setting). Among the 2,472,911 A40 and A41 codes listed for the 2,470,666 decedents, the most common were unspecified septicemia (A41.9, 94%), septicemia caused by other gram-negative organisms (A41.5, 2%), and septicemia caused by *Staphylococcus aureus* (A41.0, 2%). Among decedents, approximately 49% were aged 65–84 years, 26% were aged ≥85 years, and 25% were aged 25–64 years (4% aged

FIGURE 1. Comparison of sepsis-related mortality estimates based on death certificates* (1999–2014) with four estimates from administrative claims data† (2004–2009) — United States



* Estimates based on multiple causes of death information include the immediate cause of death, antecedent causes, the underlying cause of death, and significant conditions that were present at the time of death but were not a direct link in the chain of events leading to death (all ages included). Additional information available in National Center for Health Statistics (NCHS). National Vital Statistics System: instructions for classifying the underlying cause of death. NCHS instruction manual; part 2a. US Department of Health and Human Services, CDC, National Center for Health Statistics, Hyattsville, MD. http://www.cdc.gov/nchs/nvss/instruction_manuals.htm.

† Four approaches for identifying severe sepsis among adults (aged ≥18 years) using different combinations of *International Classification of Diseases 9th Revision Clinical Modification* administrative codes for infection (either primary or secondary) and organ dysfunction were applied to the Nationwide Inpatient Sample to generate a range of sepsis-related mortality estimates for the period 2004–2009. Additional information available in the following:

Gaieski DF, Edwards M, Kallan MJ, Carr BG. Benchmarking the incidence and mortality of severe sepsis in the United States. Crit Care Med 2013;41:1167–74.

Wang HE, Shapiro NI, Angus DC, Yealy DM. National estimates of severe sepsis in United States emergency departments. Crit Care Med 2007;35:1928–36.

Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome, and associated costs of care. Crit Care Med 2001;29:1303–10.

Martin GS, Mannino DM, Eaton S, Moss M. The epidemiology of sepsis in the United States from 1979 through 2000. N Engl J Med 2003;348:1546–54.

Dombrovskiy VY, Martin AA, Sunderram J, Paz HL. Rapid increase in hospitalization and mortality rates for severe sepsis in the United States: a trend analysis from 1993 to 2003. Crit Care Med 2007;35:1244–50.

25–44 years and 21% aged 45–64 years). Approximately 1% of decedents were aged <25 years. (Figure 2).

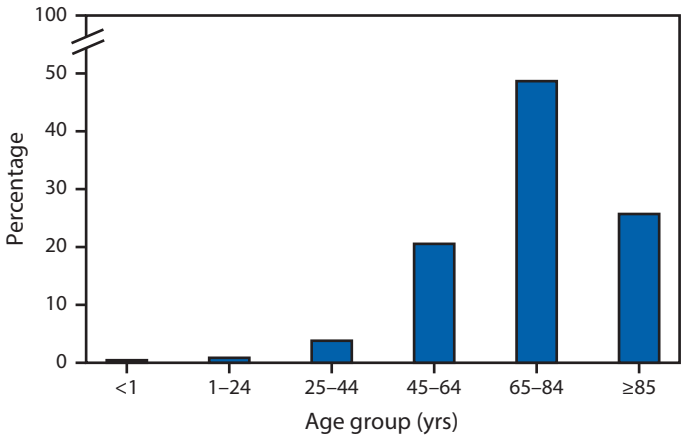
During 2004–2009, using data rounded to thousands, the annual range of published sepsis-related mortality estimates based on administrative codes (range = 168,000–381,000) was 15% to 140% higher than annual estimates generated using death certificates (multiple causes) for those years (range = 146,000–159,000) (Figure 1).

Discussion

Death records and administrative claims data are important sources of public health information. However, they provide different estimates of sepsis mortality, and recognizing the limitations of both methods for generating sepsis-related mortality estimates is important.

Death certificate certifiers are required to indicate as the underlying cause of the death a specific disease or injury that initiated the chain of events leading directly to death (*3*). Because sepsis is the final common pathway for many different severe infections, certifiers might be more likely to consider sepsis an immediate, rather than the underlying, cause of death. This practice might result in an underestimation of the importance of sepsis as a cause of death and lower estimates of sepsis-related mortality that are based on the underlying causes of death only (*8*). Furthermore, because of misunderstandings regarding the process of completing death certificates, certifiers might omit sepsis entirely as a cause of death on the death certificate. For example, the New York City medical examiner's policy of rejecting New York City death certificates with sepsis listed alone as the single underlying cause of death (without other contributing causes) has been misinterpreted by some clinicians to mean that the term "sepsis" was not permitted to be included anywhere on the death certificate, thus affecting estimates of sepsis-related mortality (*8*). In addition, it is important to note that death certificates are often completed by physicians with varying levels of training in completing

FIGURE 2. Percentage of sepsis-related deaths (N = 2,470,666) based on death certificate data, by age groups* — United States, 1999–2014



* Age was unknown for 90 decedents.

death certificates; data from a survey of resident physicians in New York City showed that only 40% reported receiving any training by their residency program regarding completion of death certificates (*9*). Finally, sepsis can be confused with other acute medical problems, such as myocardial infarction, that frequently cause mortality in the community setting.

Estimates based on administrative claims data also have limitations. Previous analyses of administrative claims data have shown that estimates of sepsis incidence have increased over time, whereas diseases and conditions that can cause sepsis (e.g., pneumonia or urinary tract infections) have decreased or remained stable (*10*). Increased reimbursement for sepsis (compared with other infectious disease diagnoses) encourages hospitals to be more careful about coding for sepsis. Improved recognition and awareness of sepsis have also likely contributed to increased documentation of sepsis over time (*10*). Finally, administrative data only capture sepsis-related deaths that occur within health care facilities. However, approximately 15% of sepsis-related deaths recorded in death certificate data occurred outside of health care facilities, suggesting sepsis mortality estimates based on administrative data might not include these deaths.

Sepsis is a complex clinical syndrome, representing a response to infection that can arise from many underlying causes. Differences in sepsis-related mortality estimates derived from death certificates and administrative claims data might be explained by limitations inherent in each data source. Current efforts focused on evaluating linkages of administrative claims data derived from inpatient medical records with death certificates will improve understanding of how mortality estimates from death certificates and administrative claims data should be interpreted. Strategies to prevent infections that lead to sepsis include vaccination for pathogens like pneumococcus, influenza, and *Neisseria meningitidis*, smoking cessation programs to reduce the risk for community-acquired pneumonia, and facility-level interventions to reduce risk for health care–associated infections. A reliable sepsis surveillance definition based on objective clinical data is needed to more accurately track national sepsis trends and enable ongoing assessment of the impact of efforts to increase sepsis awareness and prevention.

Summary

What is already known about this topic?

Sepsis is a clinical syndrome caused by response to infection. Because there is no confirmatory diagnostic test, the diagnosis of sepsis is based on clinical judgement of suspected infection. Data from both death certificate and administrative claims data have been used to assess sepsis incidence and mortality, but estimates vary depending on the surveillance definition and data source.

What is added by this report?

To highlight the challenges and variability associated with estimating sepsis mortality, CDC compared national estimates of sepsis-related mortality based on death certificates with previously published sepsis mortality estimates generated using administrative claims data. Using death certificate data for the period 1999–2014, CDC found that a total of 2,470,666 decedents (6% of all deaths) had sepsis listed among the causes of death (sepsis-related deaths); for 22% of these decedents, sepsis was listed as the underlying cause of death. For the period 2004–2009, in a previously published report, investigators analyzed administrative claims data using four approaches for identifying adult patients (aged ≥18 years) with sepsis. In data rounded to thousands, the annual range of published sepsis-related mortality estimates based on administrative claims data was 15% to 140% higher (range = 168,000–381,000) than annual estimates generated using death certificate data (multiple causes) (range = 146,000–159,000).

What are the implications for public health practice?

Sepsis is a complex clinical syndrome, representing a response to infection that can arise from many different underlying causes. A reliable sepsis surveillance definition based on objective clinical data is needed to more accurately track national sepsis trends and enable ongoing assessment of the impact of efforts to increase sepsis awareness and prevention.

Acknowledgment

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References

1. Singer M, Deutschman CS, Seymour CW, et al. The third international consensus definitions for sepsis and septic shock (sepsis-3). JAMA 2016;315:801–10. http://dx.doi.org/10.1001/jama.2016.0287
2. Gaieski DF, Edwards JM, Kallan MJ, Carr BG. Benchmarking the incidence and mortality of severe sepsis in the United States. Crit Care Med 2013;41:1167–74. http://dx.doi.org/10.1097/CCM.0b013e31827c09f8
3. National Center for Health Statistics (NCHS). National Vital Statistics System: instructions for classifying the underlying cause of death. NCHS instruction manual; part 2a. Hyattsville, MD: US Department of Health and Human Services, CDC, National Center for Health Statistics; 2016. http://www.cdc.gov/nchs/nvss/instruction_manuals.htm
4. Wang HE, Shapiro NI, Angus DC, Yealy DM. National estimates of severe sepsis in United States emergency departments. Crit Care Med 2007;35:1928–36. http://dx.doi.org/10.1097/01.CCM.0000277043.85378.C1
5. Dombrovskiy VY, Martin AA, Sunderram J, Paz HL. Rapid increase in hospitalization and mortality rates for severe sepsis in the United States: a trend analysis from 1993 to 2003. Crit Care Med 2007;35:1244–50. http://dx.doi.org/10.1097/01.CCM.0000261890.41311.E9
6. Martin GS, Mannino DM, Eaton S, Moss M. The epidemiology of sepsis in the United States from 1979 through 2000. N Engl J Med 2003;348:1546–54. http://dx.doi.org/10.1056/NEJMoa022139
7. Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome, and associated costs of care. Crit Care Med 2001;29:1303–10. http://dx.doi.org/10.1097/00003246-200107000-00002
8. Ong P, Gambatese M, Begier E, Zimmerman R, Soto A, Madsen A. Effect of cause-of-death training on agreement between hospital discharge diagnoses and cause of death reported, inpatient hospital deaths, New York City, 2008–2010. Prev Chronic Dis 2015;12:140299. http://dx.doi.org/10.5888/pcd12.140299
9. Wexelman BA, Eden E, Rose KM. Survey of New York City resident physicians on cause-of-death reporting, 2010. Prev Chronic Dis 2013;10:120288. http://dx.doi.org/10.5888/pcd10.120288
10. Rhee C, Gohil S, Klompas M. Regulatory mandates for sepsis care—reasons for caution. N Engl J Med 2014;370:1673–6. http://dx.doi.org/10.1056/NEJMp1400276

Surveillance Systems to Track Progress Toward Polio Eradication — Worldwide, 2014–2015

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Global efforts to eradicate polio began in 1988, and polio-free certification has been achieved in four of the six World Health Organization (WHO) regions. Nigeria was removed from WHO's list of countries with endemic polio in September 2015, achieving an important milestone toward interruption of wild poliovirus (WPV) transmission in the African Region (*1*). Afghanistan and Pakistan, both in the Eastern Mediterranean Region, were the only countries to report WPV cases in 2015. Previously reported outbreaks caused by WPV importation during 2013–2014 have ended (*2*,*3*). The primary means for detecting poliovirus transmission is surveillance for acute flaccid paralysis (AFP) among children aged <15 years (*4*,*5*). Stool specimens collected from children with AFP are tested for both WPV and vaccine-derived poliovirus (VDPV) in WHO–accredited laboratories within the Global Polio Laboratory Network (GPLN). In selected locations, AFP surveillance is supplemented with environmental surveillance (testing sewage for poliovirus) (*6*). Testing of stool and sewage samples includes genomic sequencing to characterize poliovirus isolates; results are used to map poliovirus transmission and identify gaps in AFP surveillance. This report presents poliovirus surveillance data from 2014 and 2015, focusing on the 20 countries in the African Region and six in the Eastern Mediterranean Region that reported a WPV or circulating VDPV (cVDPV) case during 2011–2015, including Guinea, Liberia, and Sierra Leone, which were most affected by the 2014–2015 Ebola virus disease (Ebola) outbreak.

During 2015, 10 (50%) of 20 African Region countries and all six Eastern Mediterranean Region countries met both national AFP surveillance quality indicators.* To complete and certify polio eradication, surveillance gaps must be identified and surveillance activities further strengthened, including supervision, monitoring, and specimen collection and handling.

Acute Flaccid Paralysis Surveillance

In the African Region, the number of reported AFP cases increased from 22,447 in 2014 to 26,238 in 2015. The number of WPV type 1 (WPV1) cases identified decreased from 17 in four countries in 2014 to zero in 2015. Date of onset of the last WPV1 case was July 24, 2014, in Nigeria. During 2014, a total of 34 cVDPV cases (33 cVDPV type 2 [cVDPV2] and one cVDPV type 1 [cVDPV1]) were identified in four countries. During 2015, a total of 18 cVDPV cases (eight cVDPV2 and 10 cVDPV1) were identified in three countries (Table 1).

In the Eastern Mediterranean Region, 12,546 AFP cases were reported in 2014 and 13,171 in 2015. The number of WPV1 cases identified declined from 342 in five countries in 2014 to 74 in the two countries with endemic polio in 2015 (20 in Afghanistan and 54 in Pakistan). During both 2014 and 2015, most of the WPV1 cases (89% and 73%, respectively) and all cVDPV2 cases in the region were identified in Pakistan (Table 1).

The quality of AFP surveillance is measured by two principal indicators. The first is the nonpolio AFP rate, (i.e., the number of nonpolio AFP cases per 100,000 children aged <15 years per year). A nonpolio AFP rate ≥2 is considered sufficiently sensitive to identify WPV or cVDPV cases if poliovirus is circulating (*5*). The second indicator is the collection of adequate stool specimens from ≥80% of AFP cases, indicating surveillance can effectively identify WPV and VDPV among individuals with AFP (*5*). Adequacy is defined as the collection of two stool specimens ≥24 hours apart, within 14 days of paralysis onset, and arrival at a WHO–accredited laboratory in "good" condition.†

Among the 20 countries evaluated in the African region, 13 (65%) and 10 (50%) met both national indicators in 2014 and 2015, respectively. Among the three countries affected by the Ebola outbreak, Guinea and Liberia did not meet one of the national indicators and Sierra Leone did not meet either indicator in 2015. All six Eastern Mediterranean Region countries examined met both indicators in 2014 and 2015 (Table 1). However, national-level surveillance indicators masked differences in surveillance performance at subnational levels (Figure).

Environmental Surveillance

Testing of sewage samples supplements AFP surveillance by identifying poliovirus transmission that might occur in the absence of detected AFP cases (*6*). Environmental

* National nonpolio AFP rates of ≥2 per 100,000 persons aged <15 years per year and ≥80% of AFP cases with adequate stool specimens.

† Reverse cold chain maintained and received without leakage or desiccation at a WHO–accredited laboratory. Reverse cold chain is maintained when stool specimens are stored immediately after collection at 4–8°C (32–39°F), frozen at -20°C (-4°F) when received for processing, and shipped to a WHO–accredited laboratory in dry ice or cold packs. Freezing of specimens is unnecessary if specimens can be received at a WHO–accredited laboratory within 72 hours of collection.

TABLE 1. National and subnational acute flaccid paralysis (AFP) surveillance indicators and number of confirmed wild poliovirus (WPV) and circulating vaccine-derived poliovirus (cVDPV) cases, by country, for all countries that had poliovirus transmission during 2011–2015 or were affected by the Ebola outbreak in West Africa within the World Health Organization (WHO) African Region and Eastern Mediterranean Region, 2014 and 2015*

WHO region/country	AFP cases	Regional/ national NPAFP rate†	Subnational areas with NPAFP rate ≥2§ (%)	Regional/ national AFP cases with adequate specimens¶ (%)	Subnational areas with ≥80% adequate specimens (%)	Population in areas meeting both indicators** (%)	Confirmed WPV cases*	Confirmed cVDPV cases*,††
2014								
AFR	22,447	5.4	NA	89	NA	NA	17	34
Angola	321	3.0	100	93	94	97	—	—
Cameroon	846	7.5	100	71	20	25	5	—
CAR§§	89	4.3	71	76	57	37	—	—
Chad	393	6.0	94	86	72	72	—	—
Côte d'Ivoire	394	4.6	89	86	61	74	—	—
DRC	1,831	5.4	100	82	82	76	—	—
Equatorial Guinea	32	8.0	86	16	0	0	5	—
Ethiopia§§	1,198	2.9	82	76	27	27	1	—
Gabon	42	4.9	70	29	10	0	—	—
Guinea	146	2.9	75	88	88	53	—	1
Kenya	723	3.7	100	88	100	100	—	—
Liberia	23	1.2	60	96	87	31	—	—
Madagascar	421	4.2	82	85	55	63	—	1
Mali	236	3.2	89	89	67	91	—	—
Mozambique	317	2.5	90	88	80	85	—	—
Niger§§	249	2.5	86	71	14	14	—	—
Nigeria	10,506	13.1	100	97	100	100	6	30
Republic of the Congo	114	5.1	100	87	73	73	—	—
Sierra Leone	72	2.8	75	96	100	79	—	—
South Sudan	321	6.4	70	89	80	64	—	2
EMR	12,546	6.0	NA	91	NA	NA	342	22
Afghanistan	2,421	16.7	100	92	97	99	28	—
Iraq	590	4.3	84	89	79	70	2	—
Pakistan	5,369	8.1	88	88	100	99	306	22
Somalia	420	8.1	100	97	95	99	5	—
Syrian Arab Republic¶¶	306	4.0	93	82	71	58	1	—
Yemen	578	5.8	100	95	100	100	—	—

See table footnotes on next page.

surveillance collection sites increased within Afghanistan, Nigeria, and Pakistan from 21 at the end of 2011 to 83 as of March 2015. Overall, environmental surveillance is conducted in 34 countries without recent active WPV transmission, including nine on the African continent.

In Nigeria, sampling is currently conducted at 43 sites in 10 states and the Federal Capital Territory. No WPV has been isolated since May 2014 when WPV1 was isolated from one sample in Kaduna. Continued transmission of cVDPV2 that emerged in Nigeria in 2005 and of cVDPV2 imported from Chad in 2013 was documented from samples collected in six states in 2014 and only Kaduna in 2015. Environmental sampling in Afghanistan is conducted at 14 sites in five WPV high risk provinces. WPV1 was detected in samples collected in Helmand, Kandahar, and Nangarhar in 2014 and in all five provinces in 2015. In Pakistan, sampling is conducted at 40 sites in five provinces/regions. The proportion of samples positive for WPV1 decreased from 35% in 2014 to 20% in 2015. WPV1 was detected in all five provinces/regions in both years.

Global Polio Laboratory Network

The GPLN comprises 146 WHO–accredited poliovirus laboratories in all WHO regions. GPLN member laboratories follow standardized protocols to 1) isolate and identify poliovirus, 2) conduct intratypic differentiation to identify WPV or screen for Sabin-like poliovirus (isolates that display ≤1% nucleotide sequence difference from the parental vaccine strain [≤0.6% for type 2]) and VDPV (*7*), and 3) conduct genomic sequencing. Sequencing results are used to monitor pathways of poliovirus transmission by comparing the nucleotide sequence of the coding region for one of the viral capsid proteins (VP1) of poliovirus isolates. Genomic sequencing of an isolate with ≥1.5% nucleotide divergence in the VP1-coding region from previously identified poliovirus isolates (i.e., an "orphan" virus), indicates prolonged undetected circulation and gaps in AFP surveillance.

TABLE 1. (*Continued*) National and subnational acute flaccid paralysis (AFP) surveillance indicators and number of confirmed wild poliovirus (WPV) and circulating vaccine-derived poliovirus (cVDPV) cases, by country, for all countries that had poliovirus transmission during 2011–2015 or were affected by the Ebola outbreak in West Africa within the World Health Organization (WHO) African Region and Eastern Mediterranean Region, 2014 and 2015*

WHO region/country	AFP cases	Regional/ national NPAFP rate†	Subnational areas with NPAFP rate ≥2§ (%)	Regional/ national AFP cases with adequate specimens¶ (%)	Subnational areas with ≥80% adequate specimens (%)	Population in areas meeting both indicators** (%)	Confirmed WPV cases*	Confirmed cVDPV cases*,††
2015								
AFR	26,238	6.2	NA	90	NA	NA	0	18
Angola	458	4.3	100	96	100	100	—	—
Cameroon	618	5.6	100	83	80	67	—	—
CAR	81	3.9	71	80	43	34	—	—
Chad	435	6.6	100	87	78	87	—	—
Côte d'Ivoire	353	4.0	94	90	89	94	—	—
DRC§§	2,089	5.9	100	74	9	6	—	—
Equatorial Guinea	11	3.6	43	36	0	0	—	—
Ethiopia§§	1,179	2.8	82	76	45	29	—	—
Gabon§§	61	8.9	100	33	0	0	—	—
Guinea§§	143	2.7	63	77	38	26	—	7
Kenya	624	3.2	100	85	75	83	—	—
Liberia	22	1.2	60	95	60	44	—	—
Madagascar	692	6.4	100	60	14	23	—	10
Mali	247	3.2	78	84	67	79	—	—
Mozambique	323	2.6	100	80	60	57	—	—
Niger§§	222	2.3	71	59	0	0	—	—
Nigeria	13,960	17.1	100	98	100	100	—	1
Republic of the Congo§§	117	5.3	100	78	45	29	—	—
Sierra Leone	38	1.4	25	79	25	23	—	—
South Sudan	329	6.5	100	94	90	90	—	—
EMR	13,171	6.4	NA	90	NA	NA	74	2
Afghanistan	2,738	18.9	100	93	94	94	20	—
Iraq	520	3.7	84	82	58	49	—	—
Pakistan	5,770	9.2	100	88	88	99	54	2
Somalia	281	5.4	100	96	100	100	—	—
Syrian Arab Republic¶¶	235	3.0	57	86	71	43	—	—
Yemen	539	5.4	100	91	91	95	—	—

Abbreviations: AFR = African Region; CAR = Central African Republic; DRC = Democratic Republic of the Congo; Ebola = Ebola virus disease; EMR = Eastern Mediterranean Region; NA = not available; NPAFP = nonpolio AFP.

* Data as of February 22, 2016.

† Per 100,000 persons aged <15 years per year.

§ For all subnational areas regardless of population size.

¶ Standard WHO target is adequate stool specimen collection from ≥80% of AFP cases, assessed by timeliness and condition. In this analysis, timeliness was defined as two specimens collected ≥24 hours apart (≥1 calendar day in this data set), and both within 14 days of paralysis onset. Condition was defined as specimens arriving in good condition (reverse cold chain maintained and received without leakage or desiccation) in a WHO–accredited laboratory.

** For all subnational areas regardless of population size. The two indicators are NPAFP rate of ≥2 per 100,000 persons aged <15 years per year and ≥80% of AFP cases with adequate stool specimens.

†† cVDPV was associated with two or more cases of AFP with genetically linked VDPVs. Guidelines for classification of cVDPV changed in 2015 and can be found at http://www.polioeradication.org/Portals/0/Document/Resources/VDPV_ReportingClassification.pdf.

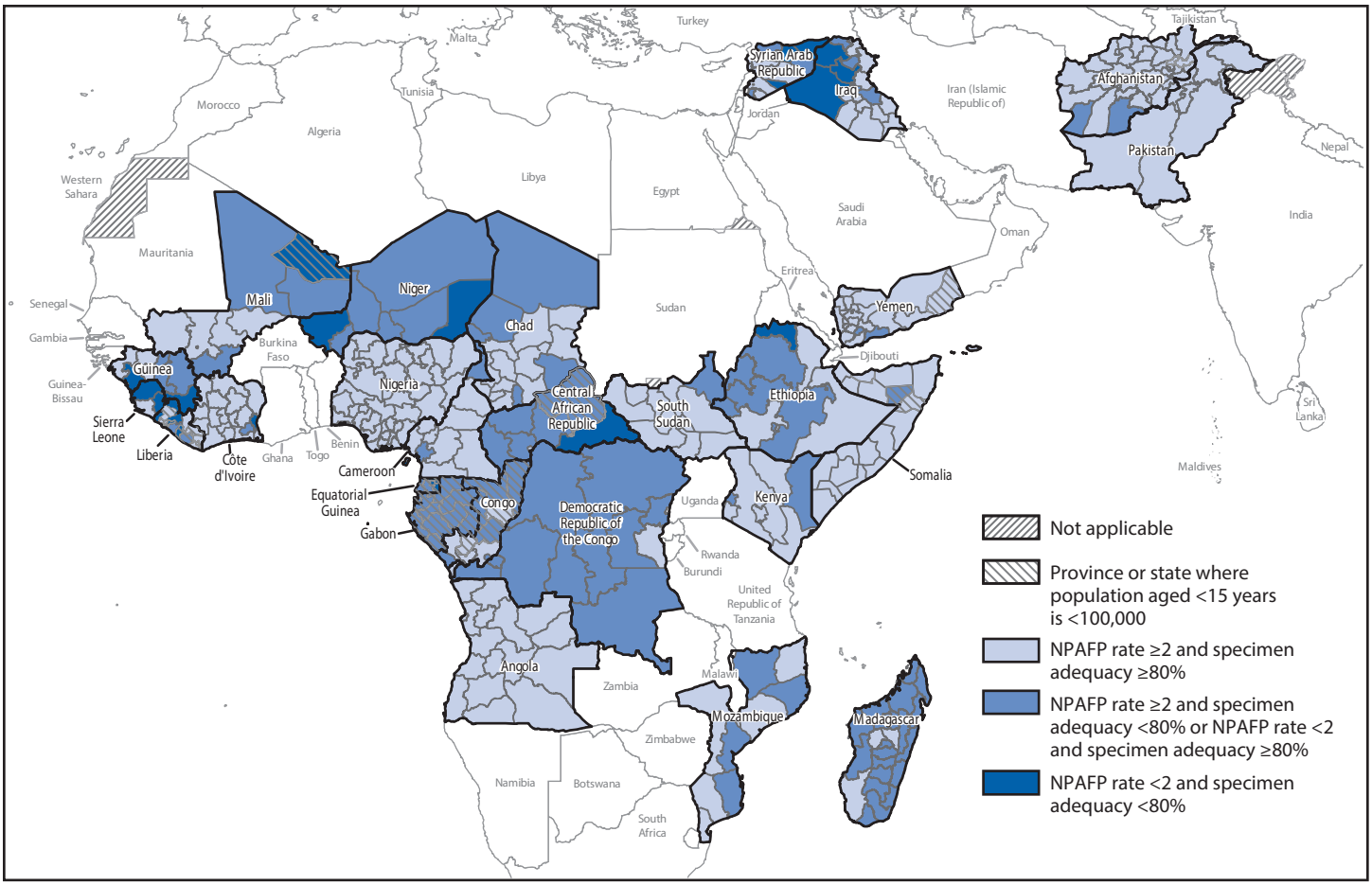
§§ Stool specimen adequacy dropped to <80% when stool condition was included with timeliness. Timeliness was defined as two specimens collected ≥24 hours apart (≥1 calendar day in this dataset), and both within 14 days of paralysis onset. Condition was defined as specimens arriving in good condition (reverse cold chain maintained and received without leakage or desiccation) in a WHO-accredited laboratory.

¶¶ The NPAFP rate for Syria is artificially low because of displaced populations and the lack of official data from areas not under government control.

To meet standard laboratory timeliness indicators for stool specimen processing, laboratories should report ≥80% of poliovirus isolation results within 14 days of specimen receipt, ≥80% of intratypic differentiation results within 7 days of isolate receipt, and ≥80% of sequencing results within 7 days of identifying isolate intratype. The standard programmatic indicator combining field and laboratory performance is to report intratypic differentiation results for ≥80% of isolates within 60 days of paralysis onset for AFP cases. This indicator takes into account the interval from paralysis onset to specimen testing (the Eastern Mediterranean Region uses a 45-day time frame). The accuracy and quality of testing at GPLN laboratories is monitored through an annual accreditation program of onsite reviews and proficiency testing.

GPLN laboratories met timeliness indicators for poliovirus isolation in all regions for both years except the Western Pacific

FIGURE. Combined performance indicators for the quality of acute flaccid paralysis (AFP) surveillance* in subnational areas (states and provinces) of 26 countries that had poliovirus transmission during 2011–2015 or were affected by the Ebola outbreak in West Africa during 2014–2015 — World Health Organization African and Eastern Mediterranean Regions, 2015†



Abbreviations: Ebola = Ebola virus disease; NPAFP = nonpolio acute flaccid paralysis.

* The Global Polio Eradication Initiative has set the following targets for countries with current or recent wild poliovirus transmission and their states/provinces: 1) NPAFP rate of ≥2 cases per 100,000 persons aged <15 years per year, and 2) adequate stool specimen collection from ≥80% of AFP cases, with specimen adequacy assessed by timeliness and condition. Timeliness was defined as two specimens collected ≥24 hours apart (≥1 calendar day in this dataset) and both within 14 days of paralysis onset. Condition was defined as specimens arriving in good condition (reverse cold chain maintained and received without leakage or desiccation) at a World Health Organization–accredited laboratory.

† Data are for AFP cases with onset during 2015, reported as of February 22, 2016.

Region in 2014 and the European Region in 2015 (Table 2). The overall timeliness indicator for onset to intratypic differentiation results was met in all regions in both years except the European Region in 2015. As of March 5, 2016, the GPLN had tested 203,698 stool specimens in 2014 and 192,250 in 2015. WPV1 was isolated from 412 AFP case samples in 2014 and from 74 in 2015. In addition, cVDPV was detected from 80 AFP case samples in 2014 and 32 in 2015.

For the first time since 2005, the majority of cVDPV cases detected globally in 2015 were caused by Type 1. Among the 31 cVDPV cases identified, 19 (61%) occurred as part of type 1 outbreaks in Laos (7), Madagascar (10) and Ukraine (2); the remaining cVDPV cases were type 2 (Guinea [7], Myanmar [2], Nigeria [1], and Pakistan [2]).

Genetic diversity declined among WPV1 isolates in 2015. In 2014, West Africa B1 (WEAF-B1) and South Asia (SOAS) were the only WPV1 genotypes circulating globally. Although WEAF-B1 genotype was detected in five countries in 2014, the only genotype detected in 2015 was SOAS from Afghanistan and Pakistan.

Sequence analysis continues to indicate that, as in 2014, WPV1 and cVDPV cases were likely missed by AFP surveillance in 2015. Orphan WPV1 isolates were associated with six of 54 WPV1 cases reported from Pakistan and two of 20 WPV1 cases reported in Afghanistan. Orphan cVDPV viruses were also isolated from stool specimens of AFP cases in Guinea, Laos, Madagascar, and Ukraine.

TABLE 2. Number of poliovirus (PV) isolates from stool specimens of persons with acute flaccid paralysis and timing of results, by World Health Organization (WHO) region, 2014* and 2015*

WHO Region	No. of specimens	No. of poliovirus isolates			(%) poliovirus isolation results on time¶	(%) ITD results within 7 days**	(%) ITD results within 60 days††
		Wild	Sabin†	cVDPV§			
2014							
African	45,856	83	4,038	37	92	86	92
Americas	1,675	—	39	—	83	100	94
Eastern Mediterranean	23,552	329	809	27	98	95	97
European	3,224	—	26	2	99	NA	82
South-East Asia	115,539	—	2,785	3	97	90	98
Western Pacific	13,852	—	352	11	78	96	81
Total§§	**203,698**	**412**	**8,049**	**80**	**91**	**93**	**91**
2015							
African	50,960	—	3,579	17	82	79	95
Americas	1,698	—	44	—	84	100	100
Eastern Mediterranean	25,827	74	951	2	93	99	95
European	3,655	—	106	4	63	93	70
South-East Asia	96,783	—	3,335	2	97	86	98
Western Pacific	13,327	—	194	7	96	98	86
Total§§	**192,250**	**74**	**8,209**	**32**	**86**	**92**	**91**

Abbreviations: cVDPV = circulating vaccine-derived poliovirus; ITD = intratypic differentiation; NA = not available.

* Data as of April 1, 2015 and March 5, 2016, respectively.

† Either concordant Sabin-like results in ITD test and VDPV screening, or ≤1% VP1 nucleotide sequence difference compared with Sabin vaccine virus (≤0.6% for type 2).

§ For PV types 1 and 3, 10 or more VP1 nucleotide differences from the respective PV; for PV type 2, six or more VP1 nucleotide differences from Sabin type 2 PV.

¶ Results reported within 14 days for laboratories in the following WHO regions: African, Americas, Eastern Mediterranean, South-East Asia, and Western Pacific. Results reported within 28 days for the European Region.

** Results of ITD reported within 7 days of receipt of specimen. As EURO performance might be underestimated because of data entry issues, it has been excluded from analysis.

†† Results reported within 60 days of paralysis onset for all WHO regions except Eastern Mediterranean Region, which reported within 45 days of paralysis onset.

§§ For the last two indicators, total represents mean (in %) of regions' performance.

Discussion

No WPV transmission was identified on the African continent in 2015. Certification of polio-free status requires at least 3 years of timely and sensitive surveillance (*8*). However, AFP surveillance indicators were not met in half of the 20 African Region countries examined. Rapid strengthening of AFP surveillance is critical to ensure timely polio-free certification of the region. This includes improving timeliness for stool collection and ensuring appropriate transport of specimens. Specimen condition was a particular concern for the Democratic Republic of the Congo, Equatorial Guinea, Ethiopia, Gabon, Niger, and the Republic of the Congo and was a factor in six countries that did not attain ≥80% specimen adequacy in 2015. Urgent efforts are also needed to improve AFP surveillance in Guinea, Liberia, and Sierra Leone, where health systems were severely disrupted by the Ebola outbreak (*9*). During 2013–2015, nonpolio AFP rates and stool specimen adequacy (i.e., timeliness and condition) declined in all three countries (*10*). Furthermore, specimen shipping and testing was suspended during portions of 2014–2015. Efforts are also needed to improve immunization services in these three countries. Since 2014, eight cVDPV2 cases have been detected in Guinea; cVDPV emergence is the result of low population immunity to poliovirus. The WHO African Regional Office is collaborating with eight high-risk countries on an initiative to strengthen AFP surveillance.

The national AFP surveillance quality indicators continued to be met in all six Eastern Mediterranean Region countries, including Afghanistan and Pakistan in 2015, where 94% and 99% of the population aged <15 years, respectively, lived in areas meeting both indicators. Nonetheless, detection of orphan viruses and environmental surveillance findings indicate continued gaps in AFP surveillance in both countries.

The findings in this report are subject to at least two limitations. First, the surveillance indicators do not fully reflect security-related issues, issues associated with mobile and difficult to access populations, or other factors that affect surveillance performance. For example, in Iraq and the Syrian Arab Republic, population movements related to conflict make interpretation of AFP surveillance indicators difficult. Second, high nonpolio AFP rates do not necessarily imply sensitive surveillance, because a proportion of reported AFP cases might not be true AFP cases and not all true AFP cases might be reported. Because AFP cases in this report are considered true AFP cases, findings are presumed to accurately depict surveillance activities in each country. Supervision and monitoring of AFP surveillance can help ensure that all true AFP cases are identified, reported, and investigated appropriately.

As the number of reported polio cases declines, sensitive AFP surveillance becomes increasingly critical, and environmental surveillance will continue to be an important supplement to AFP surveillance. The risk for WPV and cVDPV importation, and cVDPV emergence exists even in countries within polio-free regions. To promptly identify and respond to all cases of polio, surveillance performance must be continuously assessed and quality must be maintained globally.

Acknowledgments

Situational Awareness Branch, Division of Emergency Operations, CDC; Qi Chen, Beth Henderson, Jan Iber, Division of Viral Diseases, CDC; Paul Chenoweth, Ajay Goel, Polio Eradication Department, World Health Organization (WHO); Humayun Asghar, Evgeniy Gavrilin, Varja Grabovac, Nicksy Gumede-Moeletsi, Sirima Pattamadilok, Fem Paladin, Gloria Rey-Benito, WHO GPLN laboratories, WHO Global Polio Laboratory Network.

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References

1. World Health Organization. WHO removes Nigeria from polio-endemic list. Geneva, Switzerland: World Health Organization; 2015. http://www.who.int/mediacentre/news/releases/2015/nigeria-polio/en/
2. Global Polio Eradication Initiative. Key countries. Geneva, Switzerland: Global Polio Eradication Initiative, World Health Organization; 2016. http://www.polioeradication.org/Keycountries.aspx
3. Moturi EK, Porter KA, Wassilak SGF, et al. Progress toward polio eradication—worldwide, 2013–2014. MMWR Morb Mortal Wkly Rep 2014;63:468–72.
4. Levitt A, Diop OM, Tangermann RH, et al. Surveillance systems to track progress toward global polio eradication—worldwide, 2012–2013. MMWR Morb Mortal Wkly Rep 2014;63:356–61.
5. World Health Organization. WHO-recommended surveillance standard of poliomyelitis. Geneva, Switzerland: World Health Organization; 2015. http://www.who.int/immunization/monitoring_surveillance/burden/vpd/surveillance_type/active/poliomyelitis_standards/en/
6. Asghar H, Diop OM, Weldegebriel G, et al. Environmental surveillance for polioviruses in the Global Polio Eradication Initiative. J Infect Dis 2014;210:S294–303. http://dx.doi.org/10.1093/infdis/jiu384
7. Kilpatrick DR, Yang CF, Ching K, et al. Rapid group-, serotype-, and vaccine strain-specific identification of poliovirus isolates by real-time reverse transcription-PCR using degenerate primers and probes containing deoxyinosine residues. J Clin Microbiol 2009;47:1939–41. http://dx.doi.org/10.1128/JCM.00702-09
8. World Health Organization. Report of the 1st meeting of the global commission for the certification of the eradication of poliomyelitis. Geneva, Switzerland: World Health Organization; 1995. http://www.polioeradication.org/Portals/0/Document/Resources/1st%20Global%20CertCom%20Meeting%20Report%201996.PDF
9. Kieny MP, Dovlo D. Beyond ebola: a new agenda for resilient health systems. Lancet 2015;385:91–2. http://dx.doi.org/10.1016/S0140-6736(14)62479-X
10. Porter KA, Diop OM, Burns CC, Tangermann RH, Wassilak SG. Tracking progress toward polio eradication—worldwide, 2013–2014. MMWR Morb Mortal Wkly Rep 2015;64:415–20.

Summary

What is already known about this topic?

Surveillance is a cornerstone of polio eradication efforts. Acute flaccid paralysis (AFP) surveillance is the primary means of poliovirus detection, supplemented in selected countries by environmental surveillance. The Global Polio Laboratory Network facilitates laboratory identification of polioviruses and genomic analysis to track poliovirus spread.

What is added by this report?

There was no evidence of wild poliovirus circulation on the African continent in 2015. AFP surveillance indicators were examined in the World Health Organization African and Eastern Mediterranean Region countries that reported polio since 2011, or experienced the recent Ebola virus disease (Ebola) outbreak. The proportion that met both indicators in the African Region countries declined from 2014 to 2015. All Eastern Mediterranean Region countries met both indicators. Surveillance gaps continued at subnational levels. In Ebola-affected countries, AFP surveillance quality weakened at national and subnational levels.

What are the implications for public health practice?

Rapid improvements in AFP surveillance are needed in African Region countries to ensure timely polio-free certification. Sensitive and timely surveillance becomes more critical as polio cases decline. Gaps in surveillance quality must be identified and resolved, especially subnationally. As long as polioviruses circulate in any country, all countries remain at risk.

Vital Signs: Preparing for Local Mosquito-Borne Transmission of Zika Virus — United States, 2016

CDC Zika Response

On April 1, 2016, this report was posted as an MMWR Early Release on the MMWR website (http://www.cdc.gov/mmwr).

Widespread Zika virus transmission in the Region of the Americas since 2015 has heightened the urgency of preparing for the possibility of expansion of mosquito-borne transmission of Zika virus during the 2016 mosquito season (*1*). CDC and other U.S. government agencies have been working with state and local government partners on prevention and early detection of Zika virus infection and will increase these activities during April as part of their preparation for the anticipated emergence of mosquito-borne transmission of Zika virus in the continental United States.

Zika virus is spread primarily through the bite of infected *Aedes* species mosquitoes, but it can also be transmitted during sex by a man to his partners and from a pregnant woman to her developing fetus (*2*). The most common signs and symptoms of Zika virus disease are fever, rash, joint pain, and conjunctivitis. The illness is usually mild, with symptoms lasting from several days to a week. There is increasing evidence that Zika virus infection during pregnancy is associated with early pregnancy loss, microcephaly, and other pregnancy problems (*3*). CDC therefore recommends special precautions for pregnant women. Pregnant women are advised not to travel to areas with active Zika virus transmission and to consistently and correctly use condoms during sex (i.e., vaginal intercourse, anal intercourse, or fellatio) or to abstain from sex for the duration of the pregnancy with male partners who reside in or have traveled to areas with active Zika virus transmission (http://www.cdc.gov/zika/geo/index.html). Pregnant women who live in or must travel to one of these areas should talk to their health care provider and strictly follow steps to prevent Zika virus infection acquisition from mosquito bites (http://wwwnc.cdc.gov/travel/page/avoid-bug-bites) and through sexual transmission (http://www.cdc.gov/zika/transmission/sexual-transmission.html) (*4*,*5*).

On April 1, 2016, CDC is hosting a 1-day Zika Action Plan Summit, which focuses on awareness and planning for U.S. state and local jurisdictions most likely to face mosquito-borne transmission of Zika virus in the coming months (*6*). The Commonwealth of Puerto Rico, U.S. Virgin Islands, and American Samoa are already experiencing active mosquito-borne Zika virus transmission at varying levels (*7*). The U.S. government convened this summit to provide senior state and local government officials with information, plans, and tools to improve Zika preparedness, and an opportunity for them to develop effective response plans for their jurisdictions.

Persons who are planning travel should visit CDC's Travelers' Health site (http://wwwnc.cdc.gov/travel/page/zika-travel-information) for the most up-to-date travel information. Areas with active Zika virus transmission are likely to change over time and might include locations not yet listed. CDC has published interim guidelines and additional updates on Zika virus disease and will continue sharing information as more is learned. Additional publications and resources are available (Box).

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BOX. Additional publications and resources regarding Zika virus disease

- CDC's Zika website (http://www.cdc.gov/zika/)
- MMWR Zika reports (http://www.cdc.gov/mmwr/zika_reports.html)
- Zika virus health information resource guide, National Institutes of Health (https://disasterinfo.nlm.nih.gov/dimrc/zikavirus.html)
- CDC's Vital Signs fact sheet on Zika virus (http://www.cdc.gov/vitalsigns/)

References

1. CDC. Zika Virus. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. http://www.cdc.gov/zika/
2. CDC. Zika virus: transmission and risks. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. http://www.cdc.gov/zika/transmission/index.html
3. Brasil P, Pereira JP Jr, Raja Gabaglia C, et al. Zika virus infection in pregnant women in Rio de Janeiro—preliminary report. N Engl J Med. Published online March 4, 2016. http://dx.doi.org/10.1056/NEJMoa1602412
4. Petersen EE, Polen KN, Meaney-Delman D, et al. Update: interim guidance for health care providers caring for women of reproductive age with possible Zika virus exposure—United States, 2016. MMWR Morb Mortal Wkly Rep. Published online March 25, 2015. http://dx.doi.org/10.15585/mmwr.mm6512e2er
5. Oster AM, Brooks JT, Stryker JE, et al. Update: interim guidance for prevention of sexual transmission of Zika virus—United States, 2016. MMWR Morb Mortal Wkly Rep. Published online March 25, 2016. http://dx.doi.org/10.15585/mmwr.mm6512e3er
6. CDC. Zika Action Plan (ZAP) summit. Atlanta, GA: US Department of Health and Human Services, CDC; 2016. http://www.cdc.gov/zap/
7. Thomas DL, Sharp TM, Torres J, et al. Local transmission of Zika virus—Puerto Rico, November 23, 2015–January 28, 2016. MMWR Morb Mortal Wkly Rep 2016;65:154–8. http://dx.doi.org/10.15585/mmwr.mm6506e2

Notes from the Field

Thyrotoxicosis After Consumption of Dietary Supplements Purchased Through the Internet — Staten Island, New York, 2015

Angela Regina, DO[1]; Nima Majlesi, DO[2]

On June 18, 2015, a woman aged 30 years was brought to the Staten Island University Hospital Emergency Department (ED) in New York by her mother, who reported that the patient had become acutely confused at home, was repeating herself, and did not recognize her family members. She had a diagnosis of bipolar disorder for which she took lithium, and she had a history notable for polysubstance abuse (use of three or more addictive drugs in the past 12 months). Her other medications included risperidone (an antipsychotic), benztropine (an anticholinergic), and bupropion (an antidepressant). ED staff members learned from the patient's mother that the patient had started taking "diet pills" that contained a thyroid hormone, which the patient had purchased through the Internet 2 weeks earlier. The patient volunteered that she had doubled the dosage 1 week earlier, in an attempt to lose more weight.

In the ED, the patient was awake but was unaware of her location or the date. Her mother said that the patient had expressed no recent suicidal ideation. Her vital signs were within normal limits, with the exception of a heart rate of 130 beats per minute. She had a mild tremor at rest, but was not ataxic. While in the ED, her temperature increased to 101.5°F (38.6°C). Laboratory analyses indicated lithium concentration in the therapeutic range, and serum salicylates and acetaminophen concentrations were undetectable. A urine drug screen was negative for cocaine, tetrahydrocannabinol (the psychoactive component of cannabis), opioids, phencyclidine, and benzodiazepines.

Thyroid function studies indicated thyroid stimulating hormone <0.01 mIU/mL (normal range = 0.27–4.20 mIU/ml), triiodothyronine (T_3) >32.5 pg/mL (normal range = 1.80–4.60 pg/mL), and thyroxine (T_4) >7.8 ng/dL (normal range = 0.9–1.8 ng/dL). The active ingredient listed on the bottle of diet pills was "triiodothyronine hormone 25 mcg." The mother counted the pills and reported that 25 were missing from the bottle. The patient was admitted to the hospital with a diagnosis of acute thyrotoxicosis, secondary to exogenous thyroid hormone. She was initially treated with intravenous hydration and benzodiazepines. Her symptoms improved only minimally, and she remained delirious and tachycardic for >72 hours. On the third hospital day, the patient was evaluated by a toxicologist who recommended starting treatment with beta-blockers to ameliorate the symptoms of hyperthyroid-associated increased beta-adrenergic tone. Once treatment with propranolol was initiated, the patient's vital signs and mental status stabilized. Her thyroid function tests normalized during her hospital stay after discontinuing the thyroid supplements, and she was discharged on the fourth day.

The patient had purchased the product, which contained triiodothyronine (a thyroid hormone), online. Although the company's website claims that the capsules are for research purposes only, the comments on the company's webpage indicate that consumers purchase the supplements for weight loss. Pharmaceutical thyroid supplementation for patients with hypothyroidism is available by prescription only. Patients who obtain thyroid supplementation on the internet or through other sources are at risk for thyrotoxicosis because of the unpredictability of dosing and contents.

Thyrotoxicosis has previously been reported related to use of dietary supplements (*1*,*2*). During 2013, dietary supplements were among the top 25 substances most frequently involved in human toxic exposures (*3*). Dietary supplements are easily obtained, especially through the Internet, but they are not subject to the same regulatory safeguards as are drugs. Manufacturers must register their facilities with the Food and Drug Administration (FDA), but they are not required to obtain FDA approval before the development of or sale of dietary supplements (*4*,*5*). Manufacturers and distributors also are required to make certain that all claims and information on the product label and in other labeling are truthful and not misleading. FDA's authority related to supplements, however, is generally limited to postmarketing surveillance, such as adverse event monitoring and facility inspections (*6*). This adverse reaction and product was reported to MedWatch, FDA's Safety Information and Adverse Event Reporting Program.

Commercially available thyroid supplements can contain clinically relevant amounts of triiodothyronine, and consumption has the potential to cause profound metabolic derangements (*7*). The fact that these products are readily available on the Internet market, often without online disclosure of the active ingredients, poses a substantial health risk. Health care providers evaluating patients with signs and symptoms of thyrotoxicosis should inquire about the use of dietary supplements and examine product labels to ascertain the contents. In addition to supportive therapy, the use of beta-blockers should be considered after consultation with a medical toxicologist. Consumers are encouraged to exercise caution when purchasing products labeled as containing thyroid hormones, and health care providers are strongly encouraged to report adverse events associated with the use of dietary supplement products to MedWatch (http://www.fda.gov/medwatch).

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References

1. Daniels GH, Sluss P. Pure T3-thyrotoxicosis from a Mexican weight loss supplement. Endocr Pract 2013;19:559–60.
2. Dimeski G, Lampe G, Brown NN. Chinese herbal supplements the cause of thyrotoxicosis. Pathology 2013;45:185–6. http://dx.doi.org/10.1097/PAT.0b013e32835c879e
3. Mowry JB, Spyker DA, Cantilena LR Jr, McMillan N, Ford M. 2013 annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 31st annual report. Clin Toxicol (Phila) 2014;52:1032–283. http://dx.doi.org/10.3109/15563650.2014.987397
4. Food and Drug Administration. Dietary supplements. College Park, MD: US Department of Health and Human Services, Food and Drug Administration; 2016. http://www.fda.gov/Food/DietarySupplements/
5. Food and Drug Administration. Dietary supplement products & ingredients. College Park, MD: US Department of Health and Human Services, Food and Drug Administration; 2016. http://www.fda.gov/Food/DietarySupplements/ProductsIngredients/default.htm
6. Bauer BA, Elkin PL, Erickson D, Klee GG, Brennan MD. Symptomatic hyperthyroidism in a patient taking the dietary supplement tiratricol. Mayo Clin Proc 2002;77:587–90. http://dx.doi.org/10.4065/77.6.587
7. Kang GY, Parks JR, Fileta B, et al. Thyroxine and triiodothyronine content in commercially available thyroid health supplements. Thyroid 2013;23:1233–7. http://dx.doi.org/10.1089/thy.2013.0101

Notes from the Field

Mycobacterium abscessus Infections Among Patients of a Pediatric Dentistry Practice — Georgia, 2015

Gianna Peralta, MPH[1,2]; Melissa Tobin-D'Angelo, MD[1]; Angie Parham, DVM[1,3]; Laura Edison, DVM[1,4]; Lauren Lorentzson, MPH[1]; Carol Smith, MSHA[1]; Cherie Drenzek, DVM[1]

On September 13, 2015, the Georgia Department of Public Health (DPH) was notified by hospital A of a cluster of pediatric *Mycobacterium abscessus* odontogenic infections. Hospital A had provided care for nine children who developed presumptive or confirmed *M. abscessus* infection after having a pulpotomy at pediatric dentistry practice A (dates of onset: July 23, 2014–September 4, 2015). During a pulpotomy procedure, decay and the diseased pulp are removed to preserve a deciduous tooth. DPH initiated an investigation to identify the outbreak source and recommend prevention and control measures.

M. abscessus, a rapidly growing, nontuberculous mycobacterium (NTM), is found ubiquitously in the environment in water, soil, and dust. It commonly causes skin and soft tissue infection and can cause disease in multiple organs (*1*). NTM species display tolerance to commonly used disinfectants and are frequently found in the plumbing of health care facilities and water distribution systems (*2*). Improperly maintained dental unit water lines can permit growth and amplification of microorganisms, including NTM, which can form a biofilm and replicate within waterline tubing (*3*). Outbreaks have been reported in different clinic settings, including acupuncture clinics, a cosmetic surgery clinic, and a general medical clinic, although not dental clinics (*4–7*).

Probable cases were defined as occurrence of facial or neck swelling and biopsy-confirmed granulomatous inflammation among children with an illness onset date on or after January 1, 2014. Confirmed cases were those in which *M. abscessus* was isolated by laboratory culture. Active case finding included contacting all patients who had a pulpotomy since January 1, 2015, notifying area pediatricians and dentists of the outbreak, and reviewing hospital A pathology reports and *M. abscessus* positive cultures since January 1, 2014. DPH staff visited practice A on September 22, 2015, to evaluate infection control and prevention practices, and to view a mock pulpotomy demonstration. Practice A used tap water for pulpotomies without water quality monitoring or bleaching of waterlines at the end of each day, as recommended in the manufacturer guidelines.*

* http://www.midmark.com/docs/librariesprovider6/pdfs/003-1261-00.pdf?sfvrsn=2.

No other infection control deficiencies were noted. Water samples were collected for microbiologic analysis, and patient and water sample isolates were sent to CDC for molecular characterization by pulsed-field gel electrophoresis (PFGE).

Practice A had performed 1,386 pulpotomies since January 1, 2014. As of January 1, 2016, a total of 20 patients with confirmed (n = 11) or probable (n = 9) *M. abscessus* infections were identified, resulting in an attack rate of 1%; case finding is ongoing. Median patient age was 7 years (range = 3–11 years), and median incubation period was 65 days (range = 18–164 days). All patients were severely ill, requiring hospitalization at least once for a median of 7 days (range = 1–17 days); 17 patients required surgical excision and 10 received outpatient intravenous antibiotics (Table). As of April 5, 2016, no deaths have resulted from infection.

TABLE. Demographic characteristics, symptoms, diagnostic evaluations, and treatment of 20 patients with confirmed or probable *Mycobacterium abscessus* infections — Georgia, March 12, 2014–November 12, 2015

Characteristic	No. patients (N = 20)	(%)
Median age, yrs* (range)	7 (3–11)	NA
Male	11	55
Asthma	3	15
Immunocompromised	0	0
Signs and symptoms		
Pain	17	85
Osteomyelitis	14	70
Facial swelling	12	60
Lymphadenopathy	10	50
Pulmonary nodules	7	35
Fever	1	5
Diagnostic evaluation		
Neck CT	17	85
Chest radiograph	11	55
Dental radiograph	8	40
Ultrasound	5	25
Maxillofacial CT	2	10
MRI	1	5
Treatment		
Excision	17	85
Outpatient IV antibiotics by PICC†	10	50
Incision or drainage	7	35
Laboratory result		
AFB stain negative	13	65
AFB stain positive	7	35
AFB culture positive	11	55
AFB culture negative§	9	45

Abbreviations: AFB = acid-fast bacteria; CT = computed tomography; IV = intravenous; MRI = magnetic resonance imaging; NA = not available; PICC = peripherally inserted central catheter.
* Age at illness onset.
† Amikacin or amikacin and cefoxitin by PICC.
§ Two (11%) AFB cultures are pending.

All water samples from the seven dental stations had bacterial counts above the American Dental Association recommended ≤500 colony-forming units (CFU)/mL (average = 91,333 CFU/mL); *M. abscessus* was isolated from all water samples. All water and patient isolates were indistinguishable by PFGE, indicating a common source.

This outbreak was caused by contaminated water used during pulpotomies, which introduced *M. abcessus* into the chamber of the tooth during irrigation and drilling. *M. abscessus* can cause severe infection among immunocompetent children, and because *M. abscessus* is ubiquitous in the environment, it poses a contamination risk. To prevent infections associated with waterlines, dental practices should follow manufacturer guidelines to disinfect waterlines, monitor water quality to ensure recommended bacterial counts, use point-of-use water filters, and eliminate dead ends in plumbing where stagnant water can enable biofilm formation (*3*,*8*). Health care providers should promptly report suspected outbreaks of infectious diseases to public health authorities so that an investigation can be initiated and appropriate control measures implemented.

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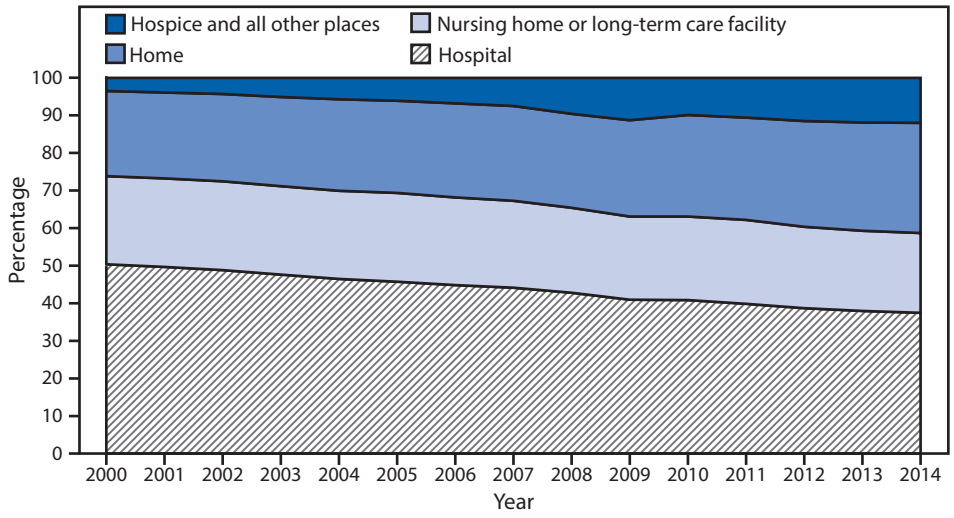
References

1. Lee MR, Sheng WH, Hung CC, Yu CJ, Lee LN, Hsueh PR. *Mycobacterium abscessus* complex infections in humans. Emerg Infect Dis 2015;21:1638–46. http://dx.doi.org/10.3201/2109.141634
2. Williams MM, Chen TH, Keane T, et al. Point-of-use membrane filtration and hyperchlorination to prevent patient exposure to rapidly growing mycobacteria in the potable water supply of a skilled nursing facility. Infect Control Hosp Epidemiol 2011;32:837–44. http://dx.doi.org/10.1086/661282
3. Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz DM; Guidelines for infection control in dental health-care settings—2003. MMWR Recomm Rep 2003;52(No. RR-17).
4. Furuya EY, Paez A, Srinivasan A, et al. Outbreak of *Mycobacterium abscessus* wound infections among "lipotourists" from the United States who underwent abdominoplasty in the Dominican Republic. Clin Infect Dis 2008;46:1181–8. http://dx.doi.org/10.1086/529191
5. Villanueva A, Calderon RV, Vargas BA, et al. Report on an outbreak of postinjection abscesses due to *Mycobacterium abscessus*, including management with surgery and clarithromycin therapy and comparison of strains by random amplified polymorphic DNA polymerase chain reaction. Clin Infect Dis 1997;24:1147–53. http://dx.doi.org/10.1086/513656
6. Griffith DE, Aksamit T, Brown-Elliott BA, et al.; ATS Mycobacterial Diseases Subcommittee; American Thoracic Society; Infectious Disease Society of America. An official ATS/IDSA statement: diagnosis, treatment, and prevention of nontuberculous mycobacterial diseases. Am J Respir Crit Care Med 2007;175:367–416. http://dx.doi.org/10.1164/rccm.200604-571ST
7. Tang P, Walsh S, Murray C, et al. Outbreak of acupuncture-associated cutaneous *Mycobacterium abscessus* infections. J Cutan Med Surg 2006;10:166–9. http://dx.doi.org/10.2310/7750.2006.00041
8. South Carolina Department of Health and Environmental Control. DHEC statement concerning mycobacteria at Greenville Health System. Columbia, SC: South Carolina Department of Health and Environmental Control; 2014. http://www.scdhec.gov/Agency/NewsReleases/2014/nr20140721-01/

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS

Percentage Distribution of Deaths,* by Place of Death†,§ — United States, 2000–2014



* Percentage was calculated as (deaths occurred in a place/all deaths) x 100.
† Excluding all deaths from external causes defined by *International Classification of Diseases, Tenth Revision* (ICD-10) codes V01-Y89 and cause-of-death codes U01–U03.
§ Deaths in hospice and all other places include those in a hospice facility; all other places other than hospital, nursing home, and home; and unknown place of death. The category "hospice" was introduced with the revised death certificate in 2003. The number of states using the revised death certificate grew from four states in 2003 to 46 states and the District of Columbia in 2014.

The percentage of deaths that occurred in a hospital decreased 25.7%, from approximately half (50.2%) in 2000 to 37.3% in 2014. During 2000–2005, the percentage of deaths that occurred in a nursing home or long-term care facility remained relatively stable, and then decreased 10.1% during 2005–2014. The percentage of deaths that occurred at a decedent's home increased 29.5% during 2000–2014. The percentage of deaths that occurred in hospice and all other places increased 242.9%, from 3.5% in 2000 to 12.0% in 2014.

Source: National Vital Statistics System. Underlying cause of death data, 2000–2014. http://wonder.cdc.gov/ucd-icd10.html.

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ISSN: 0149-2195 (Print)